Marine Mammal Science

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ARTICLE

Skin and Mucosal Lesions of Indo-Pacific Bottlenose (*Tursiops aduncus*) and Common Dolphins (*Delphinus delphis*) of South Australia, With a Focus on Pathology

Rebecca Souter[1] | Anne-Lise Chaber[1] | Luciana Möller[2] | Ikuko Tomo[3] | Lucy Woolford[1]

[1]School of Animal and Veterinary Sciences, University of Adelaide, Roseworthy, South Australia, Australia | [2]College of Science and Engineering, Flinders University, Bedford Park, South Australia, Australia | [3]South Australian Museum, Adelaide, South Australia, Australia

Correspondence: Rebecca Souter (rebecca.souter@adelaide.edu.au)

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ABSTRACT

Skin lesions are valuable indicators of individual and population health in cetaceans. To understand the occurrence of skin and mucosal lesions among dolphins inhabiting South Australian waters, this study employs opportunistic sampling of deceased dolphins during 2021–2024. A total of 52 dolphins, comprising 23 Indo-Pacific bottlenose dolphins (*Tursiops aduncus*) and 29 common dolphins (*Delphinus delphis*), underwent thorough macroscopic, microscopic, and ancillary diagnostics of identified lesions. Thirty-three individuals displayed 115 skin and 12 mucosal lesions. The most common skin lesion sites were the head (23%–34%), peduncle (25%–26%), and lateral surfaces (17%–19%). A negative association was found between body condition and the number of infectious skin lesions in *T. aduncus*. Tattoo Skin Disease was identified in 13% of *D. delphis* skin lesions and 29% of *T. aduncus* skin lesions. Additional etiologies of skin lesions included bacteria (including *Erysipelothrix rhusiopathiae*, *Mycobacterium marinum*, *Vibrio* spp.), protozoa, physiological, and trauma-related (propeller strike and shark bite). Opportunistic bacterial ulcerative dermatitis occurred predominantly in dolphins from the Adelaide Dolphin Sanctuary. Novel findings include the identification of herpesvirus in oral/genital papillomas, cutaneous lesions from an erysipelas-infected animal, and *M. marinum* skin abscessation, not previously reported in free-ranging Australian cetaceans. This study complements live-animal observational studies and provides diagnostic insight into dolphin skin lesions, supporting their use as population and ecosystem health indicators.

1 | Introduction

The infraorder Cetacea evolved from terrestrial artiodactyls and consists of two mammalian parvorders: Odontoceti and Mysticeti. Odontocetes include all tooth whales and Mysticetes include all baleen whales (Committee on Taxonomy 2024). Having evolved from a terrestrial ancestry, specialized adaptations have been acquired to reside in a completely aquatic environment (Lopes-Marques et al. 2019; Menon et al. 2022). Cetacean skin provides physical protection, acts as a thermoregulatory and sensory organ, is hydrodynamic, maintains physiological homeostasis, assists in buoyancy control, and provides an energy store (Cozzi et al. 2016; Espregueira Themudo et al. 2020). It is comprised of a thick epidermal layer (1–4.5 mm mean) and a dermis and subcutis (blubber layer), held tightly together by deep interdigitating rete pegs and dermal papillae at the dermal-epidermal junction. The epidermis is made up of only three strata (as opposed to five in terrestrial mammals):

the stratum externum, stratum spinosum, and stratum germinativum (basal layer) and has a high turnover rate to prevent biofouling and maintain hydrodynamics (Cozzi et al. 2016; Morales-Guerrero et al. 2017). Cetacean skin lacks hair follicles (with the exception of vibrissae in neonates) and sebaceous glands, and melanin is present within the cells of all epidermal layers (Morales-Guerrero et al. 2017; Espregueira Themudo et al. 2020).

The integumentary system plays an important role in cetacean immunity. The loss of specific genes (e.g., the pilosebaceous gland) in cetacean skin allows for reduced resistance in an aquatic environment but means the skin requires an increased ability to withstand external stimuli (e.g., friction, ultraviolet (UV) radiation, thermal changes, trauma) and resist pathogens (Morales-Guerrero et al. 2017; Cozzi et al. 2016). Immunosurveillance cells which migrate towards superficial layers during injury are present within the dermis (Zabka and Romano 2003), and rapid cell turnover minimizes the need for inflammatory responses to injury (e.g., scab formation) (Espregueira Themudo et al. 2020). Skin microbiome plays a crucial role in skin health. Disruption of skin microbial communities due to adverse environmental conditions can alter the barrier function and reduce skin immunity (Hargis and Sherry 2017). Pollution, eutrophication, UV radiation, hyposalinity (< 5 pars per thousand), and cold temperatures have all been associated with increased occurrence and prevalence of skin lesions in cetaceans (Wilson et al. 1999; Hart et al. 2012; Mouton and Botha 2012; Bossley and Woolfall 2014; Sanino et al. 2014; Duignan et al. 2020; Croft et al. 2020). For example, in dolphins from Australian waters, prolonged freshwater exposure caused sloughing of the epidermis and subsequent colonization of pathogenic organisms including bacteria and fungi (Duignan et al. 2020). Furthermore, skin lesion prevalence has been shown to increase in urbanized areas with greater anthropogenic influence (Mouton and Botha 2012).

Previous research on cetaceans in South Australia has included pathological surveillance of stranded and bycaught animals (Segawa and Kemper 2015; Tomo and Kemper 2022), field studies of population demographics and genetic variation (Zanardo et al. 2016), as well as life history investigations highlighting key biological characteristics of populations in this region (Kemper et al. 2014, 2019). Throughout these studies, skin lesions of multiple etiologies have been reported and identified, including vessel strike wounds (Byard et al. 2013), entanglements (Kemper et al. 2023), burn-like injuries (Bossley and Woolfall 2014), tattoo skin disease (TSD) (Tomo and Kemper 2022), and bacterial disease (Souter et al. 2021). Disease due to cetacean morbillivirus, toxoplasmosis, *Corynebacterium ulcerans*, and *Streptococcus iniae* has all been detected, highlighting the variety of infectious pathogens (some of which are zoonotic) infecting the local cetacean populations (Kemper et al. 2016; Souter et al. 2021; Tomo and Kemper 2022).

The Adelaide Dolphin Sanctuary (ADS) is a 118 km^2 marine reserve dedicated to the protection of a resident population of approximately 20–30 Indo-Pacific bottlenose dolphins (*Tursiops aduncus*) and the habitats that supports them (Department for Envrionment and Heritage (DEH) 2008; Haigh et al. 2025). This dolphin population has been studied for over 30 years through observational surveys and necropsy of deceased individuals (Adamczak et al. 2018; Bossley et al. 2017; Tomo and Kemper 2022). Resident dolphins are recognized and monitored through observations of their individual dorsal fin characteristics. The ADS is situated 15 km northwest of Adelaide Central Business District and encompasses the Port River Estuary, which is Adelaide's major shipping port and industrial hotspot (Bossley et al. 2017). Over an 11-month period between 2021 and 2022, the ADS resident dolphin population experienced a mortality event resulting in the death of 7 resident dolphins. A definitive cause of death could not be established but was characterized by progressive wasting over a 3–4 week period leading to emaciation, opportunistic infections such as otitis media, intestinal dysbiosis, and ulcerative skin lesions not described previously (Department for Envrionment and Water (DEW) 2022, Souter et al. 2023). The Port River Estuary has a known history of anthropogenic contamination (e.g., organochlorines, heavy metals, and per- and polyfluorinated substances) and anthropogenic influence (e.g., boating, fishing, dredging, and industry) (Butterfield and Gaylard 2005; Gaylard 2017; Weijs et al. 2020; Kirkwood et al. 2022), leading to environmental degradation and possible public health concerns.

As part of the investigation into ADS dolphin mortalities during 2021–2022, necropsy investigations of stranded, deceased Indo-Pacific bottlenose dolphins and common dolphins (*Delphinus delphis*) were conducted in SA to compare skin lesion pathology, etiology, and prevalence among various geographic regions. The Indo-Pacific bottlenose dolphin is a coastal species and is likely to be exposed to more anthropogenic stressors compared to common dolphins, which are mainly found in the pelagic environment (Kemper et al. 2008; Cribb et al. 2013; Filby et al. 2013; Zanardo et al. 2016). Additionally, this study aims to improve diagnostic interpretation of skin lesions during non-invasive field surveys of live dolphins in SA, creating a differential diagnosis list for gross lesions based on microscopy, microbiology, and molecular testing of necropsied samples.

2 | Materials and Methods

2.1 | Sample Collection

During 2021–2024 beach cast, deceased dolphins were opportunistically collected from the South Australian coastline. Another three dolphins stranded alive and were subsequently euthanised based on humane grounds (authorized by the SA Department for Environment and Water (DEW)) and submitted for diagnostic necropsy. Geographic coordinates were collected upon stranding incidents and dolphins were categorized into one of seven approximate geographic regions (ADS, East Gulf Saint Vincent (GSV), Kangaroo Island (KI), West GSV, East Spencer Gulf (SG), West SG, and Coorong (CO)) (Figure 1), modified from the described marine bioregions of SA (Edyvane 1999). Stranded dolphins were reported by members of the public to various collaborating organizations (DEW, South Australian Museum, Flinders University, The University of Adelaide, Australian Marine Wildlife Research and Rescue Organization), where they would be examined for level of decomposition. Only carcasses with decomposition codes 3 or below (Geraci and Lounsbury 2005) were collected and transported

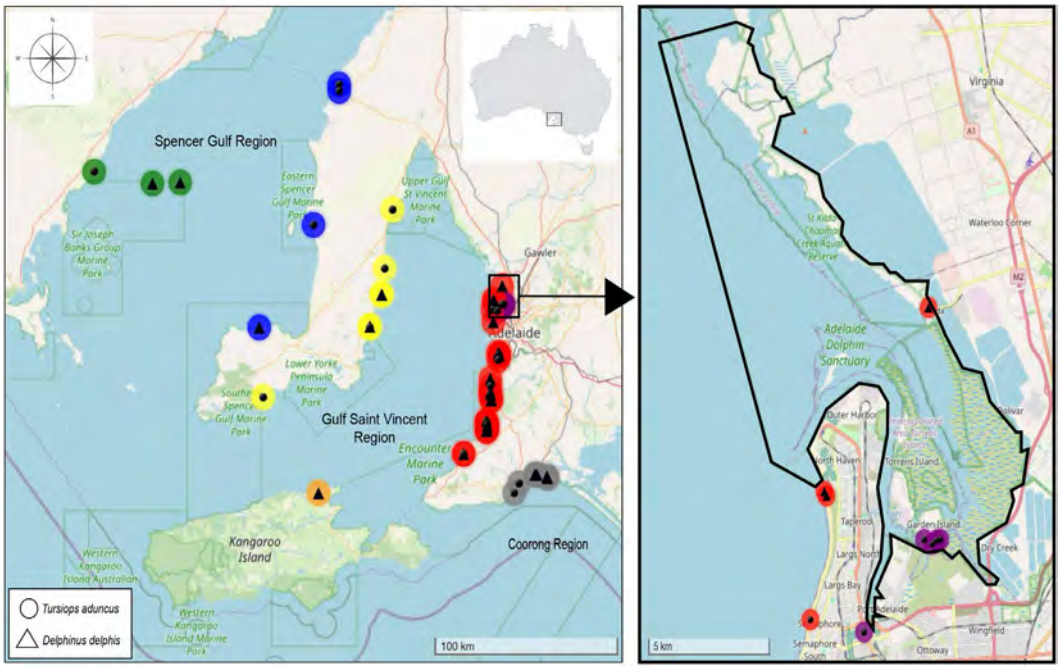

FIGURE 1 | Indo-Pacific bottlenose dolphins (*Tursiops aduncus*) and common dolphins (*Delphinus delphis*) strandings along the South Australian coastline during 2021–2024. Regions are represented by colors as follows: Green = west Spencer Gulf; blue = east Spencer Gulf; yellow = west Gulf Saint Vincent; red = east Gulf Saint Vincent; gray = Coorong; orange = Kangaroo Island. The image subset on the right displays the Adelaide dolphin Sanctuary (ADS) demarcated by the black solid line. Known resident dolphins of the ADS are represented in purple.

to the University of Adelaide Veterinary Diagnostic Laboratory, Roseworthy, SA, or the South Australian Museum, Bolivar, SA. Animals were placed in the freezer or refrigerator depending on personnel and laboratory availability for necropsy over the coming days. Two animals were retrieved from fishing nets of the South Australian Sardine Fishery.

2.2 | Macroscopic Investigation

Each dolphin was assigned a body condition score (BCS) (normal/robust, thin, or emaciated) based on criteria previously described (Joblon et al. 2014; Kemper et al. 2016; Soares et al. 2022). Skin and mucosal lesions (oral cavity, urogenital) were assigned a number, photographed, and described on a data sheet (anatomical location, size, color, shape, consistency), and a section of the lesion, including the margin of healthy and diseased tissue, was collected and placed in 10% neutral buffered formalin with an identification label. The appearance of the transverse section of each lesion was noted, described, and photographed when indicated. In fresh specimens (i.e., specimens that had not been frozen), microbiological swabs of heart blood and brain/spinal cord were collected aseptically. Additionally, swabs were collected from all specimens with lesions indicative of infectious disease, and tissue samples from these lesions were frozen and archived. A broad suite of samples (brain, heart, lung, tongue, liver, spleen, pancreas, adrenal gland, kidney, heart blood, thyroid gland, skeletal muscle, blubber, mesenteric lymph node, feces, stomach contents) was collected, frozen, and fixed from all animals as part of the investigation into morbidity and mortality. Identification of *Tursiops* species was verified by C.K. (South Australian Museum) based on examination of skull morphology (Kemper 2004).

Dolphins were assigned to one of five relative age categories (neonate, calf, juvenile, subadult, and adult) based on methodology previously described (Kemper and Gibbs 2001; Kemper et al. 2016). Neonates were classified based on the presence of neonatal folds, considered up to approximately three months old. Calves included individuals of total body length less than 1.5 m for *T. aduncus* and less than 1.3 m for *D. delphis*. Juveniles included those above 1.5 m for *T. aduncus* and above 1.3 m for *D. delphis* and were both physically and sexually immature. Subadults were sexually mature but physically immature, and adults were both sexually and physically mature. Sexual maturity was confirmed in females by examining for the presence of corpora scarring on the ovaries, lactation, or pregnancy (Kemper et al. 2019; Palmer et al. 2022). For male dolphins, a sample of testis was collected and placed in formalin for microscopic evaluation of active spermatogenesis, confirming sexual maturity. Physical maturity was determined based on complete epiphyseal fusion of thoracic vertebrae.

2.3 | Microscopic Investigation

Formalin preserved skin lesion and testis samples were trimmed, embedded in paraffin wax, cut to 4 μm using a microtome, and stained with hematoxylin and eosin. Lesions with extensive sand contamination could not be examined histologically due to difficulty in processing. Skin lesions were examined and described by an American College of Veterinary Pathologists (ACVP) boarded veterinary pathologist (LW), and special stains Gram, Ziehl-Neelsen, Grocott's methenamine silver, Martius Scarlet Blue, and Periodic Acid-Schiff were performed as appropriate. Male sexual maturity was determined microscopically by examining the testes for evidence of spermiogenesis or

spermatogenesis. Additional tissue samples and brains collected during necropsy were processed and examined as above for determination of cause of death (COD), when possible, as well as the presence of comorbidities. Modified from Van Bressem, Van Waerebeek, et al. (2009), individuals were assigned a health status based on necropsy findings. Animals were considered in "poor health" if they were emaciated and displayed evidence of disease (e.g., systemic inflammation, starvation/malnutrition, degenerative disease). Animals were considered "healthy" if the COD was associated with an acute process (e.g., traumatic injury or entanglement) or could not be determined and no other findings alluding to poor health were identified.

2.4 | Microbiology and Infectious Disease Testing

Microbiological swabs were submitted to Roseworthy Veterinary Hospital Pathology (RVHP), The University of Adelaide, South Australia. Samples were cultured on horse blood agar, MacConkey agar, and Thiosulfate-citrate-bile-salts-sucrose (TCBS) agar, and incubated at 25°C–29°C. Skin samples were incubated in aerobic conditions and other sites incubated in aerobic and anaerobic conditions for 48 h. Lesions with histological evidence of Mycobacteria were confirmed at the mycobacterium reference laboratory (MRL) in South Australia. Polymerase Chain Reaction (PCR) testing for Herpesvirus was performed on fresh or formalin-fixed paraffin-embedded (FFPE) skin or mucosal lesions suggestive of viral disease at the Australian Centre for Disease Preparedness (ACDP), Geelong using Pan-Herpes PCR (VanDevanter et al. 1996). Additionally, frozen tissue samples (e.g., lung, brain, spinal cord, spleen, and joint capsule) were selected from individuals displaying microscopic evidence of infectious disease and targeted PCR examination was performed for cetacean morbillivirus, Influenza A, herpesvirus, or *Brucella* spp. at ACDP.

2.5 | Skin Lesion Descriptions

2.5.1 | Macroscopic Features

Skin and mucosal lesions were described based on their macroscopic morphology following a recently published matrix (Ewing et al. 2023). Lesions were categorized into groups based on their primary physical appearance: focal ulceration, incisional cut/laceration, tattoo skin disease (TSD), flat, raised, depressed, hyperkeratotic, and papillomatous. TSD was assigned based on the appearance of characteristic tattoo-like, pinhole, or ring lesions as described previously (Geraci et al. 1979; Van Bressem et al. 1993, 2018, 2022; Blacklaws et al. 2013; Barnett et al. 2015).

2.5.2 | Microscopic Features

Microscopic features, many described in Segura-Göthlin, Fernández, Arbelo, Andrada Borzollino, et al. (2023), were assigned to skin lesions during histological assessment. Descriptors were used to group lesions by prominent disease mechanisms (degenerative, inflammatory, disruptive, vascular, developmental, or cellular differentiation defect), which were further grouped by degree of confidence, modified from Ewing et al. (2023): confirmed, probable, and suspect. Disease mechanism was classified as confirmed when there were characteristic and specific pathological changes and when etiological agents were visualized or confirmed by ancillary testing. When observed, the presence of intranuclear or intracytoplasmic inclusion bodies was described. A probable case displayed characteristic gross and histopathological changes, but the etiological agents were unconfirmed by direct visualization or ancillary testing. The known or most likely etiological agent was assigned to confirmed and probable cases. A suspect case was the lowest level of confidence but displayed suggested disease processes. Etiology was considered unknown in these cases.

2.5.3 | Lesion Categorization

Lesions with two or more etiologies (e.g., trauma with secondary infection) were categorized based on the primary disease mechanism or initiating event. Cofactors/comorbidities or secondary findings are considered throughout with the interpretation of results.

Lesions were classified as infectious or non-infectious etiologies based on diagnostic findings and were subsequently categorized into the following subcategories: viral, bacterial, fungal, parasitic, algal, traumatic (anthropogenic (fishing entanglement, propeller strike) and non-anthropogenic (abrasions, conspecific biting, shark attack)), inflammatory non-infectious, physiological/environmental, chemical/toxic, miscellaneous, and unknown (Townsend and Staggs 2020).

2.5.4 | Statistical Analysis

Collation of results was performed using Microsoft Excel (Version 16.96.1) and statistical analysis performed using R (Version 2024.04.2 + 764). To investigate variables (species, sex, sexual maturity, region, BCS, and health status) associated with the number of infectious skin lesions in dolphins, we conducted generalized linear modeling (GLM) using both Poisson and Negative Binomial (NB) regressions, with infectious skin lesion count as the response variable. Model performance was compared using Akaike Information Criterion (AIC), where the NB model indicated improved model fit. Logistic Regression and Generalized Additive Models (GAM) were used to test the significance of TSD occurrence between variables, in addition to seasonality. A probability (p) value of 0.05 was applied. Pairwise comparisons of estimated marginal means (EMMs) were conducted using the emmeans package in R (Lenth 2024) with adjusted p values for multiple comparisons using the Tukey method to reduce the risk of Type 1 errors, controlling family-wise error rate (Tukey 1949).

3 | Results

During the study period, 52 individuals were found stranded deceased or were euthanised and underwent necropsy examination (23 *T. aduncus* and 29 *D. delphis*). Five resident *T. aduncus* of the ADS were included, in addition to stranded

TABLE 1 | Numbers of common dolphins (*Delphinus delphis*) and Indo-Pacific bottlenose dolphins (*Tursiops aduncus*) examined in South Australia between June 2021 and April 2024 and total number of skin and mucosal lesions examined. *n* = number of animals or lesions examined.

***D. delphis* (*n* = 29)**			***T. aduncus* (*n* = 23)**		
	Skin (*n* = 53)	**Mucosal (*n* = 3)**		**Skin (*n* = 62)**	**Mucosal (*n* = 9)**
Neonate (*n* = 5)	5	0	Neonate (*n* = 4)	1	0
Immature (*n* = 17)	28	0	Immature (*n* = 10)	40	1
Mature (*n* = 7)	20	3	Mature (*n* = 7)	21	8
Female (*n* = 14)	22	2	Female (*n* = 7)	14	1
Male (*n* = 15)	31	1	Male (*n* = 16)	48	8
Normal/robust (*n* = 9)	16	0	Normal/robust (*n* = 7)	16	3
Thin (*n* = 17)	23	2	Thin (*n* = 6)	11	2
Emaciated (*n* = 3)	14	1	Emaciated (*n* = 10)	35	4

dolphins from the east GSV (18 *D. delphis*, 8 *T. aduncus*), east SG (1 *D. delphis*, 3 *T. aduncus*), CO (4 *D. delphis*, 2 *T. aduncus*), KI (one *D. delphis*), west GSV (3 *D. delphis*, 4 *T. aduncus*), and west SG (2 *D. delphis*, 1 *T. aduncus*). Of these, 14 (5 *T. aduncus* and 9 *D. delphis*) did not display skin or mucosal lesions. Skin lesions from an additional five individuals were excluded due to poor sample quality, post-mortem predation hindering interpretation of results, or histopathological examination revealing a scar.

In total, 115 skin lesions and 12 mucosal lesions from 33 individuals were included in the study (Table 1). Skin lesions were identified and examined across varying age, sex, and body conditions in *D. delphis* and *T. aduncus*. Mucosal lesions were not identified in neonates from either species (Table 1). Macroscopic photographs, gross and microscopic descriptions, and results of microbiological and molecular ancillary testing of all skin and mucosal lesions can be accessed in Table S1. Additionally, Table S1 includes COD (if known) and comorbidities identified at necropsy.

Skin lesions were most frequently reported on the head, peduncle, and on the lateral body surfaces (Figure S1). One immature female *T. aduncus* with multiple foci (22-02208) had all lesions distributed over the head. For mucosal lesions, oral lesions were most frequent (11/12), with only one genital lesion identified.

3.1 | Infectious Lesions

In total, 23 individuals (8 *D. delphis*, 15 *T. aduncus*) displayed skin lesions consistent with infectious etiology. Bacterial agents were cultured from five skin lesions from two *T. aduncus*. No fungal lesions were identified in this study. The distribution of infectious skin lesions, in order of frequency, included focal (30/71, 44%), multifocal (17/71, 25%), multifocal to coalescing (11/71, 14%), and locally extensive, irregular, or coalescing (< 10% for each). The association between infectious skin lesion occurrence and temporality (analyzed by month) in both *T. aduncus* and *D. delphis* was not found to be significant ($p = 0.9$, and $p = 0.8$, respectively). *T. aduncus* individuals were found to have significantly higher numbers of infectious skin lesions per individual compared to individual *D. delphis* ($p = 0.02$), with emaciated animals exhibiting the highest lesion burden. Of the mucosal lesions, nine were considered infectious in one *D. delphis* and six *T. aduncus*. An etiological agent was identified in two of these lesions, both in *T. aduncus*.

3.1.1 | Tattoo Skin Disease

TSD was identified in 14/52 dolphins (27%) (10 *T. aduncus*, 4 *D. delphis*), and was considered the primary etiology in 25/70 infectious skin lesions (36%) (Figure 2). Morphology of lesions presented as follows: tattoo (13/25, 52%), ring (8/25, 32%), and pinhole/ulceration (4/25, 16%) (Figure 3, Table 2). The most frequent microscopic findings identified included: acanthosis (21/25, 84%), ballooning/hydropic degeneration (17/25, 68%), spongiosis (24/25, 96%), hyperpigmentation (21/25, 84%), blunted/fused rete pegs (22/25, 88%), and ICIBs (23/25, 92%) (Figure 4, Table 2). Of all cases with TSD, the host had endured cutaneous trauma in association with the lesion or elsewhere on the body in 21% of cases (3/14). Prevalence of TSD was significantly higher in *T. aduncus* (10/23, 43%) compared to *D. delphis* (4/29, 14%) when controlling for sex and sexual maturity ($p = 0.03$). Logistic regression analysis revealed no significant effects of sex, sexual maturity, region, BCS, or health status on the prevalence of TSD across both species ($p > 0.05$). Pairwise comparisons among all combinations revealed no significant relationships. Wide confidence intervals and extreme estimates for some groups reflect low model power due to small sample sizes. When TSD occurrence was normalized with the number of strandings, there were no significant relationships identified between seasonality and TSD ($p = 0.6$).

3.1.2 | Viral

Herpesvirus was confirmed by PCR of fresh tissue in two cases with oral (23-02012) or genital (24-00224) mucosal lesions from mature, male, *T. aduncus* from West GSV. Papillomavirus testing was not performed. The individual with the genital lesion was in good body condition while the individual with the oral

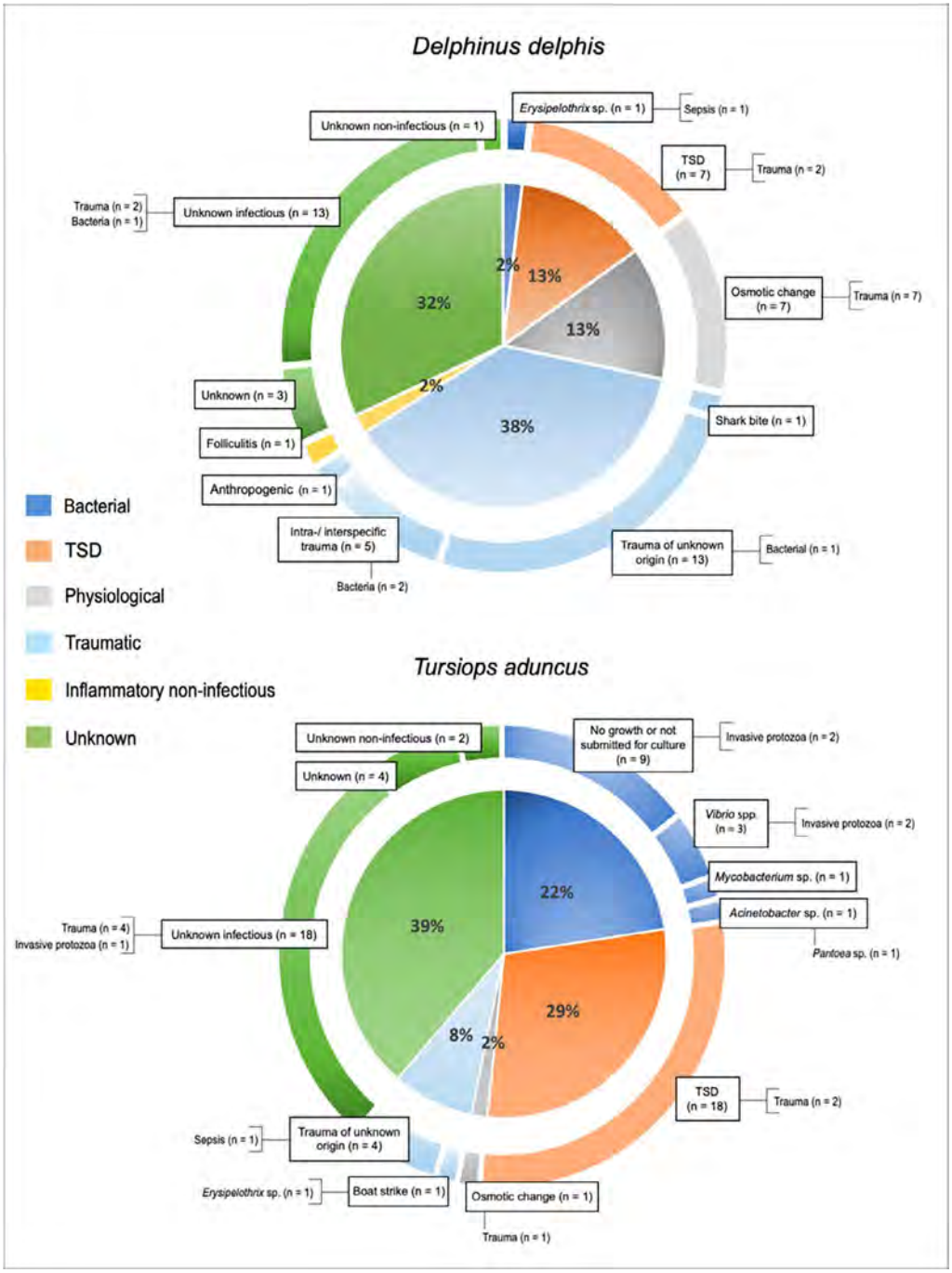

FIGURE 2 | Etiologies of skin lesions ($n = 115$) identified in common dolphins (*Delphinus delphis*) and Indo-Pacific bottlenose (*Tursiops aduncus*) in South Australia between June 2021 and April 2024. Arrows indicate lesions that had concurrent etiologies/comorbidities, which may have influenced disease susceptibility or progression. TSD = Tattoo Skin Disease.

lesion was considered thin. Lesions displayed similar macroscopic findings, presenting as circular multifocal to coalescing, 1–5 mm in diameter pink-white raised nodular plaques (Figure 5). Microscopically, acanthosis, ballooning/hydropic degeneration, blunted/fused rete pegs, submucosal inflammation, and intranuclear inclusion bodies (INIBs) were present in both samples. Hyperkeratosis, spongiosis, necrosis, mucosal inflammation, and congestion were observed in one sample (23-02012). All individuals tested negative for CeMV and Influenza virus (Table S1). No skin lesions were positive for herpesvirus (formalin fixed tissue tested only).

3.1.3 | Bacterial

Lesions of bacterial etiology (i.e., single and/or mixed infections identified through microbiological culture within the context of typical inflammatory lesions +/− direct visualization of bacteria within lesions) were detected in one *D. delphis* and three *T. aduncus* (4/52, 7.7%) and included *Erysipelothrix rhusiopathiae*, *Mycobacterium marinum*, *Vibrio harveyi*, *Vibrio gigantis*, *Acinetobacter lwoffii*, *Pantoea agglomerans*, and undetermined bacteria (not cultured, but histopathological evidence of bacterial etiology) (Figure 2). In the ADS dolphins

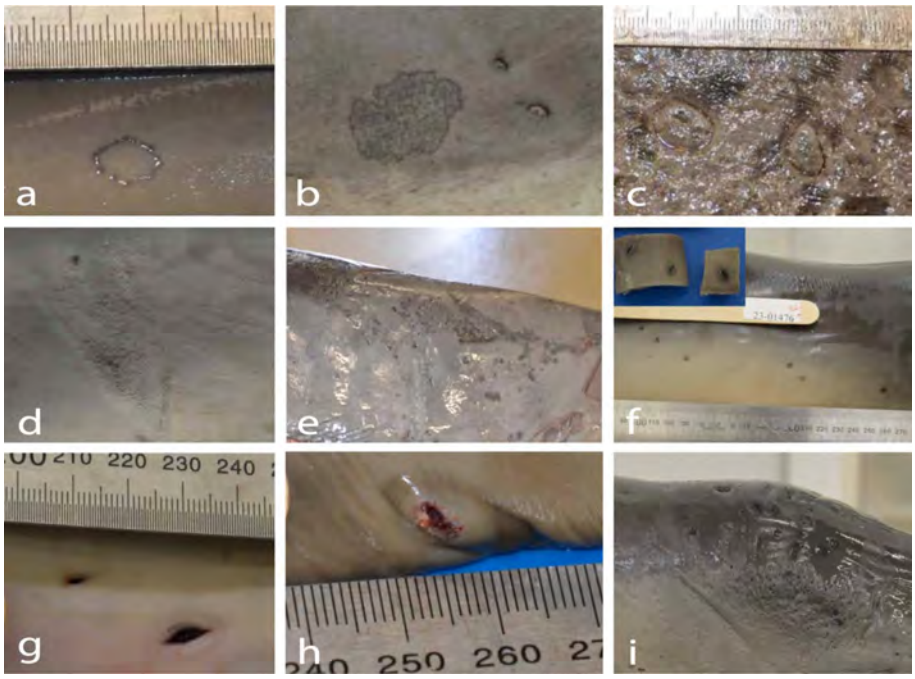

FIGURE 3 | Variation in appearance of individuals with TSD (a–f) and ulcerated lesions displaying histological evidence of intracytoplasmic inclusion bodies (ICIBs) and the presence of one or more characteristic microscopic findings for TSD including acanthosis, ballooning/hydropic degeneration, spongiosis, hyperpigmentation, and blunted/fused rete pegs (g–h). (a, g, h) 22-02828, male, sexually immature, *D. delphis*; (b) 23-02012, male, sexually mature, *T. aduncus*; (c) 24-00224, male, sexually mature, *T. aduncus*; (d) 22-00957, female, sexually immature, *D. delphis*; (e), 9: 23-03314, female, sexually mature, *D. delphis*; f: 23-01476, male, sexually immature, *T. aduncus*; (i): 23-00355, female, sexually immature (late-term aborted fetus), *T. aduncus* with TSD-like lesions and histological findings of hyperpigmentation, blunted/fused rete pegs, and eosinophilic globular material resembling ICIBs within keratinocytes of the stratum spinosum.

examined in this study, 2/5 (40%) were found to have bacterial skin lesions, compared with 1/18 (5.5%) of *T. aduncus* from outside the ADS. One immature female *T. aduncus* from the East GSV region (22-02208) had multifocal cutaneous ulcerations distributed over the head (with histopathological evidence of bacterial infection) and concurrent Toxoplasma encephalitis. An immature male *D. delphis* from west SG (22-01356) had a probable *E. rhusiopathiae* skin lesion based on typical gross and histopathological findings and a positive *E. rhusiopathiae* brain culture with associated sepsis and choroid plexitis/encephalitis. Individuals that displayed more than one bacterial lesion (3/4, 21-02676, 22-01721, 22-02208) were emaciated and considered in poor health. *Mycobacterium marinum* dermatitis was confirmed through positive culture and microscopic visualization of acid-fast intrahistiocytic bacteria in skin lesions (22-01721). This individual died of infectious vestibular disease, resulting in meningoencephalitis and debilitation. Invasive ciliated protozoa were identified as comorbidities in 27% of bacterial lesions (4/15), three of which were from *T. aduncus* within the ADS. Macroscopic appearance varied between characterized bacterial lesions (Figure 6).

3.1.4 | Unknown

Etiology was not determined in skin lesions suspected to be of infectious origin in 17 cases (10 *T. aduncus*, 7 *D. delphis*), accounting for 31/70 (44%) of such lesions. Ulcerated lesions most frequently represented this category (18/31, 62%), followed by flat (5/31, 17%), depressed (4/31, 14%), eroded (1/31, 3%), and pinhole (1/31, 3%) (Table 2). Lesions were most frequently focal (18/31, 62%) but also displayed irregular, locally extensive, multifocal, and multifocal to coalescing distributions. Most frequent microscopic findings were as follows: hyperkeratosis (16/31, 52%), acanthosis (25/31, 81%), ballooning/hydropic degeneration (27/31, 87%), spongiosis (30/31, 97%), hyperpigmentation (18/31, 58%), blunted/fused rete pegs (26/31, 84%), ICIBs (20/31, 65%), congestion (17/31, 55%), and dermal inflammation (18/31, 58%) (Table 2). An etiological agent was not identified in 7/9 (77%) infectious oral mucosal lesions. These lesions presented as either focal ulcerations of the lingual mucosa (3/7, 43%) or hyperkeratosis of the palatine mucosa (4/7, 57%). All of which were negative to pan-herpes PCR of FFPE tissues (see Table S1).

3.2 | Non-Infectious Lesions

Non-infectious skin lesions accounted for 33% of lesions examined (38/115). Lesions reported included traumatic (anthropogenic, non-anthropogenic, and trauma of unknown origin), physiological, and inflammatory non-infectious (Figure 2). One lingual mucosal ulceration was considered traumatic in origin.

3.2.1 | Trauma

A propeller injury was identified in an adult male *T. aduncus* from the West SG region (see Table S1, 22-00878, lesion 2). Grossly, this lesion was characterized by a locally extensive,

TABLE 2 | Descriptors of skin lesions commonly observed in this study (adapted from Segura-Göthlin, Fernández, Arbelo, Andrada Borzollino, et al. 2023).

	Infectious						Non-infectious			
	TSD		Bacterial		Unknown		Traumatic		Physiological	
Descriptor	*D. delphis* $n=7$ Prev. 14%	*T. aduncus* $n=18$ Prev. 43%	*D. delphis* $n=1$ Prev. 3%	*T. aduncus* $n=14$ Prev. 13%	*D. delphis* $n=13$ Prev. 24%	*T. aduncus* $n=18$ Prev. 43%	*D. delphis* $n=20$ Prev. 28%	*T. aduncus* $n=5$ Prev. 22%	*D. delphis* $n=7$ Prev. 3%	*T. aduncus* $n=1$ Prev. 4%
Macroscopic lesion type										
Tattoo	3 (43)	**10 (56)**	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)
Pinhole	1 (14)	1 (6)	0 (0)	0 (0)	0 (0)	1 (6)	0 (0)	0 (0)	0 (0)	0 (0)
Ring	3 (43)	5 (28)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)
Ulcerated/eroded	0 (0)	2 (11)	0 (0)	**13 (93)**	**12 (92)**	7 (44)	**13 (65)**	1 (25)	0 (0)	0 (0)
Pitted/depressed	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	4 (25)	0 (0)	0 (0)	0 (0)	0 (0)
Flat	0 (0)	0 (0)	**1/(100)**	1 (7)	1 (8)	4 (25)	2 (10)	0 (0)	**7 (100)**	**1 (100)**
Raised/elevated	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	1 (25)	0 (0)	0 (0)
Incisional cut/ laceration	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	5 (25)	3 (75)	0 (0)	0 (0)
Microscopic findings										
Mild hyperkeratosis	**4 (57)**	7 (39)	0 (0)	**7 (50)**	**9 (69)**	7 (39)	**10 (50)**	1 (25)	2 (29)	0 (0)
Acanthosis	**6 (86)**	**15 (83)**	**1/(100)**	**11 (79)**	**12 (92)**	**13 (72)**	9 (45)	1 (25)	0 (0)	0 (0)
Ballooning/ hydropic degeneration	**6 (86)**	**11 (61)**	**1/(100)**	**9 (64)**	**13 (100)**	**14 (78)**	**19 (95)**	1 (25)	**7 (100)**	0 (0)
Spongiosis	**7 (100)**	**17 (94)**	**1/(100)**	**14 (100)**	**13 (100)**	**17 (94)**	**19 (95)**	**3 (75)**	**7 (100)**	**1 (100)**
Necrosis	0 (0)	0 (0)	**1/(100)**	6 (43)	0 (0)	2 (11)	2 (10)	0 (0)	0 (0)	0 (0)
Satellitosis	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	1 (6)	0 (0)	0 (0)	0 (0)	0 (0)
Hyperpigmentation	**5 (71)**	**16 (89)**	**1/(100)**	1 (7)	5 (38)	**13 (72)**	**10 (50)**	**2 (50)**	0 (0)	0 (0)
Blunted/fused rete pegs	**4 (57)**	**18 (100)**	**1/(100)**	**9 (64)**	**12 (92)**	**14 (78)**	**10 (50)**	**2 (50)**	2 (29)	0 (0)

(Continues)

TABLE 2 | (Continued)

	Infectious						Non-infectious			
	TSD		Bacterial		Unknown		Traumatic		Physiological	
Descriptor	***D. delphis* $n=7$ Prev. 14%**	***T. aduncus* $n=18$ Prev. 43%**	***D. delphis* $n=1$ Prev. 3%**	***T. aduncus* $n=14$ Prev. 13%**	***D. delphis* $n=13$ Prev. 24%**	***T. aduncus* $n=18$ Prev. 43%**	***D. delphis* $n=20$ Prev. 28%**	***T. aduncus* $n=5$ Prev. 22%**	***D. delphis* $n=7$ Prev. 3%**	***T. aduncus* $n=1$ Prev. 4%**
ICIBs	**7 (100)**	**16 (89)**	0 (0)	0 (0)	**11 (85)**	**9 (50)**	**11 (55)**	1 (25)	0 (0)	0 (0)
Epidermal inflammation	0 (0)	0 (0)	**1/(100)**	6 (43)	2 (15)	3 (17)	5 (25)	1 (25)	0 (0)	0 (0)
Congestion	2 (29)	5 (28)	**1/(100)**	**10 (71)**	**8 (62)**	**9 (50)**	**17 (85)**	**3 (75)**	2 (29)	0 (0)
Dyskeratosis/ apoptosis	0 (0)	0 (0)	0 (0)	0 (0)	1 (8)	3 (17)	1 (5)	0 (0)	0 (0)	0 (0)
Pearl corns	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	1 (6)	2 (10)	0 (0)	0 (0)	0 (0)
Erosion	0 (0)	0 (0)	0 (0)	7 (50)	4 (31)	1 (6)	6 (30)	1 (25)	0 (0)	0 (0)
Ulceration	0 (0)	2 (11)	0 (0)	**12 (86)**	**10 (77)**	3 (17)	**12 (60)**	**2 (50)**	0 (0)	0 (0)
Dermal inflammation	1 (14)	2 (11)	**1/(100)**	**13 (93)**	**8 (62)**	**10 (56)**	**16 (80)**	**3 (75)**	0 (0)	0 (0)
Dermal fibrosis	0 (0)	0 (0)	0 (0)	**12 (86)**	6 (46)	**10 (56)**	6 (30)	1 (25)	0 (0)	0 (0)
Steatitis	0 (0)	0 (0)	0 (0)	5 (36)	4 (31)	1 (6)	4 (20)	1 (25)	0 (0)	0 (0)
Dermal-epidermal separation	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	1 (5)	1 (25)	0 (0)	0 (0)

Note: Most common features ($\geq 50\%$) are highlighted in bold.
Abbreviations: ICIBs = intracytoplasmic inclusion bodies; n = number of lesions examined; Prev. = prevalence; TSD = tattoo skin disease.

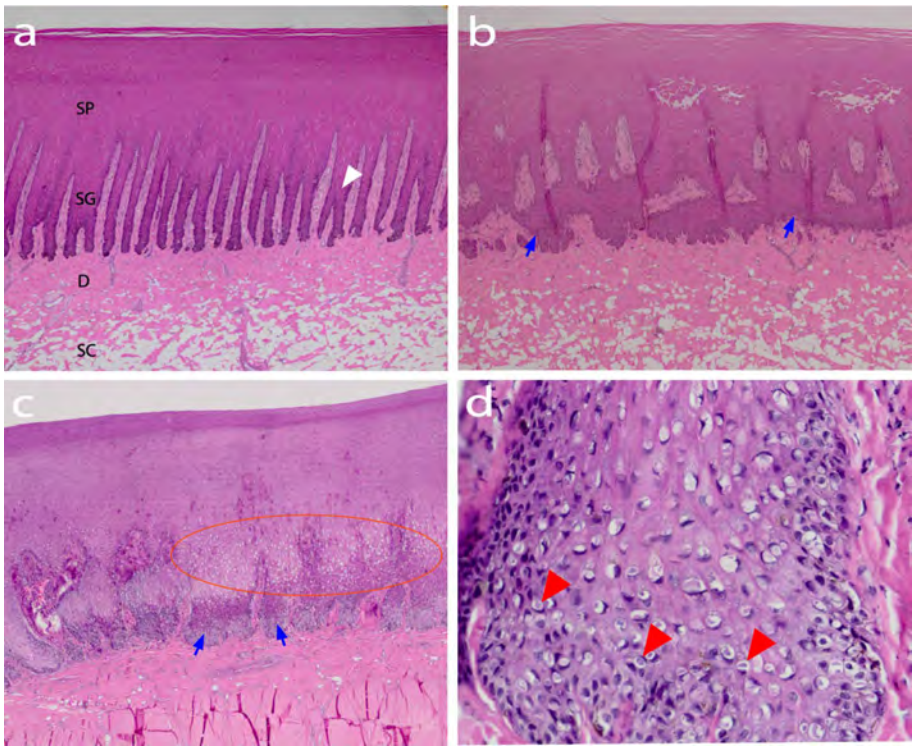

FIGURE 4 | Microscopy of common dolphin (*Delphinus delphis*) skin stained with hematoxylin and eosin. (a) normal skin, SE = stratum externum, SP = stratum spinosum, SG = stratum germinativum, D = dermis, SC = subcutis (blubber layer), white arrowhead = rete pegs; (b) Section of skin from immature male (22-01356) with tattoo skin disease (TSD) displaying hyperkeratosis (yellow arrowhead), ballooning/hydropic degeneration, spongiosis, and blunting and fusion of rete pegs (blue arrow); (c) section of skin from mature female (23-02805) with an ulcerated skin lesion displaying acanthosis, ballooning/hydropic degeneration (orange ellipse), spongiosis, hyperpigmentation, blunted/fused rete pegs (blue arrow), and dermal fibrosis; (d) distal rete peg of 23-02805 displaying abundant intracytoplasmic inclusion bodies (ICIBs) (red arrowheads).

180 mm in length, curvilinear, sharply demarcated, deeply penetrative incisional cut on the peduncle extending into the underlying blubber, muscular, and skeletal tissues. Secondary bacterial colonization (*E. rhusiopathiae*) was confirmed through microbiology of the peduncle wound, heart blood, and brain, and histological evidence of septicaemia. One adult female *D. delphis* had a 300 mm in length, linear incisional cut extending into the underlying subcutis on the melon. This lesion is suspected to be of anthropogenic origin (see Table S1, 23-03314, lesion 8). Shark predation was reported in one male *D. delphis* calf from KI. Grossly, the lesion was extensive and circular, with penetrative incised wounds (see Table S1, 23-01992, lesion 8). The same individual displayed multiple lesions indicative of osmotic disruption (see below in 3.2.2.) (Figure 7). Intra- or interspecific trauma was reported in four lesions, characterized by linear, parallel incisional cuts, consistent with rake marks. Three of these lesions had secondary gram-negative bacterial invasion. Additionally, extensive rake marks were reported in a sexually mature female *D. delphis* from CO. Microscopically, there was basal hyperplasia, blunting of epidermal pegs, and ICIBs within keratinocytes along the rake marks. Rake mark scarring was a common finding in stranded dolphins across all species and age classes and was not included in further analyses. Other presumed traumatic lesions of unknown origin were determined based on the presence of histological features such as ballooning/hydropic degeneration, spongiosis, congestion, erosion or ulceration, and dermal inflammation. One case displayed steatitis and another case was interpreted as a frictional wound, characterized by spongiosis, congestion, dermal inflammation, dermal-epidermal separation, and bulla formation.

3.2.2 | Physiological

Lesions of physiological change or osmotic disruption were reported in two cases with concurrent severe traumatic injuries. One case was an immature male *D. delphis* from KI (23-01992) and the other was an immature male *T. aduncus* from east GSV (22-02189). These lesions were characterized grossly as focal or multifocal to coalescing, indistinctly white fringed, hypopigmented, smooth, flat discs or ellipses, at times forming an irregularly shaped patch (Figure 7). Microscopically, lesions displayed hyperkeratosis (25%), ballooning/hydropic degeneration (88%), spongiosis (100%), blunted/fused rete pegs (25%), and congestion (25%) (Table 2). No inflammation or evidence of infectious agents was seen in tissue sections.

3.2.3 | Inflammatory Non-Infectious

One case of folliculitis was observed in the remnant vibrissae of a male neonate *D. delphis* from EB. Grossly, the lesion was a focal, 7 mm in diameter, flat, gray to black gradient, targetoid with a pitted centre. Microscopically, within the follicular lumen were infiltrates of neutrophils and lesser macrophages with mural exocytosis of low numbers of neutrophils and perifollicular to

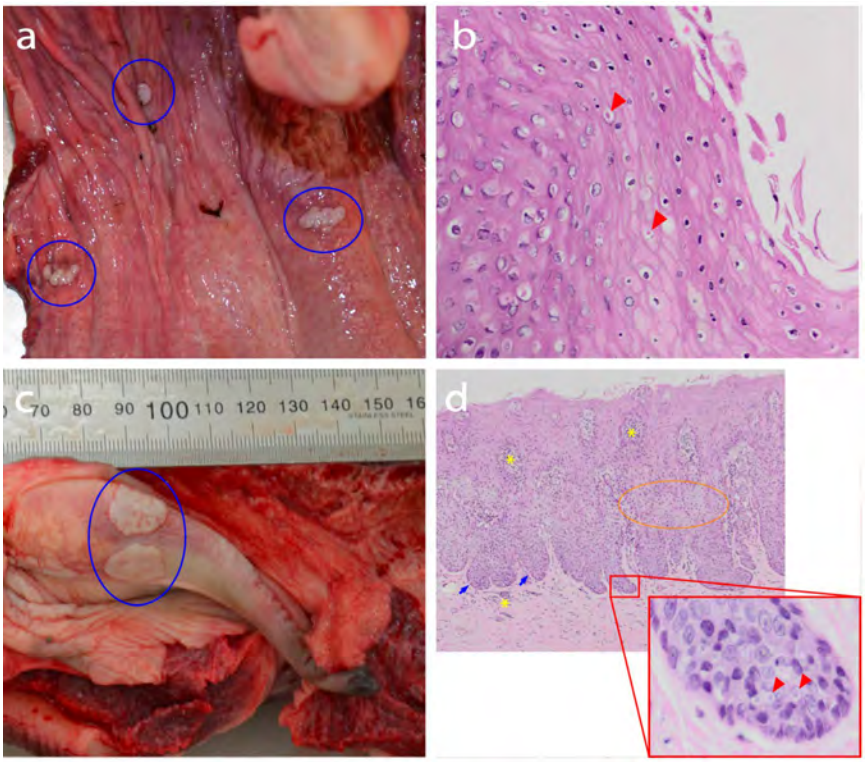

FIGURE 5 | Gross and microscopic findings in orogenital papillomas in Indo-Pacific bottlenose dolphins (*Tursiops aduncus*) stranded in South Australia. Gross lesions are characterized by coalescing, white, nodular to corrugated, hyperplastic raised plaques in the pharyngeal (a) and genital mucosa (c) (blue ellipses). Microscopic features (b, d) include acanthosis, ballooning/hydropic degeneration (orange ellipse), blunted/fused rete pegs (blue arrows), lymphoplasmacytic epidermal and dermal inflammation (yellow asterisk), + intranuclear inclusion bodies (INIBs) (red arrowheads). (a, b) 23-02012, male, sexually mature *T. aduncus*; (c, d) 24-00224, male, sexually mature *T. aduncus*. Lesional tissue was positive by pan-herpesviral PCR. Papillomavirus tests were not performed.

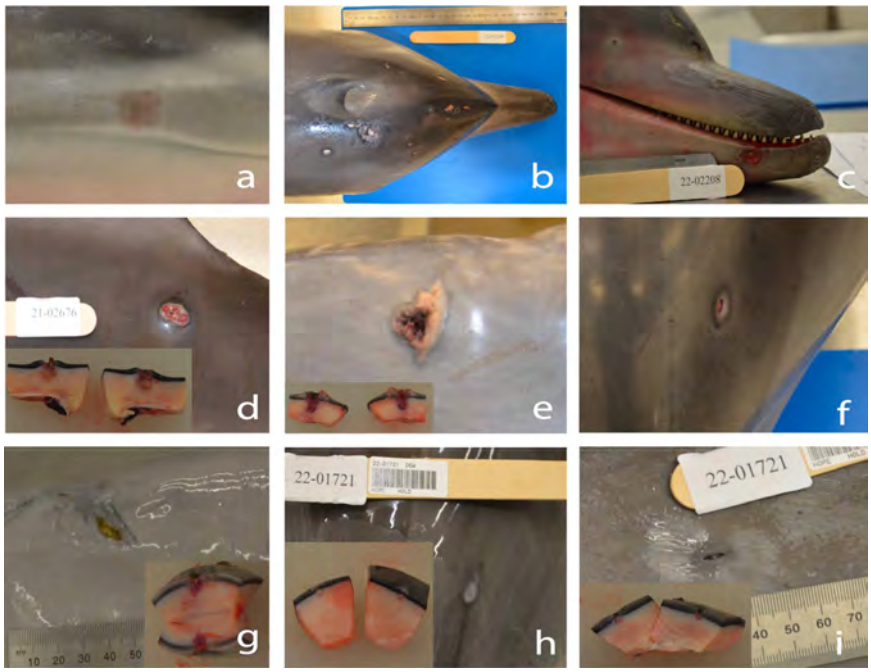

FIGURE 6 | Variation in macroscopic appearance of bacterial lesions determined by microscopy and microbiological culture in some cases. (a) 22-01356, male, sexually immature, *D. delphis*, confirmed *Erysipelothrix rhusiopathiae* sepsis and encephalitis; (b, c) 22-02208, female, sexually immature, *T. aduncus*, concurrent toxoplasma encephalitis, bacterial agent not cultured; (d–f) 21-02676, male, sexually immature, *T. aduncus*, *Vibrio harveyi* cultured from lesions d and e, organism not cultured for lesion f; (g–i) 22-01721, male, sexually immature, *T. aduncus*. *Mycobacterium marinum* confirmed in lesion h, no aerobic or anaerobic growth at 48 h in lesions g and i. Lesions d–i are from ADS resident *T. aduncus*.

FIGURE 7 | Macroscopic appearance of epidermal changes observed in a physiological response to primary traumatic injury (shark bite) in a male, sexually immature common dolphin (*Delphinus delphis*), 23-01992.

interstitial infiltration of lymphocytes, neutrophils, and plasma cells; no infectious agents were seen. The epidermis was otherwise acanthotic, with hyperkeratosis, spongiosis, and mild blunting of rete pegs noted, with no evidence of intracytoplasmic inclusion bodies.

4 | Discussion

Many studies utilize observational techniques to assess skin lesion prevalence and indirectly monitor population health of free-ranging cetaceans (Hupman et al. 2017; Toms et al. 2020; Hawkins et al. 2022). This research methodology has limitations in that only a portion of the animal is observed, and clinical presentations of specific etiologies can vary significantly or share overlapping features, making interpretations difficult. Necropsy investigations of deceased cetaceans, as performed in the present study, allow detailed topographic, macroscopic, and microscopic assessment of lesions, and adequate tissue sampling for ancillary diagnostics, assisting in the determination of causation and helping to refine interpretation of images from observational surveys.

Topographically, the most common locations for skin lesions observed in this study were the head/melon (23%–34%) and peduncle (25%–26%). Due to dolphins generally being partially submerged during field studies of free-ranging individuals, body regions assessed for skin lesion prevalence are usually limited to areas most frequently observed (dorsal fin, flank, peduncle, rostrum, melon, and tail fluke) (Hawkins et al. 2022; Nicholls et al. 2023). The peduncle has been reported as the most common region for skin lesions observed in *D. delphis* in New Zealand (91.1%) (Hupman et al. 2017).

In this study, the prevalence of infectious skin lesions was observed in individuals representing all body conditions; however, for *T. aduncus*, a higher number of infectious skin lesions per individual was observed in emaciated dolphins. This was particularly evident for bacterial lesions where 75% of individuals in this category displayed multiple lesions and were emaciated and in poor health. It is possible that these patterns vary by population, species, and skin lesion etiology (Soares et al. 2022). The results from the present study support the use of observational surveys as a valid tool for assessing skin lesion prevalence in free-ranging dolphins and may serve to provide a reliable indicator for population and environmental health; however, observational studies are limited in that etiology cannot be investigated.

TSD was identified in 27% of dolphins examined across a broad geographic distribution. Polymerase Chain Reaction and sequencing have shown that TSD is caused by different strains of cetacean poxvirus (Van Bressem et al. 1993; Barnett et al. 2015; St. Leger et al. 2018; Rodrigues et al. 2020; Luciani et al. 2022). ICIBs were observed in 92% (23/25) of TSD lesions, suggesting poxviral involvement; however, without molecular testing or electron microscopy, etiology cannot be confirmed. Prevalence of TSD was significantly higher in *T. aduncus*. South Australia *T. aduncus* display coastal residency (Cribb et al. 2013; Zanardo et al. 2016) and are likely exposed to an increased number of stressors such as wastewater, stormwater discharge, and anthropogenic pressures. TSD is considered a health indicator in dolphins, with prevalence reported to increase with poor health (Van Bressem, Van Waerebeek, et al. 2009) and during periods of environmental disturbance (e.g., heavy rainfall in estuarine environments) (Fury and Reif 2012). Infection has also been reported with coinfections (Groch et al. 2020; Segura-Göthlin, Fernández, Arbelo, Andrada Borzollino, et al. 2023; Stephens

et al. 2014), which may suggest an immune compromised state. However, there were no significant relationships observed between TSD prevalence and BCS, health status, or season in the present study.

There were no significant relationships observed between TSD and sex or sexual maturity. Sex has not previously been associated with TSD in other studies (except in Peruvian Burmeister's porpoise, *Phocoena spinipinnis*) (Van Bressem and Van Waerebeek 1996; Van Bressem, Van Waerebeek, et al. 2009; Powell et al. 2018); however, in captivity, male *T. truncatus* appear more vulnerable to infection (Van Bressem et al. 2018). The effect of age on TSD prevalence differs between free-ranging populations, possibly due to different age categorization techniques among studies. In some populations, TSD prevalence has been reported to be higher in mature animals, consistent with poor health (Van Bressem, Van Waerebeek, et al. 2009) or sexual maturity increasing sociality and contact with infected individuals (Wearmouth and Sims 2008; Fury et al. 2013; Powell et al. 2020). Several studies have linked reduced TSD prevalence in young calves to protection conferred by maternal antibodies (Van Bressem and Van Waerebeek 1996; Van Bressem, Van Waerebeek, et al. 2009; Barnett et al. 2015; Powell et al. 2018; Van Bressem et al. 2018). However, the duration of antibody-mediated protection following initial infection remains unclear (Powell et al. 2018). TSD could infer disease in naïve individuals in the absence of maternal antibodies (Van Bressem et al. 1999; Powell et al. 2018). Four pre-weaned individuals (<1.5 m in length for *T. aduncus* and <1.3 m for *D. delphis*) in this study displayed TSD (22-00957, 22-01356, 22-02820, 23-01476), one of which displayed chronic disease of undetermined origin and another with systemic inflammation and sepsis. A late-term aborted fetus (23-00355) displayed TSD-like lesions along the dorsum (Figure 3i), with microscopic findings including hyperpigmentation, blunted/fused rete pegs, and eosinophilic globular material resembling ICIBs within keratinocytes of the stratum spinosum. Unlike CeMV (Van Bressem et al. 2014), evidence of vertical transmission has not yet been reported for poxvirus in cetaceans, and further investigations with ancillary diagnostics are required to confirm or refute poxviral involvement in this lesion.

Skin lesions suspected to be of infectious origin but with undetermined etiology accounted for 27% of skin lesions. Common microscopic findings were similar to those seen with TSD, including the presence of ICIBs; however, many of these lesions were more severe, displaying congestion, dermal inflammation, and dermal fibrosis. Although TSD is not usually associated with marked adverse health outcomes, extensive lesions and fatal outcomes have been reported (Geraci et al. 1979; Duignan et al. 2018). Ulcerative skin lesions of harbor porpoises (*Phocoena phocoena*) with molecular investigations indicative of poxvirus have been reported previously in captive conditions (van Elk et al. 2000). Chronic TSD can progress to pitting, disrupted epithelium, and opportunistic pathogen invasion (Geraci et al. 1979; Duignan et al. 2018; Van Bressem et al. 2022). TSD lesions with central pitting were observed in this study, and eight dolphins with TSD displayed ulcerated lesions of unknown etiology (suspected infectious based on microscopy). Three individuals with TSD had concurrent traumatic lesions, and ICIBs were observed in the stratum germinativum in several traumatic skin lesions not typical grossly for TSD. This suggests a route for viral transmission (i.e., through bites or a disrupted epidermis).

The oral and genital papillomas in this study were positive for herpesvirus (HV). HV has previously been unreported in dolphins from Australia (Souter et al. 2024). Genital and oropharyngeal plaques in dolphins have been associated with *Gammaherpesvirinae*, which do not cause systemic disease and are suggested to spread by sexual contact (Benson et al. 2006; van Elk et al. 2009; Ewing et al. 2020; Townsend and Staggs 2020). The contribution of papillomavirus (PV) infection was not investigated here. At present, the question as to whether HVs or PVs are causative for orogenital papillomas in dolphins is unresolved (Van Bressem et al. 1996; Bossart et al. 2005; Benson et al. 2006; Van Bressem and Raga 2011; Rehtanz et al. 2012; Duignan et al. 2018) and is an area of ongoing research (Horowitz et al. 2023). HV and PV coinfection in genital papillomas has been reported in *T. truncatus* (Rehtanz et al. 2012; Cruz et al. 2014). PV-related disease in dolphins has not been confirmed in Australia; however, respiratory, oral, anal, and urogenital papillomatosis (without a confirmed etiological agent) have been reported in a *T. aduncus* from the ADS (Byard et al. 2010). All skin lesions tested in this study were negative for HV; however, these samples were all formalin-preserved, possibly limiting the reliability of testing due to nucleic acid degradation (Koshiba et al. 1993). The absence of targeted molecular testing in fresh skin lesion samples is a limitation of this study, and HV prevalence is possibly underestimated. Further molecular and sequencing investigations into HV prevalence in skin lesions would be useful to improve our understanding of HV impact in SA dolphins.

Bacterial skin lesions were observed in 7.7% of dolphins examined in this study and displayed varying clinical presentations. One juvenile female *T. aduncus* (22-02208) exhibited multifocal cutaneous ulcerations over the head and rostrum and concurrent protozoal encephalitis. Due to being frozen prior to necropsy, bacterial cultures were not performed on this individual; however, similar pathological findings were reported in a deceased *D. delphis* calf that died of *Streptococcus iniae* sepsis in SA (Souter et al. 2021). *E. rhusiopathiae* was considered the most likely etiological agent of a flat, rhomboid erythematous skin lesion in a *D. delphis* calf (22-01356) displaying a marked neutrophilic vasculitis. A heavy *E. rhusiopathiae* culture was obtained from the brain and lesions in other organs indicated sepsis and encephalitis. This individual was in good body condition, suggesting an acute process. *E. rhusiopathiae* is a known causative agent of skin disease in free-ranging and captive dolphins (Melero et al. 2011; Townsend and Staggs 2020; Sacristán et al. 2022), as well as systemic disease without skin lesions (Díaz-Delgado et al. 2015; Lee et al. 2022; Danil et al. 2023), but had not yet been reported as a cause for skin lesions in dolphins from Australia.

The highest prevalence of bacterial skin lesions was in ADS dolphins. Two ADS residents with multifocal bacterial lesions displayed similar pathological findings of otitis media and debilitation (see Table S1). The ADS residents are exposed to a wide range of stressors including boat traffic, anthropogenic pollutants, and environmental degradation (Bossley et al. 2017; Kirkwood et al. 2022). Epidermal microbial communities play a

crucial role in skin immunity, and disruptions to these communities can occur from adverse environmental conditions or an immunocompromised host, resulting in colonization by pathogenic organisms (Hargis and Sherry 2017; Duignan et al. 2020). *Mycobacterium marinum* was cultured from a dermal abscess of an ADS resident male. To the authors' knowledge, this is the first detection of this pathogen in cutaneous lesions in free-ranging cetaceans (Bowenkamp et al. 2001; Van Bressem et al. 2008). *M. marinum* is a waterborne pathogen known to infect humans and fish, often colonizing secondary to initial trauma (Petrini 2006; Hashish et al. 2018). Similarly, *Vibrio* spp. is an opportunistic aquatic bacterium known to be pathogenic in marine fish and invertebrates (Van Bressem et al. 2008; Zhang et al. 2020). Additionally, other bacterial organisms cultured from ADS resident dolphin skin lesions included *Acinetobacter lwoffii* and *Pantoea agglomerans*, both of which are considered opportunistic (Regalado et al. 2009; Dutkiewicz et al. 2016; Wan et al. 2023).

Marine mammals have a known tendency to accumulate toxic anthropogenic contaminants, some of which are immunomodulatory (Fair et al. 2013; Desforges et al. 2016). The Port River Estuary (within the ADS) is a known hotspot for contamination from industry, landfill, wastewater, and stormwater outflows. High levels of per- and polyfluorinated alkyl substances (PFAS), metals, pharmaceutical agents, and organochlorines have previously been reported in dolphins, fish, and environmental samples from this region (Butterfield and Gaylard 2005; Gaylard 2017; Weijs et al. 2020; Souter et al. 2025 in review). These contaminants are usually accumulated through dietary sources and undergo biomagnification in dolphins, resulting in elevated concentrations in tissues (Foord et al. 2023). The extent to which anthropogenic pollutants are affecting dolphin health is still being studied, in addition to the synergistic effects of multiple contaminants, infectious disease, and stress (Marsili et al. 2019).

Non-infectious etiologies contributed to 33% of examined skin lesions. Anthropogenic injuries are well documented in dolphins (Moore et al. 2013; Correia et al. 2023). One bottlenose dolphin from SG had a peduncle injury consistent with a propeller wound. This animal succumbed to a secondary *E. rhusiopathiae* septicaemia. Traumatic injuries provide an entry for pathogenic microorganisms and subsequent septic infections (Hargis and Sherry 2017). Two cases with severe traumatic injuries, including a shark bite injury, displayed features of epidermal oedema at other sites of the body. These changes are consistent with altered osmolality due to loss of epidermal contiguity (Deming et al. 2020; Duignan et al. 2020). In the case of the shark bite wound, the animal was observed alive for a period prior to euthanasia, allowing time for these physiological changes to occur.

5 | Conclusion

This pathological investigation of skin and mucosal lesions in dolphins from South Australian waters includes individuals that died from acute (e.g., anthropogenic trauma) and chronic (e.g., infectious disease) mortality causes, representing both healthy and unhealthy populations. This study documents a diversity of skin lesions, reports herpersvirus, *Erysipelothrix rhusiopathiae*, and *Mycobacterium marinum* infection in free-ranging dolphins from Australian temperate waters, and supports traumatic injury as an entry point for pathogenic organisms and altered osmolality due to loss of epidermal contiguity. Furthermore, findings are suggestive of adverse impacts associated with residence in a heavily urbanized environment (the Adelaide Dolphin Sanctuary) on dolphin epidermal health and immunity, with a higher prevalence of opportunistic bacterial infections observed in dolphins of this region. These results improve our understanding of skin lesions in *T. aduncus* and *D. delphis*, provide a catalogue to assist in determining differential diagnoses, and support the use of observational surveys to assess dolphin and environmental health. Future studies on skin lesions in South Australian dolphins should focus on molecular investigations and phylogenetic analysis of the poxviruses causing Tattoo Skin Disease, as well as investigations on potential disease progression and comorbidities. Furthermore, investigations into the effects of confounding factors (e.g., toxicological, environmental, immunological) on dolphin skin health are necessary. Small sample sizes limited the statistical power of this study, and future investigations would benefit from collecting a larger number of samples and data over a longer period.

Author Contributions

Rebecca Souter: conceptualisation, data curation, formal analysis, investigation, methodology, validation, visualization, software, writing – original draft, writing – review and editing. **Anne-Lise Chaber:** data curation, funding acquisition, investigation, supervision, writing – review and editing. **Luciana Möller:** conceptualisation, data curation, funding acquisition, investigation, supervision, writing – review and editing. **Ikuko Tomo:** data curation, investigation, writing – review and editing. **Lucy Woolford:** conceptualization, data curation, funding acquisition, investigation, methodology, project administration, resources, supervision, visualization, writing – review and editing.

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Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability Statement

The data that supports the findings of this study is available in Supporting Information of this article.

References

Adamczak, S. K., C. Kemper, and I. Tomo. 2018. "Strandings of Dolphins in the Adelaide Dolphin Sanctuary, South Australia." *Journal of Cetacean Research and Management* 19: 105–111. https://doi.org/10.47536/jcrm.v19i1.418.

Barnett, J., A. Dastjerdi, N. Davison, et al. 2015. "Identification of Novel Cetacean Poxviruses in Cetaceans Stranded in South West England." *PLoS One* 10: e0124315. https://doi.org/10.1371/journal.pone.0124315.

Benson, K. A. S., C. A. Manire, R. Y. Ewing, et al. 2006. "Identification of Novel Alpha-and Gammaherpesviruses From Cutaneous and Mucosal Lesions of Dolphins and Whales." *Journal of Virological Methods* 136, no. 1–2: 261–266. https://doi.org/10.1016/j.jviromet.2006.03.033.

Blacklaws, B. A., A. M. Gajda, S. Tippelt, et al. 2013. "Molecular Characterization of Poxviruses Associated With Tattoo Skin Lesions in UK Cetaceans." *PLoS One* 8: e71734. https://doi.org/10.1371/journal.pone.0071734.

Bossart, G. D., S.-J. Ghim, M. Rehtanz, and J. Goldstein. 2005. "Orogenital Neoplasia in Atlantic Bottlenose Dolphins (*Tursiops truncatus*)." *Aquatic Mammals* 31: 473–480.

Bossley, M. I., A. Steiner, R. W. Rankin, and L. Bejder. 2017. "A Long-Term Study of Bottlenose Dolphins (*Tursiops aduncus*) in an Australian Industrial Estuary: Increased Sightings Associated With Environmental Improvements." *Marine Mammal Science* 33, no. 1: 277–290. https://doi.org/10.1111/mms.12368.

Bossley, M. I., and M. A. Woolfall. 2014. "Recovery From Severe Cutaneous Injury in Two Free Ranging Bottlenose Dolphins (*Tursiops* spp.)." *Journal of Marine Animals and Their Ecology* 7, no. 1: 12–16.

Bowenkamp, K. E., J. S. Frasca, A. Draghi, et al. 2001. "*Mycobacterium marinum* Dermatitis and Panniculitis With Chronic Pleuritis in a Captive White Whale (*Delphinapterus leucas*) With Aortic Rupture." *Journal of Veterinary Diagnostic Investigation* 13, no. 6: 524–530. https://doi.org/10.1177/104063870101300613.

Butterfield, N., and S. Gaylard. 2005. "The Heavy Metal Status of South Australian Dolphins, Environment Protection Authority." https://www.epa.sa.gov.au/files/8556_heavy_dolphins.pdf.

Byard, R. W., A. Machado, L. Woolford, and W. Boardman. 2013. "Symmetry: the Key to Diagnosing Propeller Strike Injuries in Sea Mammals." *Forensic Science, Medicine, and Pathology* 9, no. 1: 103–105. https://doi.org/10.1007/s12024-012-9335-0.

Byard, R. W., I. Tomo, C. M. Kemper, et al. 2010. "Unusual Causes of Fatal Upperaerodigestive Tract Obstruction in Wild Bottlenose Dolphins (*Tursiops aduncus*)." *Forensic Science, Medicine, and Pathology* 6: 207–210.

Committee on Taxonomy. 2024. "List of Marine Mammal Species and Subspecies. Society for Marine Mammalogy." www.marinemammalscience.org.

Correia, A. M., E. Dietterle, A. Dinis, and F. Alves. 2023. "Defining a Common Language to Assess External Deformities in Free-Ranging Cetaceans." *Mammal Review* 53, no. 3: 189–205. https://doi.org/10.1111/mam.12318.

Cozzi, B., S. Huggenberger, and H. A. Oelschläger. 2016. *Anatomy of Dolphins: Insights Into Body Structure and Function, Chapter 2 – General Appearance and Hydrodynamics (Including Skin Anatomy)*. Academic Press.

Cribb, N., C. Miller, and L. Seuront. 2013. "Indo-Pacific Bottlenose Dolphin (*Tursiops aduncus*) Habitat Preference in a Heterogeneous, Urban, Coastal Environment." *Aquatic Biosystems* 9, no. 1: 3. https://doi.org/10.1186/2046-9063-9-3.

Croft, L. A., R. Laughlin, M. Manley, and H. H. Nollens. 2020. "Water Temperature Fluctuations as a Key Driver of Cetacean Pox (Tattoo) Lesions in Bottlenose Dolphins *Tursiops truncatus*." *Diseases of Aquatic Organisms* 139: 69–79. https://doi.org/10.3354/dao03472.

Cruz, D., M. Rodríquez, V. Kouri, et al. 2014. "Concurrent Papillomavirus-and Herpesvirus-Infection in Atlantic Bottlenose Dolphins (*Tursiops truncatus*) Inhabiting the Cuban Coast." *Marine Mammal Science* 30, no. 4: 1285–1600. https://doi.org/10.1111/mms.12124.

Danil, K., K. M. Colegrove, M. A. Delaney, A. Mena, N. Stedman, and E. Wurster. 2023. "Systemic Erysipelas Outbreak Among Free-Ranging Bottlenose Dolphins, San Diego, California, USA, 2022." *Emerging Infectious Diseases* 29, no. 12: 2561–2563. https://doi.org/10.3201/eid2912.230811.

Deming, A. C., N. L. Wingers, D. P. Moore, et al. 2020. "Health Impacts and Recovery From Prolonged Freshwater Exposure in a Common Bottlenose Dolphin (*Tursiops truncatus*)." *Frontiers in Veterinary Science* 7: 235. https://doi.org/10.3389/fvets.2020.00235.

Department for Envrionment and Heritage (DEH). 2008. *Adelaide Dolphin Sanctuary Management Plan 2008*. Government of South Australia. https://cdn.environment.sa.gov.au/environment/docs/consgen-dolphinsanctmanagplan.pdf.

Department for Envrionment and Water (DEW). 2022. *Investigation Into Dolphin Deaths in the Adelaide Dolphin Sanctuary 2022*. Government of South Australia. https://cdn.environment.sa.gov.au/environment/images/927956-NPWS-Investigation-into-Dolphins-Deaths-Report-WEB-Version-v2.pdf.

Desforges, J.-P. W., C. Sonne, M. Levin, U. Siebert, S. De Guise, and R. Dietz. 2016. "Immunotoxic Effects of Environmental Pollutants in Marine Mammals." *Environment International* 86: 126–139. https://doi.org/10.1016/j.envint.2015.10.007.

Díaz-Delgado, J., M. Arbelo, E. Sierra, et al. 2015. "Fatal *Erysipelothrix rhusiopathiae* Septicemia in Two Atlantic Dolphins (*Stenella Frontalis* and *Tursiops truncatus*)." *Diseases of Aquatic Organisms* 116, no. 1: 75–81. https://doi.org/10.3354/dao02900.

Duignan, P. J., N. S. Stephens, and K. Robb. 2020. "Fresh Water Skin Disease in Dolphins: a Case Definition Based on Pathology and Environmental Factors in Australia." *Scientific Reports* 10, no. 1: 21979. https://doi.org/10.1038/s41598-020-78858-2.

Duignan, P. J., M.-F. Van Bressem, G. Cortés-Hinojosa, and S. Kennedy-Stoskopf. 2018. "Viruses of Marine Mammals." In *CRC Handbook of Marine Mammal Medicine: Health, Disease, and Rehabilitation, Third Edition*, edited by L. A. Dierauf and F. M. D. Gulland. Taylor and Francis Publishers.

Dutkiewicz, J., B. Mackiewicz, M. K. Lemieszek, M. Golec, and J. Milanowski. 2016. "*Pantoea agglomerans*: a Mysterious Bacterium of Evil and Good. Part III. Deleterious Effects: Infections of Humans, Animals and Plants." *Annals of Agricultural and Environmental Medicine* 23, no. 2: 197–205. https://doi.org/10.5604/12321966.1203878.

Edyvane, K. 1999. "Conserving Marine Biodiversity in South Australia-Part 2-Identification of Areas of High Conservation Value in South Australia." *Aquatic Science Centre, Adelaide* 39: 281.

Espregueira Themudo, G., L. Q. Alves, A. M. Machado, et al. 2020. "Losing Genes: the Evolutionary Remodeling of cetacea Skin." *Frontiers in Marine Science* 7: 592375. https://doi.org/10.3389/fmars.2020.592375.

Ewing, R. Y., D. S. Rotstein, W. A. Mclellan, et al. 2020. "Macroscopic and Histopathologic Findings From a Mass Stranding of Rough-Toothed Dolphins (*Steno bredanensis*) in 2005 on Marathon Key, Florida, USA." *Frontiers in Veterinary Science* 7: 572. https://doi.org/10.3389/fvets.2020.00572.

Ewing, R. Y., M. N. Sutton, H. M. Herring, et al. 2023. "Standardizing Gross Descriptions of Skin Lesions in Common Bottlenose Dolphins (*Tursiops truncatus*) Stranded in Southwest Florida, 2015–2019." *Frontiers in Marine Science* 10: 1269075. https://doi.org/10.3389/fmars.2023.1269075.

Fair, P. A., T. Romano, A. M. Schaefer, et al. 2013. "Associations Between Perfluoroalkyl Compounds and Immune and Clinical Chemistry Parameters in Highly Exposed Bottlenose Dolphins (*Tursiops truncatus*)." *Environmental Toxicology and Chemistry* 32, no. 4: 736–746. https://doi.org/10.1002/etc.2122.

Filby, N. E., M. Bossley, and K. A. Stockin. 2013. "Behaviour of Free-Ranging Short-Beaked Common Dolphins (*Delphinus delphis*) in Gulf St Vincent, South Australia." *Australian Journal of Zoology* 61, no. 4: 291–300. https://doi.org/10.1071/ZO12033.

Foord, C. S., D. Szabo, K. Robb, B. O. Clarke, and D. Nugegoda. 2023. "Hepatic Concentrations of Per-and Polyfluoroalkyl Substances (PFAS) in Dolphins From South-East Australia: Highest Reported Globally." *Science of the Total Environment* 908: 168438. https://doi.org/10.1016/j.scitotenv.2023.168438.

Fury, C. A., and J. S. Reif. 2012. "Incidence of Poxvirus-Like Lesions in Two Estuarine Dolphin Populations in Australia: Links to Flood Events." *Science of the Total Environment* 416: 536–540. https://doi.org/10.1016/j.scitotenv.2011.11.056.

Fury, C. A., K. E. Ruckstuhl, and P. L. Harrison. 2013. "Spatial and Social Sexual Segregation Patterns in Indo-Pacific Bottlenose Dolphins (*Tursiops aduncus*)." *PLoS One* 8, no. 1: e52987. https://doi.org/10.1371/journal.pone.0052987.

Gaylard, S. 2017. *Per and Polyfluorinated Alkyl Substances (PFAS) in the Marine Environment – Preliminary Ecological Findings*. Adelaide: Environmental Protection Authority. https://www.epa.sa.gov.au/files/12580_report_pfas_marine.pdf.

Geraci, J., B. Hicks, and D. St Aubin. 1979. "Dolphin Pox: a Skin Disease of Cetaceans." *Canadian Journal of Comparative Medicine* 43, no. 4: 399–404.

Geraci, J. R., and V. J. Lounsbury. 2005. *Marine Mammals Ashore: a Field Guide for Strandings*. National Aquarium in Baltimore.

Groch, K. R., J. Díaz-Delgado, E. B. Santos-Neto, et al. 2020. "The Pathology of Cetacean Morbillivirus Infection and Comorbidities in Guiana Dolphins During an Unusual Mortality Event (Brazil, 2017–2018)." *Veterinary Pathology* 57, no. 6: 845–857. https://doi.org/10.1177/0300985820954550.

Haigh, K., G. Parra, L. Möller, A. Steiner, and M. Bossley. 2025. "Long-Term Demographic Trends of Near Threatened Coastal Dolphins Living in an Urban Estuary." *Ecology and Evolution* 15, no. 1: e70834. https://doi.org/10.1002/ece3.70834.

Hargis, A. M., and M. Sherry. 2017. "Chapter 17 - The Integument." In *Pathologic Basis of Veterinary Disease*, edited by J. F. Zachary, 6th ed., 1009–1146. Mosby.

Hart, L. B., D. S. Rotstein, R. S. Wells, et al. 2012. "Skin Lesions on Common Bottlenose Dolphins (*Tursiops truncatus*) From Three Sites in the Northwest Atlantic, USA." *PLoS One* 7, no. 3: e33081. https://doi.org/10.1371/journal.pone.0033081.

Hashish, E., A. Merwad, S. Elgaml, et al. 2018. "*Mycobacterium marinum* Infection in Fish and Man: Epidemiology, Pathophysiology and Management; a Review." *Veterinary Quarterly* 38, no. 1: 35–46. https://doi.org/10.1080/01652176.2018.1447171.

Hawkins, E. R., M. Gustavsson, L. Pogson-Manning, H. Pheloung, and C. Jaehnichen. 2022. "Prevalence of Skin Lesions and Injuries in Australian Humpback Dolphins (*Sousa sahulensis*) and Indo-Pacific Bottlenose Dolphins (*Tursiops aduncus*) in Moreton Bay, Queensland." *Aquatic Mammals* 48, no. 4: 297–313. https://doi.org/10.1578/AM.48.4.2022.297.

Horowitz, M. L. F., H. Kurtis H, C. A. Pelton, et al. 2023. *Determining the Viral Etiology of Orogenital Papillomatosis and Squamous Cell Carcinoma in Bottlenose Dolphins (Tursiops truncatus): Diagnostic Advancements Through Deep Sequencing and the Development of Immortalized Dolphin Cell Lines*. IAAAM.

Hupman, K. E., M. D. Pawley, C. Lea, et al. 2017. "Viability of Photo-Identification as a Tool to Examine the Prevalence of Lesions on Free-Ranging Common Dolphins (*Delphinus* sp.)." *Aquatic Mammals* 43, no. 3: 264–278. https://doi.org/10.1578/AM.43.3.2017.264.

Joblon, M. J., M. A. Pokras, B. Morse, et al. 2014. "Body Condition Scoring System for Delphinids Based on Short-Beaked Common Dolphins (*Delphinus delphis*)." *Journal of Marine Animals and Their Ecology* 7, no. 2: 5–13.

Kemper, C., M. Bossley, and P. Shaughnessy. 2008. "Marine mammals of Gulf St. Vincent, Investigator Strait and Backstairs Passage." In *Natural History of Gulf St Vincent*, edited by S. A. Shepherd, S. Bryars, I. Kirkegaard, P. Harbison, and J. T. Jennings, 339–352. Royal Society of South Australia.

Kemper, C., and S. Gibbs. 2001. "Dolphin Interactions With Tuna Feedlots at Port Lincoln, South Australia and Recommendations for Minimising Entanglements." *Journal of Cetacean Research and Management* 3: 283–292.

Kemper, C., M. Talamonti, M. Bossley, and A. Steiner. 2019. "Sexual Maturity and Estimated Fecundity in Female Indo-Pacific Bottlenose Dolphins (*Tursiops aduncus*) From South Australia: Combining Field Observations and Postmortem Results." *Marine Mammal Science* 35, no. 1: 40–57. https://doi.org/10.1111/mms.12509.

Kemper, C., I. Tomo, J. Bingham, et al. 2016. "Morbillivirus-Associated Unusual Mortality Event in South Australian Bottlenose Dolphins Is Largest Reported for the Southern Hemisphere." *Royal Society Open Science* 3: 160838. https://doi.org/10.1098/rsos.160838.

Kemper, C., I. Tomo, G. Bovari, D. Hamer, S. Gibbs, and T. S. Fellowes. 2023. "Characterising Injuries and Pathologies of Common Dolphin *Delphinus delphis* Mortalities in the South Australian Sardine Fishery." *Diseases of Aquatic Organisms* 156: 99–114. https://doi.org/10.3354/dao03765.

Kemper, C. M. 2004. "Osteological Variation and Taxonomic Affinities of Bottlenose Dolphins, *Tursiops* spp., From South Australia." *Australian Journal of Zoology* 52, no. 1: 29–48. https://doi.org/10.1071/ZO03026.

Kemper, C. M., E. Trentin, and I. Tomo. 2014. "Sexual Maturity in Male Indo-Pacific Bottlenose Dolphins (*Tursiops aduncus*): Evidence for Regressed/Pathological Adults." *Journal of Mammalogy* 95, no. 2: 357–368. https://doi.org/10.1644/13-MAMM-A-007.1.

Kirkwood, R., M. Deveney, and S. Goldsworthy. 2022. "Review of Threats to Dolphin Health in the Adelaide Dolphin Sanctuary: A Report to the Department for Environment and Water."

Koshiba, M., K. Ogawa, S. Hamazaki, T. Sugiyama, O. Ogawa, and T. Kitajima. 1993. "The Effect of Formalin Fixation on DNA and the Extraction of High-Molecular-Weight DNA From Fixed and Embedded Tissues." *Pathology Research and Practice* 189: 66–72. https://doi.org/10.1016/S0344-0338(11)80118-4.

Lee, K., S. Y. Park, H. W. Seo, et al. 2022. "Pathological and Genomic Findings of *Erysipelothrix rhusiopathiae* Isolated From a Free-Ranging Rough-Toothed Dolphin *Steno bredanensis* (Cetacea: Delphinidae) Stranded in Korea." *Frontiers in Veterinary Science* 9: 774836. https://doi.org/10.3389/fvets.2022.774836.

Lenth, R. 2024. "Emmeans: Estimated Marginal Means, aka Least-Squares Means. R Package Version 1.10.0." https://cran.r-project.org/package=emmeans.

Lopes-Marques, M., A. M. Machado, L. Q. Alves, et al. 2019. "Complete Inactivation of Sebum-Producing Genes Parallels the Loss of Sebaceous Glands in Cetacea." *Molecular Biology and Evolution* 36, no. 6: 1270–1280. https://doi.org/10.1093/molbev/msz068.

Luciani, L., G. Piorkowski, X. De Lamballerie, K. Van Waerebeek, and M.-F. Van Bressem. 2022. "Detection of Cetacean Poxvirus in Peruvian Common Bottlenose Dolphins (*Tursiops truncatus*) Using a Pan-Poxvirus PCR." *Viruses* 14, no. 9: 1850. https://doi.org/10.3390/v14091850.

Marsili, L., G. Di Guardo, S. Mazzariol, and S. Casini. 2019. "Insights Into Cetacean Immunology: Do Ecological and Biological Factors Make the Difference?" *Frontiers in Immunology* 10: 1219. https://doi.org/10.3389/fimmu.2019.01219.

Melero, M., C. Rubio-Guerri, J. L. Crespo, et al. 2011. "First Case of Erysipelas in a Free-Ranging Bottlenose Dolphin (*Tursiops truncatus*) Stranded in the Mediterranean Sea." *Diseases of Aquatic Organisms* 97: 167–170.

Menon, G. K., P. M. Elias, J. S. Wakefield, and D. Crumrine. 2022. "Cetacean Epidermal Specialization: A Review." *Anatomia, Histologia, Embryologia* 51: 563–575. https://doi.org/10.1111/ahe.12829.

Moore, M. J., J. V. Der Hoop, S. G. Barco, et al. 2013. "Criteria and Case Definitions for Serious Injury and Death of Pinnipeds and Cetaceans Caused by Anthropogenic Trauma." *Diseases of Aquatic Organisms* 103, no. 3: 229–264. https://doi.org/10.3354/dao02566.

Morales-Guerrero, B., C. Barragán-Vargas, G. R. Silva-Rosales, et al. 2017. "Melanin Granules Melanophages and a Fully-Melanized Epidermis Are Common Traits of Odontocete and Mysticete Cetaceans." *Veterinary Dermatology* 28: 213–250. https://doi.org/10.1111/vde.12392.

Mouton, M., and A. Botha. 2012. "Cutaneous Lesions in Cetaceans: an indicator of Ecosystem Status." In *New Approaches to the Study of Marine Mammals*, 123–151. InTech. https://doi.org/10.5772/54432.

Nicholls, C. R., K. J. Peters, D. Cagnazzi, D. Hanf, and G. J. Parra. 2023. "Incidence of Shark-Inflicted Bite Injuries on Australian Snubfin (*Orcaella heinsohni*) and Australian Humpback (*Sousa sahulensis*) Dolphins in Coastal Waters Off East Queensland, Australia." *Ecology and Evolution* 13, no. 5: e10026. https://doi.org/10.1002/ece3.10026.

Palmer, E. I., E. L. Betty, S. Murphy, M. R. Perrott, A. N. Smith, and K. A. Stockin. 2022. "Reproductive Biology of Female Common Dolphins (*Delphinus delphis*) in New Zealand Waters." *Marine Biology* 169, no. 12: 158. https://doi.org/10.1007/s00227-022-04139-3.

Petrini, B. 2006. "*Mycobacterium marinum*: Ubiquitous Agent of Waterborne Granulomatous Skin Infections." *European Journal of Clinical Microbiology and Infectious Diseases* 25: 609–613. https://doi.org/10.1007/s10096-006-0201-4.

Powell, S. N., M. M. Wallen, S. Bansal, and J. Mann. 2018. "Epidemiological Investigation of Tattoo-Like Skin Lesions Among Bottlenose Dolphins in Shark Bay, Australia." *Science of the Total Environment* 630: 774–780. https://doi.org/10.1016/j.scitotenv.2018.02.202.

Powell, S. N., M. M. Wallen, M. L. Miketa, et al. 2020. "Sociality and Tattoo Skin Disease Among Bottlenose Dolphins in Shark Bay, Australia." *Behavioral Ecology* 31, no. 2: 459–466. https://doi.org/10.1093/beheco/arz207.

Regalado, N. G., G. Martin, and S. J. Antony. 2009. "*Acinetobacter lwoffii*: Bacteremia Associated With Acute Gastroenteritis." *Travel Medicine and Infectious Disease* 7, no. 5: 316–317. https://doi.org/10.1016/j.tmaid.2009.06.001.

Rehtanz, M., G. D. Bossart, P. A. Fair, J. S. Reif, S.-J. Ghim, and A. B. Jenson. 2012. "Papillomaviruses and Herpesviruses: Who Is Who in Genital Tumor Development of Free-Ranging Atlantic Bottlenose Dolphins (*Tursiops truncatus*)?" *Veterinary Microbiology* 160: 297–304.

Rodrigues, T. C., K. Subramaniam, A. Varsani, et al. 2020. "Genome Characterization of Cetaceanpox Virus From a Managed Indo-Pacific Bottlenose Dolphin (*Tursiops aduncus*)." *Virus Research* 278: 197861. https://doi.org/10.1016/j.virusres.2020.197861.

Sacristán, C., A. C. Ewbank, A. M. Sánchez-Sarmiento, et al. 2022. "Erysipelas in a Stranded Common Bottlenose Dolphin: First Report in a South American Odontocete." *Brazilian Journal of Microbiology* 53, no. 4: 2199–2203. https://doi.org/10.1007/s42770-022-00810-5.

Sanino, G. P., M.-F. Van Bressem, K. Van Waerebeek, and N. Pozo. 2014. "Skin Disorders of Coastal Dolphins at Añihue Reserve, Chilean Patagonia: a Matter of Concern." *Boletín Del Museo Nacional De Historia Natural, Chile* 63: 127–157.

Segawa, T., and C. Kemper. 2015. "Cetacean Strandings in South Australia (1881–2008)." *Australian Mammalogy* 37, no. 1: 51–66. https://doi.org/10.1071/AM14029.

Segura-Göthlin, S., A. Fernández, M. Arbelo, et al. 2023. "Viral Skin Diseases in Odontocete Cetaceans: Gross, Histopathological, and Molecular Characterization of Selected Pathogens." *Frontiers in Veterinary Science* 10: 1188105. https://doi.org/10.3389/fvets.2023.1188105.

Soares, E. D., M. Cantor, A. P. F. R. L. Bracarense, K. R. Groch, and C. Domit. 2022. "Health Conditions of Guiana Dolphins Facing Cumulative Anthropogenic Impacts." *Mammalian Biology* 102: 1589–1604. https://doi.org/10.1007/s42991-022-00299-3.

Souter, R., A. L. Chaber, L. Möller, and L. Woolford. 2024. "Understanding Causes of Morbidity and Mortality in Southern Hemisphere Small Odontoceti: a Scoping Review." *Mammal Review* 55: e12371. https://doi.org/10.1111/mam.12371.

Souter, R., A.-L. Chaber, K. Lee, A. Machado, J. Lam, and L. Woolford. 2021. "Fatal *Streptococcus iniae* Infection in a Juvenile Free-Ranging Short-Beaked Common Dolphin (*Delphinus delphis*)." *Animals* 11, no. 11: 3123. https://doi.org/10.3390/ani11113123.

Souter, R., L. Lettoof, C. Kemper, et al. 2025. "Tracking Contaminant Trends: PFAS, Metals and Metalloids in Deceased Indo-Pacific Bottlenose (*Tursiops aduncus*) and Common Dolphins (Delphinus delphis) From South Australia, In Review."

Souter, R., I. Tomo, A.-L. Chaber, et al. 2023. *Temporal, Spatial and Pathological Findings in Small Cetacean Strandings in South Australia 2021–2023*. 69th Annual Scientific Meeting of the Australian Mammal Society.

St. Leger, J., S. Raverty, and A. Mena. 2018. "Chapter 22 – Cetacea." In *Pathology of Wildlife and Zoo Animals*, edited by K. A. Terio, M. Denise, and J. St. Leger, 533–564. Elsevier Science & Technology.

Stephens, N., P. J. Duignan, J. N. Wang, et al. 2014. "Cetacean Morbillivirus in Coastal Indo-Pacific Bottlenose Dolphins, Western Australia." *Emerging Infectious Diseases* 20, no. 4: 666–670. https://doi.org/10.3201/eid2004.131714.

Tomo, I., and C. M. Kemper. 2022. "Strandings in St Vincent Gulf Bioregion, South Australia: 12-Year Study Monitors Biology and Pathology of Cetaceans." *Oceans* 3, no. 4: 439–463. https://doi.org/10.3390/oceans3040030.

Toms, C. N., T. Stone, and T. Och-Adams. 2020. "Visual-Only Assessments of Skin Lesions on Free-Ranging Common Bottlenose Dolphins (*Tursiops truncatus*): Reliability and Utility of Quantitative Tools." *Marine Mammal Science* 36, no. 3: 744–773. https://doi.org/10.1111/mms.12670.

Townsend, F., and L. Staggs. 2020. "Atlas of Skin Diseases of Small Cetaceans."

Tukey, J. W. 1949. "Comparing Individual Means in the Analysis of Variance." *Biometrics* 5: 99–114. https://doi.org/10.2307/3001913.

Van Bressem, M. F., P. J. Duignan, A. Banyard, et al. 2014. "Cetacean Morbillivirus: Current Knowledge and Future Directions." *Viruses* 6, no. 12: 5145–5181. https://doi.org/10.3390/v6125145.

Van Bressem, M. F. and J. A. Raga. 2011. "Viruses of Cetaceans." In *Studies in Viral Ecology* 2: 309–332. Wiley.

Van Bressem, M. F., K. Van Waerebeek, F. J. Aznar, et al. 2009. "Epidemiological Pattern of Tattoo Skin Disease: a Potential General Health indicator for Cetaceans." *Diseases of Aquatic Organisms* 85, no. 3: 225–237. https://doi.org/10.3354/dao02080.

Van Bressem, M. F., K. Van Waerebeek, and P. J. Duignan. 2018. "Epidemiology of Tattoo Skin Disease in Captive Common Bottlenose Dolphins (*Tursiops truncatus*): Are Males More Vulnerable Than Females?" *Journal of Applied Animal Welfare Science* 21: 305–315.

Van Bressem, M. F., K. Van Waerebeek, and P. J. Duignan. 2022. "Tattoo Skin Disease in Cetacea: A Review, With New Cases for the Northeast Pacific." *Animals* 12: 3581. https://doi.org/10.3390/ani12243581.

Van Bressem, M. F., K. Van Waerebeek, L. Flach, et al. 2008. "Skin Diseases in Cetaceans. International Whaling Commission. Conference: Workshop on Cetacean Skin Diseases, 30–31 May."

Van Bressem, M. F., K. Van Waerebeek, G. E. Piérard, and C. Desaintes. 1996. "Genital and Lingual Warts in Small Cetaceansfrom Coastal Peru." *Diseases of Aquatic Organisms* 26: 1–10.

Van Bressem, M. F., K. Van Waerebeek, and J. A. Raga. 1999. "A Review of Virus Infections of Cetaceans and the Potential Impact of Morbilliviruses, Poxviruses and Papillomaviruses on Host Population Dynamics." *Diseases of Aquatic Organisms* 38, no. 1: 53–65. https://doi.org/10.3354/dao038053.

Van Bressem, M. F., K. Van Waerebeek, J. C. Reyes, D. Dekegel, and P.-P. Pastoret. 1993. "Evidence of Poxvirus in Dusky Dolphin (*Lagenorhynchus obscurus*) and Burmeister's Porpoise (*Phocoena spinipinnis*) From Coastal Peru." *Journal of Wildlife Diseases* 29: 109–113.

Van Bressem, M. F., and K. Van Waerebeek. 1996. "Epidemiology of Poxvirus in Small Cetaceans From the Eastern South Pacific." *Marine Mammal Science* 12, no. 3: 371–382. https://doi.org/10.1111/j.1748-7692.1996.tb00590.x.

van Elk, C. E., M. W. Van De Bildt, A. A. De Jong, A. D. Osterhaus, and T. Kuiken. 2009. "Herpesvirus in Bottlenose Dolphins (*Tursiops truncatus*): Cultivation, Epidemiology, and Associated Pathology." *Journal of Wildlife Diseases* 45, no. 4: 895–906. https://doi.org/10.7589/0090-3558-45.4.895.

van Elk, N., U. Siebert, G. Desportes, and K. Anderson. 2000. "Skin Pathology, Possibly Caused by Pox Virus, Leading to Severe Health Problems in Harbor Porpoises From European Waters. Annual Conference-American Association of Zoo Veterinarians. American Association of zoo Veterinarians; 1998, 194–194."

VanDevanter, D. R., P. Warrener, L. Bennett, et al. 1996. "Detection and Analysis of Diverse Herpesviral Species by Consensus Primer PCR." *Journal of Clinical Microbiology* 34, no. 7: 1666–1671. https://doi.org/10.1128/jcm.34.7.1666-1671.1996.

Wan, X., J. Li, M. Ao, et al. 2023. "The Intestinal Microbiota of a Risso's Dolphin (*Grampus griseus*): Possible Relationships With Starvation Raised by Macro-Plastic Ingestion." *International Microbiology* 26: 1001–1007. https://doi.org/10.1007/s10123-023-00355-z.

Wearmouth, V. J., and D. W. Sims. 2008. "Sexual Segregation in Marine Fish, Reptiles, Birds and Mammals: Behaviour Patterns, Mechanisms and Conservation Implications." *Advances in Marine Biology* 54: 107–170. https://doi.org/10.1016/S0065-2881(08)00002-3.

Weijs, L., A. Covaci, G. Stevenson, C. Kemper, I. Tomo, and F. Leusch. 2020. "Concentrations of Some Legacy Pollutants Have Increased in South Australian Bottlenose Dolphins From 1989 to 2014." *Environmental Research* 189: 109834. https://doi.org/10.1016/j.envres.2020.109834.

Wilson, B., H. Arnold, G. Bearzi, et al. 1999. "Epidermal Diseases in Bottlenose Dolphins: Impacts of Natural and Anthropogenic Factors." *Proceedings of the Royal Society of London, Series B: Biological Sciences* 266, no. 1423: 1077–1083. https://doi.org/10.1098/rspb.1999.0746.

Zabka, T. S., and T. A. Romano. 2003. "Distribution of MHC II (+) Cells in Skin of the Atlantic Bottlenose Dolphin (*Tursiops truncatus*): An Initial Investigation of Dolphin Dendritic Cells." *Anatomical Record Part A: Discoveries in Molecular, Cellular, and Evolutionary Biology* 273: 636–647.

Zanardo, N., G. J. Parra, and L. M. Möller. 2016. "Site Fidelity, Residency, and Abundance of Bottlenose Dolphins (*Tursiops* sp.) in Adelaide's Coastal Waters, South Australia." *Marine Mammal Science* 32, no. 4: 1381–1401. https://doi.org/10.1111/mms.12335.

Zhang, X.-H., X. He, and B. Austin. 2020. "*Vibrio harveyi*: a Serious Pathogen of Fish and Invertebrates in Mariculture." *Marine Life Science & Technology* 2, no. 3: 231–245. https://doi.org/10.1007/s42995-020-00037-z.

Supporting Information

Additional supporting information can be found online in the Supporting Information section. **Table S1:** Macroscopic and microscopic descriptions of examined traumatic and non-traumatic skin and mucosal lesions from stranded and deceased common dolphins (*Delphinus delphis*) and Indo-Pacific bottlenose dolphins (*Tursiops aduncus*) from South Australia between June 2021 and April 2024. BCS = body condition score. COD = cause of death, GSV = Gulf St. Vincent, CO = Coorong, KI = Kangaroo Island, SG = Spencer Gulf. (*) indicates mucosal lesions. NP = not performed. ICIBs = intracytoplasmic inclusion bodies. INIBs = intranuclear inclusion bodies. −ve = negative. PCR = polymerase chain reaction, ZN = Ziehl-Neelson stain, PAS = Periodic acid-Schiff stain, GMS = Grocott's methenamine silver stain, MSB = Martius Scarlet Blue stain. **Figure S1:** Topography of skin lesions examined in common dolphins (*Delphinus delphis*) and Indo-Pacific bottlenose (*Tursiops aduncus*) of South Australia between June 2001 and April 2024. Lesions were categorized into the following topographic regions: Head, dorsal fin, dorsum, laterals (thoracic and abdominal regions), peduncle, pectoral fin, ventrum, and tail flukes.