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Unchartered waters: Climate change likely to intensify infectious disease outbreaks causing mass mortality events in marine mammals

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Abstract

Infectious disease emergence has increased significantly over the last 30 years, with mass mortality events (MMEs) associated with epizootics becoming increasingly common. Factors influencing these events have been widely studied in terrestrial systems, but remain relatively unexplored in marine mammals. Infectious disease-induced MMEs (ID MMEs) have not been reported ubiquitously among marine mammal species, indicating that intrinsic (host) and/or extrinsic (environmental) ecological factors may influence this heterogeneity. We assess the occurrence of ID MMEs (1955–2018) across extant marine mammals (n = 129) in relation to key life-history characteristics (sociality, trophic level, habitat breadth) and environmental variables (season, sea surface temperature [SST] anomalies, El Niño occurrence). Our results show that ID MMEs have been reported in 14% of marine mammal species (95% CI 9%–21%), with 72% (n = 36; 95% CI 56%–84%) of these events caused predominantly by viruses, primarily morbillivirus and influenza A. Bacterial pathogens caused 25% (95% CI 14%–41%) of MMEs, with only one being the result of a protozoan pathogen. Overall, virus-induced MMEs involved a greater number of fatalities per event compared to other pathogens. No association was detected between the occurrence of ID MMEs and host characteristics, such as sociality or trophic level, but ID MMEs did occur more frequently in semi-aquatic species (pinnipeds) compared to obligate ocean dwellers (cetaceans; χ^2 = 9.6, p = .002). In contrast, extrinsic factors significantly influenced ID MMEs, with seasonality linked to frequency (χ^2 = 19.85, p = .0002) and severity of these events, and global yearly SST anomalies positively correlated with their temporal occurrence (Z = 3.43, p = 2.7e-04). No significant association was identified between El Niño and ID MME occurrence (Z = 0.28, p = .81). With climate change forecasted to increase SSTs and the frequency of extreme seasonal weather events, epizootics causing MMEs are likely to intensify with significant consequences for marine mammal survival.

KEYWORDS

cetacean, climate change, infectious disease, mass mortality, morbillivirus, pinniped, sea surface temperature

1 | INTRODUCTION

Mass mortality events are incidences in nature resulting in a devastating number of fatalities within the same species over a brief period of time. MMEs can affect a diverse range of species, from corals (Garrabou, Perez, Sartoretto, & Harmelin, 2001) and bivalves (Vázquez-Luis et al., 2017), to migratory birds (Newton, 2007) and terrestrial mammals (Kock et al., 2018). The classification of a MME is dependent upon the population size and conservation status of the species involved. As such, MMEs can vary significantly in size, ranging from the death of a few individuals to hundreds of thousands. One of the largest MMEs recorded occurred during the spring of 2015 in central Kazakhstan and involved the deaths of over 200,000 critically endangered saiga antelopes (*Saiga tatarica*) within a 3 week period (Kock et al., 2018). While MMEs have devastating effects for the species in question (e.g., by reducing genetic diversity, increasing Allee effect potential and amplifying the chance of local extirpation or even extinction, Lande, 1993), they can also have cascading effects within ecosystems, with changes in community structure occurring across trophic levels (Palmer, Stephens, Ward, & Willis, 2015).

Mass mortality events are most frequently correlated with infectious disease outbreaks (herein ID MME), but they can also be caused by human influences (e.g., environmental contamination), biotoxicity and/or climate driven factors, such as heat stress and weather (Fey et al., 2015). In marine mammal species, MMEs have been associated with a wide array of drivers, including brevitoxins (Bossart, Baden, Ewing, Roberts, & Wright, 1998; Flewelling et al., 2005), starvation (Preen & Marsh, 1995) and infectious disease (Aguilar & Raga, 1993; Härkönen et al., 2006).

Over the last 30 years, infectious disease outbreaks in marine mammal species have increased substantially (Gulland & Hall, 2007; Harvell et al., 1999; Simeone, Gulland, Norris, & Rowles, 2015). While causal factors influencing pathogen transmission and outbreak potential in terrestrial mammals have been widely studied, the factors prompting these events in marine mammals are still mostly unknown (Kim, Dobson, Gulland, & Harvell, 2005).

Across species, heterogeneity in infectious disease dynamics and outcomes can be attributed to host, pathogen and/or environmental factors, with contact rates determining disease transmission potential. For directly transmitted pathogens, contact rates between susceptible individuals are dependent on the hosts' physiological and behavioural characteristics (VanderWaal & Ezenwa, 2016; Young, 1994). For example, in terrestrial animals, highly social species are more likely to succumb to infectious disease than solitary species, due to host aggregation and consequent increases in contact rates (Sanderson, Jobbins, & Alexander, 2013). Likewise, species that feed at different trophic levels have varying exposure risk. In some terrestrial host–pathogen systems, carnivores are known to have higher seroprevalence levels to multihost pathogens, such as *Toxoplasma gondii*, compared to browsers (Hollings, Jones, Mooney, & McCallum, 2013). For environmentally transmitted pathogens, contact rate is determined by space use. As such, species inhabiting a variety of niches or occupying larger home ranges are more likely to experience infectious disease outbreaks, due to the increased probability of contact with spatially restricted environmental reservoirs. These species may also have increased contact with infected individuals from the same or different species. In marine mammals, this may include increased contact with terrestrial hosts (Bradley & Altizer, 2007; VanderWaal & Ezenwa, 2016). Anthropogenic change resulting in altered host distributions and densities is another important factor influencing disease transmission potential (Alexander et al., 2018; Flint, Hawley, & Alexander, 2016).

Extrinsic ecological factors, such as weather and climate variables, also play a critical role in infectious disease dynamics (Harvell et al., 2002). The naturally occurring climatic phenomenon, El Niño/Southern Oscillation (ENSO), comprising the warming (El Niño) and cooling (La Niña) of sea surface temperatures (SSTs; Holton & Dmowska, 1989), affects weather on a seasonal scale. In turn, seasonal weather patterns affect temperature, precipitation, humidity and sunlight hours, among other factors. Seasonal variations in weather are known to impact the spread and longevity of infectious diseases within populations by altering pathogen growth, survival and virulence, as well as by altering host social behaviour leading to increased contact rates (e.g., congregating during the breeding season in spring, seasonal migration patterns) or heightened susceptibility (e.g., parturition in spring; Altizer et al., 2006). Interactions between infectious disease dynamics and seasonal drivers are expected to increase under current climate change predictions (Cai et al., 2014; Cann, Thomas, Salmon, Wyn-Jones, & Kay, 2013).

While global changes in climate have occurred historically due to alterations in the Earth's orbit (Hays, Imbrie, & Shackleton, 1976), the current rapid rate of climate change from anthropogenic sources is of extreme concern (Steffen et al., 2018). One of the most measurable indicators of climate change is temperature, with SST anomalies (deviations from the long-term average SST), in particular, mirroring global warming trends (Hansen, Ruedy, Sato, & Lo, 2010). Furthermore, increases in SSTs have already resulted in disease outbreaks in many marine species, including corals (Bruno et al., 2007), sea stars (Eisenlord et al., 2016), oysters (Cook, Folli, Klinck, Ford, & Miller, 1998) and abalone (Moore, Robbins, & Friedman, 2000).

Here we evaluate ID MMEs in marine mammals by pathogen type and species, and investigate the potential relationships between intrinsic and extrinsic ecological factors on the occurrence of these events. We discuss our results in the context of the conservation and management implications for marine mammals.

2 | MATERIALS AND METHODS

2.1 | Data collection

2.1.1 | Host life-history data

A list of 129 extant marine mammals species, together with data on taxonomic rank (genus and family), social behaviour (sociality), food

preferences (trophic level), habitat use (marine biome) and number of habitat layers used (habitat breadth) were extracted using the International Union for Conservation of Nature (IUCN) Red List of Threatened Species (IUCN, 2018), the Catalogue of Life (Life, 2018) and the PanTHERIA database (Jones et al., 2009).

Categorical variables pertaining to host ecology were defined as follows. Sociality was dichotomized into two groups: solitary and social. While it is well known that sociality manifests along a continuum (Jennions & Macdonald, 1994) and is defined by the mode and scale in which conspecifics aggregate and cooperate, for simplicity we classified species into solitary; rarely aggregates or associates outside the breeding period, and social; aggregates year round with or without social composition. For example, Caspian seals (*Pusa caspica*) and harbour seals (*Phoca vitulina*) may share access holes and/or congregate during breeding seasons, but were considered solitary. Gregarious species, and those that occur in small groups or as couples, including those who employ fission–fusion dynamics, such as Hector's dolphins (*Cephalorhynchus hectori*) and Australian snubfin dolphins (*Orcaella heinsohni*), were all considered social. Trophic level was categorized into three groups: herbivore, omnivore and carnivore, and was dependent on the position a species occupied within the food chain. Habitat was defined by the marine biome(s) that a species occupies. Coastal biomes consist of locations near a coast or shoreline, pelagic biomes consist of ocean far from the coast and benthic biomes are located below pelagic biomes, on or close to the bottom of the ocean. Rivers and streams, and lakes and ponds were additional marine biomes used for classifying marine habitat. Many species spanned more than one biome, with 13 different categories of marine biome combinations documented. Habitat breadth was defined as one; solely occurs in water (obligate ocean dweller), or two; frequents both land and water (semiaquatic).

2.1.2 | Infectious disease-associated MME data

To collect data for our dependent variable, occurrence of an ID MME, we used Google Scholar, PubMed and ISI Web of Knowledge databases to search for reported MMEs or massive die-off events in published papers and reputable sources. Researchers reporting these events used a combination of examination methods to confirm that an infectious disease was the cause of mortality. Methods included a combination of natural history, clinical symptoms, post-mortem and gross pathology, histopathology and immunohistology, and polymerase chain reaction (PCR) analysis and sequencing. Searches included one or more of the following phrases: *die off**, *die-off**, *kill**, *mass kill**, *mass mortalit**, *mass death** or *unusual mortality event*, as described in Fey et al. (2015), as well as aetiologic categories and terms related to disease (e.g., *illness**, *patho**, *disease**, *infec**, *myco* parasite**). Each term was searched in association with genus names (e.g., *Arctocephalus* AND *disease**). Global peer-reviewed articles, reports and books published between 1955 and 2018 documenting disease and mortality were collected and considered in this analysis. These were organized by species, author and year to ensure that events were not duplicated. Only literature containing reports on wild animals and documents in English were examined. This potentially biased our results, limiting events to species living in proximity to English speaking countries or to scientists who publish only in English speaking journals. Thus, for species living around parts of Central and South America, Asia, Europe and Africa, ID MMEs may be more extensive than reported in the present study. Additionally, our ID MME database included infectious disease-induced unusual mortality event (UME) data from the National Oceanic and Atmospheric Administration (NOAA) Fisheries marine mammal active and closed UME database (NOAA, 2018a). Details of these events were confirmed through personal communications with a NOAA Fisheries staff member (D. Fauquier pers. comm.). As defined by the Marine Mammal Protection Act, an UME has occurred if strandings, morbidities or mortalities in marine mammals have met one or more of the following criteria: 1) compared to previous years, there has been a significant increase in number, 2) a spatial, temporal, or demographic (i.e. sex or age) shift in these events has occurred, 3) vulnerable populations of marine mammals have been affected, 4) population declines have been reported, 5) the individuals involved display similar behaviour, clinical symptoms or pathological findings (NOAA, 2018b). Due to the stringent UME response and reporting requirements enforced by the United States Marine Mammal Protection Act (1972; Gulland & Hall, 2007), our data set is skewed towards ID MMEs occurring within United States waters and has also likely resulted in the underrepresentation of tropical marine mammal species. For each ID MME, we collected the date of occurrence, geographic location, species involved, number of fatalities, and the pathogen implicated in the epizootic. Any environmental correlate described in the original publication as potentially having influenced the outbreak, such as unseasonably warm SSTs, premature disappearance of sea ice or high rainfall, was also collected (Supporting Information 1). Additionally, notes on host behaviour during the outbreak (e.g., migration, overcrowding), as well as the population demographic affected by the event (e.g., sex and age) were compiled, if described by the original source or follow-up peer-reviewed manuscripts (Supporting Information 1). Events that involved more than one species were still classified as one ID MME, but all species involved in that event were included in our statistical analyses.

2.1.3 | Climate data

SST anomaly and El Niño index data were accessed through the International Research Institute for Climate and Society (IRI) Climate Data Library (http://iridl.ldeo.columbia.edu/). We extracted monthly global Extended reconstructed SST (ERSST) anomalies data (1955–2018), which is based on ERSST Version 5 (ERSSTv5; Huang et al., 2017), and Niño 3.4 index data (1955–2018), presented as average SST anomalies in the equatorial Pacific (5°N–5°S, 170–120°W).

The Niño 3.4 index is classified as the most representative indicator of ENSO (Bamston, Chelliah, & Goldenberg, 1997) and is constructed by averaging five consecutive 3 month SST anomalies. Both data sets were collated into yearly averages. As the SST anomaly data is based on a global and yearly average, we also used the plot function provided by the NOAA Earth System Research Laboratory (W/NP24, C. M. B. & NCEP/NWS/NOAA, 2018b) to capture any SST anomalies that occurred within the same geographic location and within the first month of an infectious disease outbreak causing a MME (Supporting Information 2). The plot function contained historical data from 1981 onwards, and as such we were unable to plot the three outbreaks occurring before this time.

2.2 | Statistical analyses

2.2.1 | Host life-history and infectious disease-associated MMEs

All statistical analyses were conducted in the open source statistical software package R 3.6.1 (R Core Team, 2019). *FactoMineR* (Husson, Josse, Le, Mazet, & Husson, 2016) was used to conduct multivariate exploratory data analysis, specifically, multiple correspondence analysis (MCA). MCA is ideal for the investigation of data sets with multiple qualitative variables and allows for the exploration of potential interactions between variables and categories (Di Franco, 2016). We used MCA to investigate potential relationships between host life-history characteristics and the occurrence of ID MMEs.

Our life-history database originally consisted of 129 species and seven categorical variables, (order, family, sociality, trophic level, marine biome, habitat breadth and our dependent variable, the occurrence of an ID MME). Before carrying out the analysis, we removed species that lacked data for any of the variables, and removed variables and categories that were not balanced or whose frequencies were too low, in order to avoid biasing our results (Di Franco, 2016). Numerous MCA analyses were carried out, with each analysis refining our results by identifying and removing variables that did not contribute substantially to the explained variance within the data set (Di Franco, 2016). The final data set consisted of 119 species and five categorical variables (order, sociality, trophic level, habitat breadth and occurrence of an ID MME; Table 1). Data points that contributed the most to the clustering of data were determined through the construction of a correlation matrix using *corrplot* in *FactoMineR*, and associations between variables were visualized in graphs using the *factoextra* package. (Kassambara & Mundt, 2017). We performed cluster analysis on our MCA results using hierarchical clustering on principal components (HCPC) to identify subgroups (clusters) of similar individuals within our data set using the *HCPC* function in *FactoMineR*.

After potential correlations between variables were identified in the MCA and HCPC, we used the R package, *GoodmanKruskal*, which measures Goodman and Kruskal's tau (τ) to quantify asymmetric associations between categorical variables (i.e. between two categorical variables (x and y), both forward $\tau(x,y)$ and backward associations $\tau(y,x)$ are determined) (Pearson, 2016). This test produces a plot with both a graphical and numerical representation of τ. Two-way Pearson's chi-square tests of independence were used to determine associations between ID MME occurrence and life-history variables, with the level of significance set at $p < .05$. R package, *ggplot2* (Wickham, 2016), was used to create graphs.

TABLE 1 The host-related variables and categories used in multiple correspondence analyses (MCA) to assess potential linkages to infectious disease-associated mass mortality events (ID MMEs)

Variable	Number of categories	Category
Order	2	Carnivora (37), Cetartiodactyla (82)
Family	18	Balaenidae (4), Balaenopteridae (7), Delphinidae (37), Eschrichtiidae (1), Iniidae (1), Kogiidae (2), Lipotidae (1), Monodontidae (2), Mustelidae (2), Odobenidae (1), Otariidae (15), Phocidae (18), Phocoenidae (7), Physeteridae (1), Platanistidae (1), Pontoporiidae (1), Ursidae (1), Ziphiidae (17)
Sociality	2	Social (80), Solitary (39)
Trophic level	2	Carnivore (92), Omnivore (27)
Marine biome	12	Benthic + Coastal (3), Coastal (48), Coastal + Pelagic (34), Coastal + Pelagic + Benthic (1), Coastal + Pelagic + Rivers and streams (2), Coastal + Reef (2), Coastal + Rivers and streams (5), Coastal + Rivers and streams + Reef (2), Lakes and ponds (1), Pelagic (17), Pelagic + Benthic (1), Rivers and streams (3)
Habitat breadth	2	OneHabitat (84), TwoHabitats (35)
Occurrence of ID MMEs	2	IDMME (18), NoIDMME (101)

Note: Variables used in the final MCA analysis are highlighted in grey, and the number of species associated with each category is shown in brackets.

2.2.2 | Environmental factors and infectious disease-associated MMEs

A four-way Pearson's chi-square test of independence was used to determine associations between ID MME occurrence and season, with the level of significance set at $p < .05$. Given that our dependent variable (ID MME occurrence) was comprised of count data, we used a Poisson regression with the log-link function to determine relationships between ID MME occurrence and climate variables (SST anomalies and Nino3.4) using the *glm* (generalized linear model) function in R. We tested two models (one with SST anomalies as the predictor, and the other with both SST anomalies and Nino3.4 as predictors), then carried out a two degree-of-freedom chi-square test to determine the best model. We obtained confidence intervals for the coefficient estimates using the *confint* function. The R package *sandwich* was used to calculate the robust standard errors and associated *p*-values (Zeileis, 2004). We carried out a goodness of fit chi-square test on the residual deviance obtained from the model by using the probability chi-square function (*pchisq*) in R. Using the R package *msm*, we computed the robust exponentiated coefficients, which are equivalent to the incident rate ratio (Jackson, 2011). We used the *predict* function to determine the predicted (expected) number of ID MMEs and plotted these against yearly global SST anomalies using the R package *ggplot2* (Wickham, 2016). The observed number of ID MMEs was also plotted onto the graph.

2.3 | Evaluation of conservation implications

To evaluate the potential conservation implications of ID MMEs, the IUCN conservation status (*Critically endangered*, *Endangered*, *Vulnerable*, *Near threatened*, *Least concern*, *Data deficient*), as well as species population trends (*Increasing*, *Stable*, *Decreasing*, *Data deficient*), were collected from the IUCN Red List of Threatened Species (IUCN, 2018) for each marine mammal species ($n = 129$).

3 | RESULTS

Overall, 14% (95% CI 9%–21%) of extant marine mammal species have experienced MMEs caused by infectious disease outbreaks (Figure 1). Of these species ($n = 18$), 56% were pinnipeds (95% CI 34%–75%), 39% (95% CI 20%–61%) were cetaceans and one was a fissiped (sea otter; *Enhydra lutris*; 6%, 95% CI 0%–26%). All ID MMEs in cetaceans were caused by viral pathogens ($n = 12$; 100%, 95% CI 76%–100%), while both viral (61%, 95% CI 41%–79%) and bacterial pathogens (39%, 95% CI 22%–59%) were implicated as causative agents in ID MMEs in pinnipeds. The fissiped suffered from the sole protozoan pathogen-induced MME. When considering the location of ID MMEs, 83% (95% CI 68%–92%) occurred in the Northern Hemisphere, with only six ID MMEs occurring in the Southern Hemisphere (Figure 1). These findings again may be associated with surveillance bias.

While the majority of ID MMEs involved a single marine mammal species (89%, 95% CI 75%–96%), two ID MMEs involved two species (harbour seal and grey seal; *Halichoerus grypus*), and another three ID MMEs involved three or more species (Supporting Information 1). While the overall mean number of ID MMEs per species was two (mode = one), since 1955, the harbour seal has experienced 10 of these events (Figure 1).

Viruses were identified as the main aetiological agents responsible for ID MMEs in marine mammals (72%, 95% CI 56%–84%), specifically morbilliviruses (53%, 95% CI 37%–68%) and influenza A viruses (14%, 95% CI 6%–29%). Collectively, virus-induced MMEs

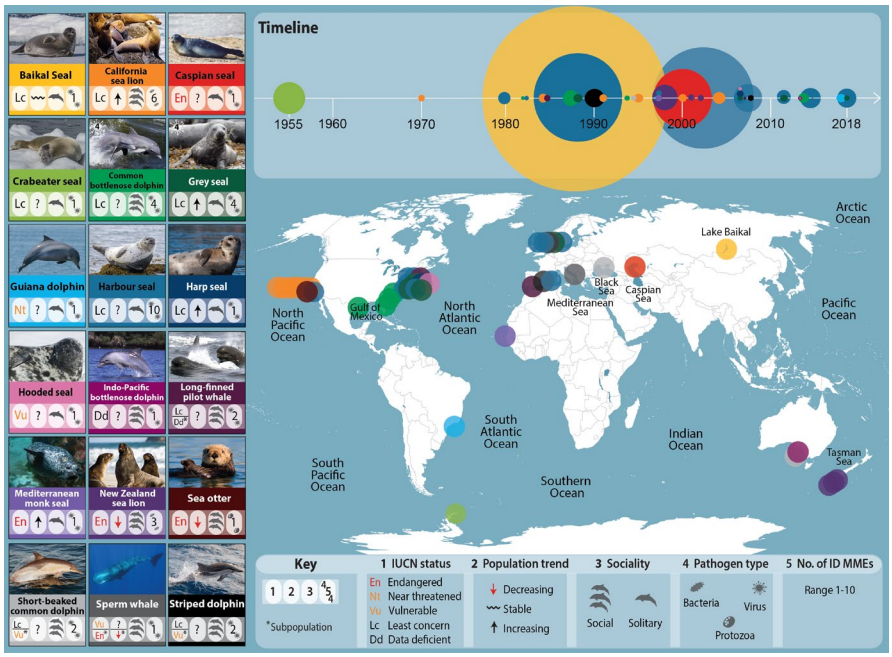

FIGURE 1 Temporal and spatial distribution of epizootics causing mass mortality events (ID MMEs) in marine mammals. The timing and number of fatalities of each ID MME ($n = 36$) are indicated along a scaled timeline (1955–2018). The current IUCN conservation status and population trend for each species is noted, along with the sociality of the host, pathogen type responsible for the ID MME and the number of reported ID MMEs for each species since 1955

caused the greatest number of fatalities, on average causing 20 times more deaths than bacteria-induced MMEs (average 7,029 deaths per virus-induced MME vs. 350 deaths per bacteria-induced MME). Bacteria accounted for 25% (95% CI 14%–41%) of epizootics resulting in a MME, with leptospirosis (17%, 95% CI 8%–32%) and *Klebsiella pneumoniae* (8%, 95 CI 3%–22%) being the two bacterial pathogens involved. Only one protozoan pathogen, *Sarcocystis neurona*, was identified as the cause of a single MME.

3.1 | Host life-history and infectious disease-associated MMEs

Multiple correspondence analysis provided important insights into underlying structures in our data allowing us to visualize potential linkages between host life-history variables and ID MME occurrence. Before carrying out the analysis, we removed species that lacked data for any of the variables (n = 6) and we removed the categories, *Herbivores* (n = 3) and the remaining Sirenia (n = 1), due to low frequencies within our data set. Incidentally, no ID MMEs have been documented in herbivores. Through repeated refining of our MCA output, our final analysis revealed that five variables were required to describe the variation within our data set, consisting of 119 species (Table 1). The variable categories, family and marine biome, were not used in our final analysis as they were not balanced and introduced potential biases into our results. MCA output in the form of eigenvalues represents the contribution of each dimension in explaining the differences between categorical variables. A higher eigenvalue indicates a larger overall variance between the variables in that dimension. Our largest eigenvalues were found in the first (Dim1, 0.57) and second (Dim2, 0.19) dimensions, representing 76.12% of the cumulative variation present in the data set. Order (R^2 = 0.95, p = 2.71e-77) and habitat breadth (R^2 = 0.91, p = 2.23e-62) explained the greatest amount of variation in Dimension 1, with *Carnivora* (R^2 = 0.8, p = 2.71e-77) and *TwoHabitats* (R^2 = 0.79, p = 2.23e-62) being the dominant categories. In Dimension 2, ID MME occurrence (R^2 = 0.72, p = 2.68e-34) was the dominant variable, with *IDMME* being the dominant category (R^2 = 0.52, p = 2.68e-34). A graph of the first two dimensions was created, with the categories of data that explained the greatest amount of variation in the data set plotted (Figure 2).

Categories of the same variable did not cluster together on our two-dimensional graph (Figure 2) indicating that they occur independently of one another (Di Franco, 2016). For example, ID MME occurrence (*IDMME*) and no ID MME occurrence (*NoIDMME*) do not cluster together. Likewise, ID MME occurrence (*IDMME*) does not cluster with any host life-history variables and is therefore occurring independently of these variables (Figure 2). When hierarchical clustering was performed on our MCA, two clusters were identified based primarily on order and habitat breadth (Table 2). *IDMME* appears in both clusters, however, it was more significantly correlated with clade 2 (p < .05, v test > 2), clustering with *Carnivora*, *TwoHabitats*, *Omnivore* and *Solitary* (Table 2).

From the output of the Goodman and Kruskal's tau analysis, it is evident that order is strongly associated with habitat breadth (τ(Order, Habitat breadth) = 0.92), and the reverse association,

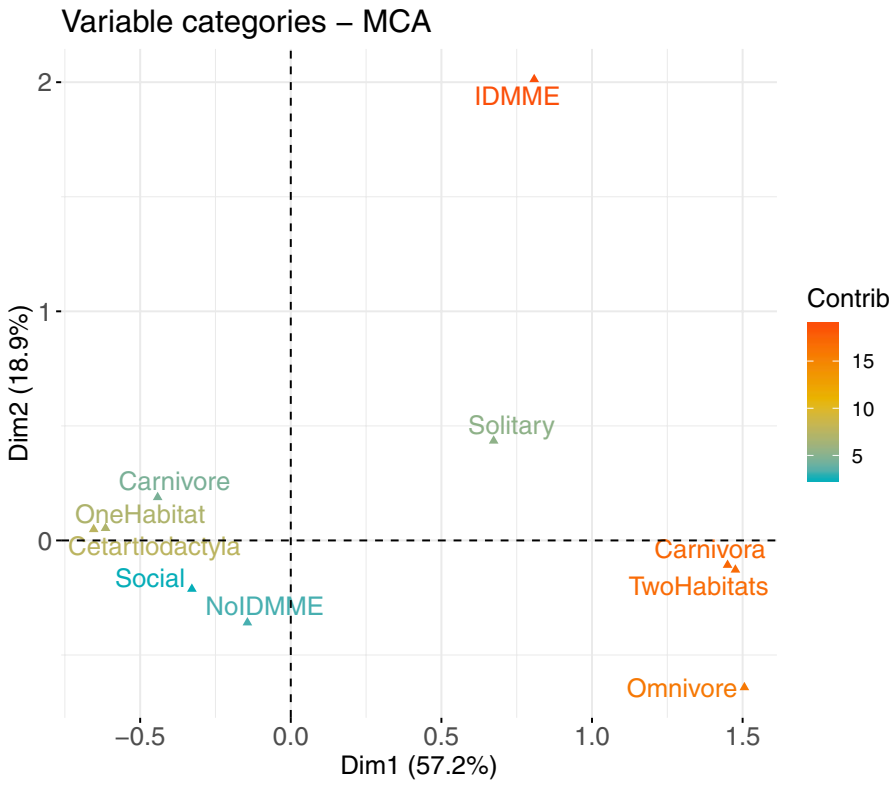

FIGURE 2 Two-dimensional graph of the multiple correspondence analysis (MCA) on marine mammal host traits and infectious disease-associated mass mortality events (ID MMEs). The first two dimensions of the MCA were plotted to provide an examination of associations between categorical variables. The colour of the categorical variable label provides an indication of the relative contribution of that variable to the variance present within the data set (red being greatest)

TABLE 2 Description of the two clusters identified using hierarchical clustering on principal components (HCPC) on the multiple correspondence analysis (MCA) output

	Variable	Category	Cla/Mod	*p* Value	*v* Test
Clade 1	Order	*Cetartiodactyla*	100%	<.001	11.71
	Habitat breadth	*OneHabitat*	98%	<.001	10.99
	Trophic level	*Carnivore*	88%	<.001	8.24
	Sociality	*Social*	81%	<.001	4.03
	ID MME	*NoIDMME*	74%	.005	2.80
	ID MME	*IDMME*	39%	.005	−2.80
	Sociality	*Solitary*	44%	<.001	−4.03
	Trophic level	*Omnivore*	4%	<.001	−8.24
	Habitat breadth	*TwoHabitats*	0%	<.001	−10.99
	Order	*Carnivora*	0%	<.001	−11.71
Clade 2	Order	*Carnivora*	100%	<.001	11.71
	Habitat breadth	*TwoHabitats*	100%	<.001	10.99
	Trophic level	*Omnivore*	96%	<.001	8.24
	Sociality	*Solitary*	56%	<.001	4.03
	ID MME	*IDMME*	61%	.005	2.80
	ID MME	*NoIDMME*	26%	.005	−2.80
	Sociality	*Social*	19%	<.001	−4.03
	Trophic level	*Carnivore*	12%	<.001	−8.24
	Habitat breadth	*One Habitat*	2%	<.001	−10.99
	Order	*Cetartiodactyla*	0%	<.001	−11.71

Note: Occurrence of a mass mortality event due to infectious disease (*IDMME*; dark grey) primarily grouped in clade 2 with the categorical variables *Carnivora*, *TwoHabitats*, *Omnivore and Solitary*. All significant categorical variables for each clade are highlighted in light grey ($p < .05$, *v* test > 2).

while slightly less, is also true (τ(Habitat breadth, Order) = 0.8; Figure 3). This indicates a strong correlation between these two variables, which confirms what we see on the HCPC. Trophic level also showed some correlation to both order (τ(Trophic level, Order) = 0.59) and habitat breadth (τ(Trophic level, Habitat breadth) = 0.48), with similar levels of correlation seen in the reserve associations (τ(Order, Trophic level) = 0.6, τ(Habitat breadth, Trophic level) = 0.41; Figure 3). Again, this is shown in the HCPC, where a greater percentage of carnivores (88%) are clustered with Cetartiodactyla and one habitat, while omnivores (96%) are primarily clustered with Carnivora and two habitats. In contrast, correlations were lacking between ID MME and all four life-history variables (Figure 3).

We employed two-way Pearson's chi-square tests of independence to confirm associations between ID MME occurrence and *Carnivora*, *TwoHabitats*, *Omnivore* and *Solitary*, which were clustered together in the HCPC analysis. We found significant associations between order and ID MME occurrence, with more ID MMEs occurring in Carnivora (30%, 95% CI 17%–46%) than Cetartiodactyla (9%, 95% CI 5%–18%; $\chi^2 = 8.92$, $p = .003$). Likewise, significantly more ID MMEs occurred in species occupying two habitats (28%, 95% CI 16%–44%) than in species occupying one habitat (10%, 95% CI 5%–18%; $\chi^2 = 6.98$, $p = .008$). However, the Goodman and Kruskal's tau analysis shows that order and habitat breadth are highly correlated, which is predicted given that all Cetartiodactyla inhabit one habitat, and all but two Carnivora inhabit two habitats. In contrast, ID MMEs occurred equally between social (13%, 95% CI 7%–22%) and solitary species (21%, 95% CI 11%–36%), with no significant difference identified between ID MME incidences ($\chi^2 = 1.31$, $p = .25$), as indicated in our MCA analysis. Likewise, no significant difference in ID MME incidence was identified between carnivores (13%, 95% CI 8%–21%) and omnivores (22%, 95% CI 11%–41%; $\chi^2 = 1.37$, $p = .24$).

3.2 | Seasonal factors and infectious disease-associated MMEs

Overall, ID MME occurrence was significantly impacted by season ($\chi^2 = 19.85$, $p = .0002$), with half of these events occurring in summer (50%, 95% CI 34%–66%), 28% (95% CI 16%–44%) occurring in spring, 14% (95% CI 2%–29%) in autumn and 8% (95% CI 3%–22%) in winter. In cetaceans, ID MMEs were split evenly between the summer (42%, 95% CI 19%–68%) and autumn months (42%, 95% CI 19%–68%), with one event each occurring in spring and winter (Figure 4). The majority of ID MMEs in pinnipeds occurred during the summer (62%, 95% CI 44%–77%) and spring months (31%, 95% CI 17%–49%; Figure 4). Collectively, viral outbreaks resulting in MMEs ($n = 26$) spanned all seasons, with the majority occurring in spring (31%, 95% CI 17%–50%) and summer (38%, 95% CI 22%–57%). Bacteria-induced MMEs ($n = 9$) occurred solely in spring and summer, with 89% (95% CI 57%–98%) of these events manifesting

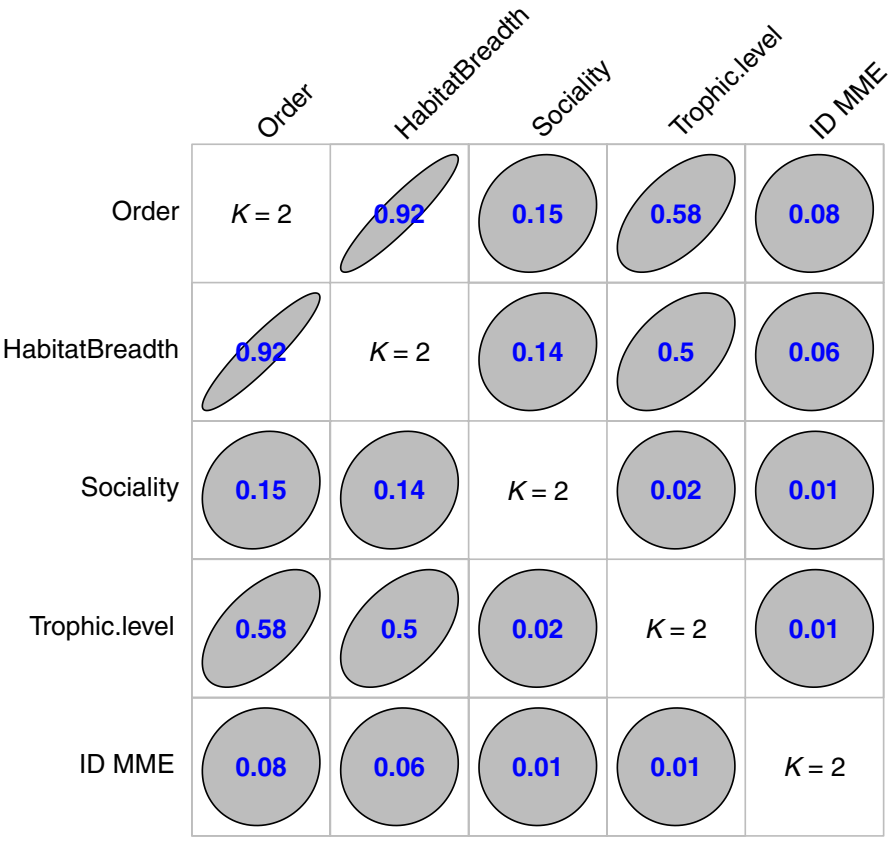

FIGURE 3 Goodman and Kruskal's tau output quantifying asymmetric associations between our five variables (order, habitat breadth, sociality, trophic level and ID MME occurrence). The plot shown illustrates both a graphical and numerical representation of τ. The more highly correlated the variables are, the closer the value to 1 and the more oblong the circle appears. For example, for highly associated variables $\tau = 1$, and the circle will appear as a diagonal line. For variables that are uncorrelated, $\tau = 0$ and the circle will be perfectly round. *K* is the number of categories within each variable, with each variable in this plot having two categories [Colour figure can be viewed at wileyonlinelibrary.com]

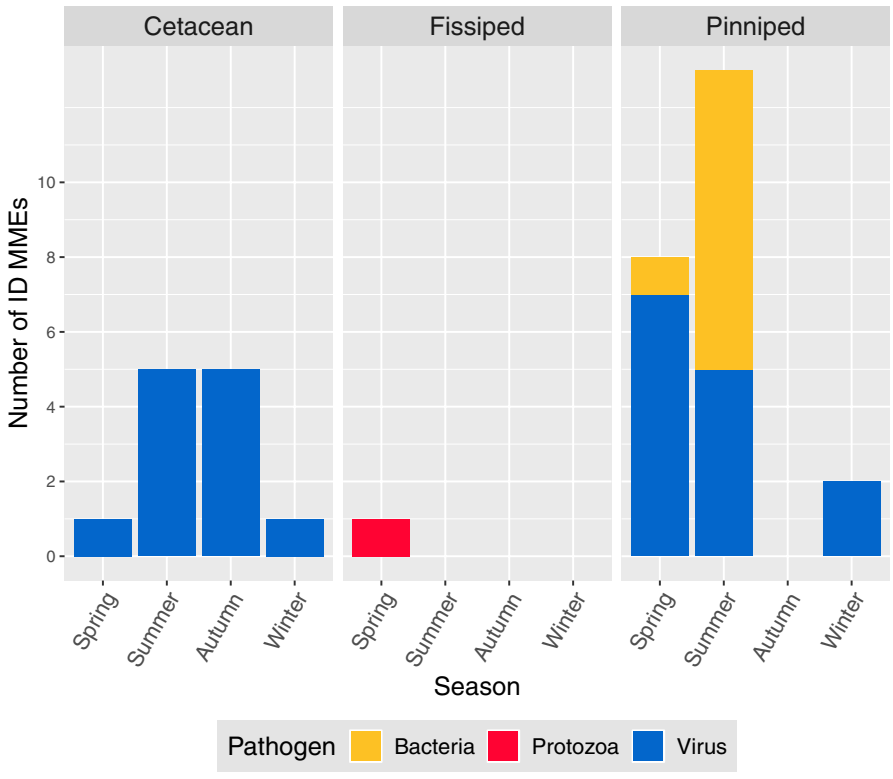

FIGURE 4 Infectious disease-induced mass mortality events (ID MMEs) in marine mammals by pathogen type and season (1955–2018). ID MMEs are separated by marine mammal group (cetacean, fissiped and pinniped) and pathogen type (bacteria, protozoa and virus)

in summer (Figure 4). The only protozoan pathogen-induced MME occurred in the spring. Not including the Baikal seal ID MME where over 100,000 fatalities occurred in a single event during winter, on average, the most fatalities occurred in spring (6,205 fatalities per ID MME outbreak in spring vs. 431 fatalities per ID MME outbreak in summer).

3.3 | Climate factors and infectious disease-associated MMEs

Poisson regressions were conducted to determine relationships between ID MME occurrence and climate variables (SST anomalies and Nino3.4; Table 3). A two degree-of-freedom chi-square test determined that Nino3.4 was not an important predicator of ID MME occurrence (χ^2 = 0.07903, p = .7786) and therefore was not included in the final model (model 2). The goodness of fit chi-square test for model 2 was not significant indicating that the model fits the data (residual deviance = 61.83, df = 62, p = .48). We determined the incident rate ratio from the robust exponentiated coefficient from model 2 (IRR = 11.82, 95% CI 3.13–44.63), signifying that the incident rate of ID MMEs increases by 11.82% for every unit increase in yearly global SST anomalies. Likewise, plotting the predicted number of ID MMEs against yearly global SST anomalies showed an upward trend in the occurrence of ID MMEs with increasing yearly global SST anomalies (Figure 5). When regional SST anomalies were assessed, we found that 61% of infectious disease outbreaks causing MMEs occurred during periods of elevated SSTs (Supporting Information 1 and 2).

3.4 | Evaluation of conservation implications

When considering the conservation status of marine mammals affected by ID MMEs, two species had a negative change in status following an ID MME. These included the Caspian seal (8 years after ID MME; *Vulnerable* to *Endangered*), and the hooded seal (*Cystophora cristata*; 2 years after ID MME; *Lower Risk/least concern [LRLC]* to *Vulnerable*). ID MMEs affecting these species were caused by morbilliviruses. Additionally, two species changed from no IUCN status/ data deficient to a negative status: Mediterranean striped dolphin (No IUCN status to *Vulnerable*) and Guiana dolphin (*Data Deficient* to *Near Threatened*). Four species (Mediterranean monk seal; *Monachus monachus*, Mediterranean sperm whale, New Zealand sea lion and sea otter) remained at the same conservation status following ID MMEs (*Critically Endangered*, *Endangered*, *Vulnerable* and *Endangered*,respectively). Currently, 25% (95% CI 11%–47%) of species/subspecies that have previously succumbed to ID MMEs are considered *Endangered*, 15% (95% CI 5%–36%) are *Vulnerable*, 5% (95% CI 1%–24%) are *Near Threatened*, 10% (95% CI 3%–30%) are *Data Deficient* and 45% (95% CI 26%–66%) are of *Least Concern* (Figure 1). The majority of these species currently have unknown population trends (60%, 95% CI 39%–78%), while 15% (95% CI 5%–36%)

TABLE 3 The results from two Poisson regression models, showing the robust standard errors, 95% confidence intervals (CI) and associated p-values. The final model is shown in grey

Variables	Coefficients	Robust standard errors	95% CI		Z Value	Robust p-value	AIC
			Lower	Upper			
Model 1							
SSTanom	2.4	0.76	1.03	3.76	3.14	5.97e-04	125.1
Nino3.4	0.08	0.28	−0.55	0.71	0.28	.81	
Model 2							
SSTanom	2.47	0.68	1.14	3.8	3.43	2.7e-04	123.2

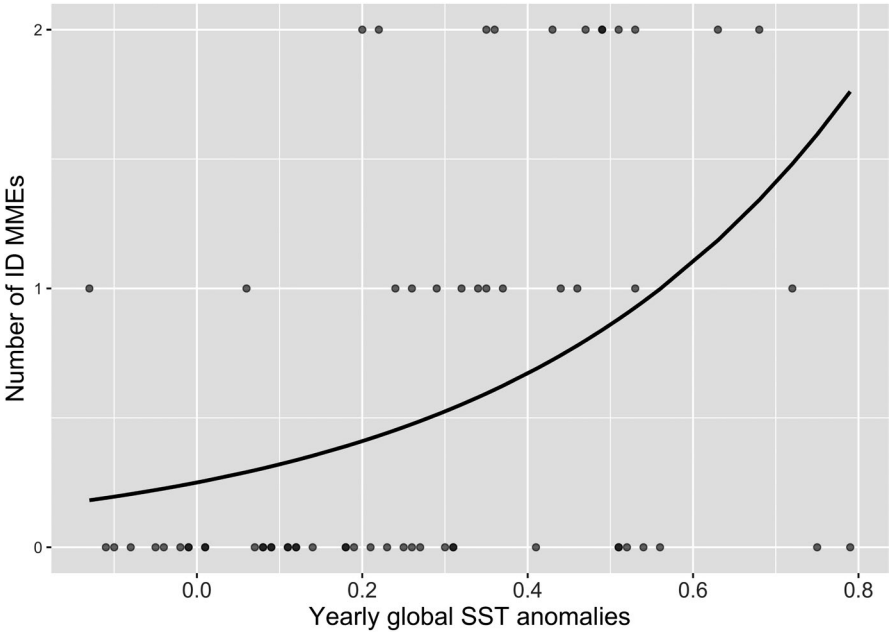

FIGURE 5 Number of infectious disease-induced mass mortality events (ID MMEs) in marine mammals plotted against yearly global sea surface temperature (SST) anomalies (1955–2018). The predicted (expected) number of ID MMEs are shown (black line)

have decreasing population trends, 20% (95% CI 8%–42%) are increasing in population size and 5% (95% CI 1%–24%) have stable populations (Figure 1).

4 | DISCUSSION

Since 1955, infectious disease outbreaks have resulted in 36 separate MMEs affecting 18 marine mammal species. Viruses were the main etiological agents triggering these epizootics, specifically, morbilliviruses and influenza A viruses. The dominant occurrence of virus-associated MMEs in obligate ocean dwellers is likely due to their mode of transmission, with viruses commonly spread through aerosols, or through contact with ocular, oral and/or respiratory fluids favouring transmission in gregarious species (Van Bressem et al., 2014). However, molluscs, such as bivalves and gastropods, can also act as pathogen reservoirs for viruses, including avian influenza (H3N8; Burge et al., 2016) and morbillivirus (Kondratov et al., 2003), respectively. Furthermore, morbilliviruses are multihost in nature, an important attribute that may allow populations to meet host density thresholds necessary for pathogens to emerge and persist (Keeling & Rohani, 2011). This is especially important in marine landscapes which are expansive and characterized by variable and often low host species density. Reoccurring ID MMEs from morbilliviruses were seen in three species (common bottlenose dolphin; *Tursiops truncatus*, harbour seal and striped dolphin) and are likely due to the size and structure of these populations, as well as untimely contact with infected sympatric or migratory species (Supporting Information 1; Gulland & Hall, 2007; Härkönen et al., 2006).

Influenza A was the only other virus implicated in MMEs in marine mammals, but was predominately associated with pinnipeds. Indirect or direct (i.e. faecal–oral route, waterborne transmission) disease transmission between birds and seals, spurred by close contact with birds or bird faeces at pinniped-occupied sites (Harris et al., 2017), has been identified as the possible source of transmission in all four MMEs caused by influenza A viruses in harbour seals (Anthony et al., 2012; Hinshaw et al., 1984; Webster et al., 1981; Zohari, Neimanis, Härkönen, Moraeus, & Valarcher, 2014), and in the only MME caused by influenza A in long-finned pilot whales (Hinshaw et al., 1986).

Similar to influenza A virus transmission, bacterial and protozoan pathogens implicated in MMEs in pinnipeds and sea otters are more likely transmitted through ingestion of contaminated water or food from infected terrestrial hosts (Supporting Information 1). This mode of transmission is more associated with semiaquatic marine mammals that span the marine–terrestrial interface, as recognized by the lack of cetacean MMEs caused by these pathogens. Bacterial pathogens, *Leptospira* and *K. pneumoniae*, accounted for a quarter of infectious disease outbreaks resulting in MMEs, but have only occurred in two marine mammal species, the California sea lion (*Zalophus californianus*; n = 6 leptospirosis outbreaks causing MMEs) and the New Zealand sea lion (*Phocarctos hookeri*; n = 3 *K. pneumoniae* outbreaks causing MMEs). Leptospirosis has been a recurring source of mortality for California sea lions since 1970, with outbreaks ensuing every 3–4 years, and MMEs from leptospirosis occurring every 4–10 years (Dierauf et al., 1985; Gulland et al., 1996; Vedros, Smith, Schonewald, Migaki, & Hubbard, 1971). Interspecies transmission from pigs and small rodents has been suggested as the cause of these outbreaks (Gulland et al., 1996). In 1998, *K. pneumoniae* was introduced into the New Zealand sea lion population (Castinel et al., 2007). While the origin of this pathogen is unknown, transmission among colonies has been potentially linked to cattle on the islands or to researchers working between islands (Baker, 1999). Since the last ID MME in the New Zealand sea lion, *K. pneumoniae* has become endemic within this population and is now a regular cause of pup mortality (Roe et al., 2015). The New Zealand sea lion is currently considered *Endangered*, with the presence of this pathogen threatening population recovery, and greatly influencing this species' vulnerability to extirpation.

The sole ID MME caused by a protozoan pathogen occurred in sea otters located in Morro Bay, California (Miller et al., 2010). Multiple exposure routes were implicated in this outbreak, with heavy rainfall proceeding the event likely flushing concentrated infectious material (originally from infected opossum faeces) from storm water drains into the Bay (Miller et al., 2010). Concurrently, sea otters were concentrated along the coast to consume razor clams that may have contained high concentrations of *S. neurona* sporocytes (Miller et al., 2010). Additionally, evidence suggests that infected sea otters possessed high concentrations of domoic acid, an algae-produced biotoxin, which may have also resulted in immunosuppression and subsequent infection by *S. neurona* (Miller et al., 2010; Supporting Information 1).

4.1 | Host factors and infectious disease-associated MMEs

4.1.1 | Sociality

Both solitary and social marine mammal species were equally affected by ID MMEs, differing significantly from reports in terrestrial carnivores. Indeed, infectious disease has been implicated in population declines in 45% of social terrestrial carnivores, but only in 3% of solitary terrestrial carnivores (Sanderson et al., 2013). The dichotomy between marine and terrestrial mammalian species is likely influenced by a number of factors. Firstly, unlike terrestrial carnivores, the majority of marine mammal species are not territorial. Territoriality adds spatial structuring that may restrict landscape connectivity and host species density, with consequent impacts on pathogen transmission potential (Craft, Volz, Packer, & Meyers, 2011). In aquatic environments with non-territorial species and multihost pathogens, landscape connectivity can be extensive, facilitating pathogen spread and persistence. Secondly, recent studies suggest that marine mammal species that were considered solitary may in fact have more complex social structures

than traditionally understood (Sah et al., 2016). For example, hauling out behaviour in harbour seals may temporarily increase host aggregation and contact despite the lack of gregarious behaviours in this species. Hauling out by pinnipeds involves temporarily leaving the water between foraging for rest, reproduction and molting, as well as predator avoidance and thermoregulation (London, Ver Hoef, Jeffries, Lance, & Boveng, 2012). Indeed, the concentration of seals at haul-out sites have been implicated in outbreaks of morbillivirus in Caspian seals (Kuiken et al., 2006) and crabeater seals (*Lobodon carcinophaga*; Laws & Taylor, 1957). Thirdly, disease transmission studies have traditionally focused on population-level dynamics assuming that all individuals within a population have identical levels of susceptibility and exposure to the disease agent in question (Fairbanks & Hawley, 2012). However, recently it has been shown that the social organization, as well as the type and the focus of social interactions within a group, can determine the disease outcome for individuals, and subsequently, the overall outcome for the group or population (Drewe, 2010; Fairbanks & Hawley, 2012). In many instances, a sex and age bias is present, as seen in leptospirosis outbreaks in California sea lions (Norman, DiGiacomo, Gulland, Meschke, & Lowry, 2008), and morbillivirus outbreaks in striped dolphins (Aguilar & Raga, 1993) and harbour seals (Härkönen et al., 2006), among other species (Supporting Information 1). Some mammalian species may avoid the trade-off between the benefits of group living and the negative impacts of increased contact with conspecifics and infectious disease transmission by employing fission–fusion group dynamics (Kashima, Ohtsuki, & Satake, 2013). In societies employing this mechanism, a group may disperse into smaller groups to avoid disease spread or resource competition, and may fuse back together into a larger group for predator avoidance or information exchange (Kashima et al., 2013). Likewise, fragmented social networks with interconnected subgroups may constrain pathogens to a few subgroups and delay onward transmission (Sah, Leu, Cross, Hudson, & Bansal, 2017).

4.1.2 | Trophic level

Despite a significant amount of research indicating that bioaccumulation can have significant influences on immune regulation in marine mammal species, we did not find any evidence that trophic level contributed to ID MMEs in marine mammals. Marine mammals are highly susceptible to environmental contamination through direct anthropogenic pollution along coasts and shorelines and through bioaccumulation of pollutants due to feeding at high trophic levels within the food web (Desforges et al., 2016; Jessup et al., 2004). Organochlorine (OC) pollutants, polychlorinated biphenyls (PCBs) and heavy metals are the main environmental pollutants in marine mammals (and terrestrial carnivores (Rodríguez-Jorquera et al., 2017)) and due to their highly accumulative nature have known detrimental effects on both the innate and adaptive immune systems, potentially increasing susceptibility to infectious disease agents (Desforges et al., 2016; Lafferty & Gerber, 2002). Pollutants, such as OCs and PCBs, may have increased susceptibility to ID MMEs in Caspian seals (Kuiken et al., 2006), striped dolphins (Aguilar & Borrell, 1994), harbour seals (Härkönen et al., 2006) and sea otters (Miller et al., 2010; Supporting Information 1), however, most of these cases have not been validated. Despite previous research, our results are not surprising given that bioaccumulation is a complex process, and is not solely a function of trophic level position, but also of body size, age, sex, geographic location, marine biome preference and water quality (e.g., pH, salinity and SST), among many other factors (Borgå, Fisk, Hoekstra, & Muir, 2004).

4.1.3 | Order and Habitat breadth

While all marine mammals rely on the marine environment, the degree of reliance differs between species and is an important determinant of ID MME occurrence. Cetaceans (order: Cetartiodactyl) and sirenians (order: Sirenia) are fully dependent on the marine environment, while pinnipeds and fissipeds (order: Carnivora) are semiaquatic, spending some time on land. Anthropogenic land change is an important driver of infectious disease, with the land–water interface an important focus for cross-species transmission and disease spread into novel or altered habitats (Patz et al., 2004). This is particularly true for marine environments, with many marine mammal diseases being water-borne zoonoses. At coastal sites, rainfall is a known cause of pathogenic microorganism transport, with heavy rain mobilizing pathogens and disseminating them into marine environments, such as implicated in the protozoan-associated MME in sea otters (Miller et al., 2010), as well as in *T. gondii* infection in sea otters (Figure 6; Gibson et al., 2011; VanWormer et al., 2016) and beluga whales (*Delphinapterus leucas*; Alekseev et al., 2017). This may explain the higher level of epizootics seen in pinniped species predominately associated with coastal areas compared to cetaceans, many of which are pelagic. However, this could also be due to a detection bias that may have skewed our results. Unlike semiaquatic species, obligate ocean dwellers are challenging to survey such that decreases in population size and/or the occurrence of mortality events might go unobserved (Taylor, Martinez, Gerrodette, Barlow, & Hrovat, 2007). This will be particularly true for cetaceans that inhabit pelagic biomes and those that occupy the Indian and South Atlantic Oceans where survey efforts are minimal (Taylor et al., 2007). Additionally, ocean topographic features, surface ocean currents and weather influence the frequency and distribution of observed marine mammal stranding patterns (McLellan, Friedlaender, Mead, Potter, & Pabst, 2002). Furthermore, the lack of detection of pathogens, specifically morbilliviruses, in cetacean MMEs may also be due to a central nervous system tropism that results in virus colonisation and persistence solely in the brain, a tissue that may not always be sampled during necropsies in the field (reviewed in Van Bressem et al., 2014). Tissue decay or autolysis may also inhibit pathogen screening.

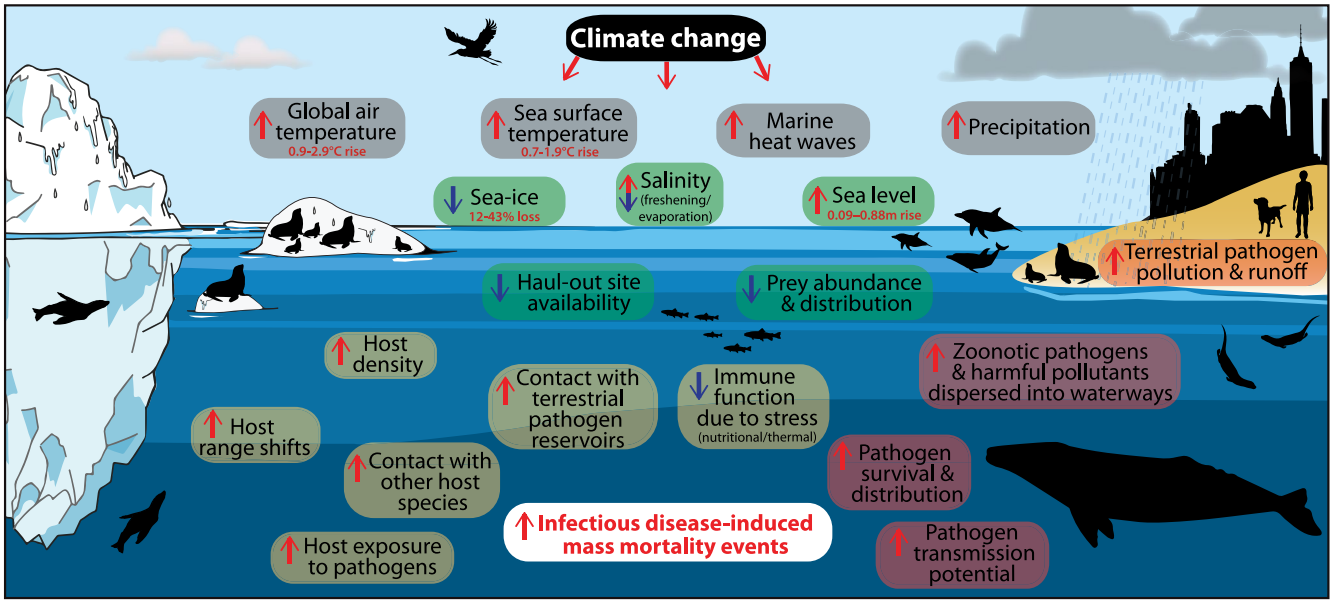

FIGURE 6 Proposed mechanisms for increased infectious disease epizootics causing mass mortality events (ID MMEs) in marine mammals due to climate change. Climate drivers (grey) have the potential to create cascading effects on the host (yellow), pathogen (red) and environment (green), resulting in increased ID MMEs in marine mammal species. Red text provides estimated range impacts on specified climate-impacted variables, as projected to occur by 2070 in relation to the mean levels observed from 1986–2005 (Rintoul et al., 2018).

4.2 | Environmental factors and infectious disease-associated MMEs

Environmental factors are known to catalyse MMEs (Fey et al., 2015). Indeed, climate-controlled meteorological-related factors, such as weather, temperature and oxygen levels, contributed to nearly a quarter of globally occurring MMEs in a recent study conducted across all animal taxa (Fey et al., 2015). Likewise, we have discovered that climate factors, such as seasonality and SST anomalies, influence the temporal occurrence of ID MMEs in marine mammals. The following discussion is based on reported and published data, where cold-water species are dominant and tropical species are undoubtedly underrepresented. Due to this, our discussion is likely skewed towards cold-water species in regions where infectious disease dynamics are presumably influenced by climate variables in differing ways than in tropical climates.

4.2.1 | Seasonality

Seasonality was important in determining the temporal occurrence and severity of past ID MMEs in marine mammals. The increased occurrence of ID MMEs in the summer months is likely due to the amplifying effects of climate change on natural weather patterns. During summer months, cloud cover is reduced allowing a natural increase in SSTs, which are also likely to exceed normal ranges under the influence of global warming. Indeed, out of the 18 ID MMEs that occurred during the summer months, 89% (95% CI 67%–97%) occurred during years with positive global SST anomalies. Additionally, increased intensity and frequency of precipitation, and events associated with these events, such as runoff, flooding and erosion, generally occur during the summer months. As discussed previously, rainfall and urban runoff may aid in microbial and chemical dissemination into marine environments, altering host susceptibility and exposure to pathogens.

While most ID MMEs transpired during the summer months, ID MMEs occurring in spring recorded the most fatalities. For pinnipeds, spring is the start of the breeding season when seals congregate at haul-out sites for long durations. Increased density, and hence, increased contact between potential hosts, may magnify infectious disease transmission potential at these sites. Premature sea ice loss due to increased SSTs may have also resulted in increased contact between pinnipeds and terrestrial disease hosts, while simultaneously increasing host density at haul-out sites, intensifying the threat of infectious disease emergence and transmission (Benson, Croll, Marinovic, Chavez, & Harvey, 2002; Figure 6). In cetaceans, viral outbreaks occurred equally across the summer and autumn months. Seasonal migration likely plays a significant role in outbreaks during the autumn months, when emigration is also highest.

4.2.2 | Climate change

While there is still a dearth of data directly linking climate change to increases in infectious disease outbreaks in marine mammals (Burge et al., 2014), climate-mediated changes are expected to have significant impacts on disease outbreaks in these species (Figure 6). These impacts will no doubt alter both pathogen and host characteristics,

including distribution, susceptibility and survival (Burek, Gulland, & O'Hara, 2008). Indeed, immigration of marine species is predicted to significantly increase in the Southern Ocean and the Arctic due to climate change (Cheung et al., 2009), potentially increasing contact between species, and subsequently, the transmission potential of pathogens.

The warming of marine waters has been identified as a key driver of infectious disease outbreaks in marine systems (Harvell et al., 2002). We discovered that at least 61% of all epizootics resulting in a MME occurred during positive SST anomalies regionally. We chose the first month of the outbreak to focus on regional SST anomalies as most pathogens causing ID MMEs have incubation times that would occur within this window (e.g., morbillivirus incubation time is approximately 3–14 days (Heide-Jorgensen & Harkonen, 1992), leptospirosis is 10–14 days (Norman et al., 2008) and influenza A is less than 3 days (St Aubin et al., 1982)), however, these times may not reflect some host–pathogen systems, with pathogen dose, route of exposure and host susceptibility likely varying. Likewise, using global SSTs alone may not provide the fine-scale insights needed to determine causation. However, similar associations were seen between ID MMEs and global SSTs, and ID MMEs and regional SSTs, indicating that this combination of methods likely provides an accurate representation of the influence of SST anomalies on ID MMEs in marine mammals.

In contrast, our analyses did not indicate that ENSO significantly influences these events. El Niño events are classified as the average of five consecutive 3-month SST anomaly means greater or equal to +0.5°C, while SST anomalies are calculated by subtracting the 30 year climatological SST mean from daily SSTs. Hence, while El Niño likely represents anomalies that are part of the normal climate cycle, SST anomalies indicate unusual departures from the norm and are therefore likely more representative of a changing climate.

Marine mammals are endothermic and need to maintain a constant body temperature. A shift outside their ideal temperature range necessitates additional energy expenditure, potentially increasing stress responses and weakening the immune system. Warming of the oceans has already changed haul-out patterns (location and duration; Lavigne & Schmitz, 1990) due to the early loss of sea ice. Apart from increasing contact rates between conspecifics and terrestrial disease hosts (Benson et al., 2002; Figure 6), early loss of sea ice may also impact the length of time newborns can spend suckling, leading to weaning juveniles having lower body weights and increased susceptibility to infectious disease (Kuiken et al., 2006). This may also occur due to decreased prey availability associated with warming waters (Laake, Lowry, DeLong, Melin, & Carretta, 2018). Additionally, warmer waters may have a significant impact on the pathogen itself by allowing range expansion and/or altered survival rates due to salinity changes (Figure 6; Tucker, Sommerville, & Wootten, 2000).

While all oceans are warming, it appears that oceans in the Northern Hemisphere are warming faster than in the Southern Hemisphere (Abraham et al., 2013). This is particularly true for the North Atlantic (Abraham et al., 2013), where many marine mammal epizootics have already occurred (Figure 1). Indeed, a greater percentage of ID MMEs have occurred in the Northern Hemisphere, with only three disease events occurring in the Southern Hemisphere. However, as stated earlier, this may be indicative of an observation bias considering marine mammals are more well-studied in the North-Eastern Pacific and northern Atlantic oceans (Schipper et al., 2008).

The recently observed anomaly of marine heat waves (MHWs), described as prolonged warm SSTs lasting from days to months (Frölicher & Laufkötter, 2018), further increase the vulnerability of marine mammals to ID MMEs. MHWs have already been documented in regions where marine mammals are already susceptible to MMEs, including the Mediterranean Sea (2003; Black, Blackburn, Harrison, Hoskins, & Methven, 2004), the northwest Atlantic (2012; Chen, Gawarkiewicz, Lentz, & Bane, 2014) and the northeast Pacific Ocean ('The Blob'; 2013–2015; Di Lorenzo & Mantua, 2016). The MHW in the northeast Pacific Ocean was implicated in the largest recorded harmful algal bloom and caused mass strandings of California sea lions, Guadalupe fur seals (*Arctocephalus townsendi*), baleen whales (*Mysticeti* spp.), fin whales (*Balaenoptera physalus*) and sea birds (reviewed in Cavole et al., 2016). MHWs are expected to increase in magnitude and occurrence with global warming (Oliver et al., 2018). Correspondingly, marine mammals that require ice and cold water environments to survive, as well as those species inhabiting semienclosed seas, such as the Mediterranean Sea, equatorial regions and subarctic environments are predicted to be the most vulnerable to climate change (Cheung et al., 2009; Helmuth et al., 2013). Indeed, local extinctions from climate change are predicted to occur predominately in these regions, as well as in the north Atlantic Ocean and the northeast Pacific Ocean (Cheung et al., 2009), regions where MHWs and infectious disease have already caused MMEs in marine mammals (Figure 1).

4.3 | Conservation and management implications

Due to the limited availability of baseline data for many marine mammal species, it is difficult to predict the current and future impact of ID MMEs, especially in the face of climate change (Helmuth et al., 2013). Currently, 37% of marine mammals that have historically suffered from an ID MME have a negative IUCN status (*Endangered* or *Vulnerable*), with over a quarter having decreasing population trends. While IUCN status is not solely a reflection of MME occurrence in these species, ID MMEs could greatly influence overall vulnerability to extirpation. Climate-related factors are also known to magnify non-climate threats in marine species (Helmuth et al., 2013), resulting in the exacerbation of current species-specific and ecosystem stressors.

Two mortality events were included in our data set even though they were not identified by the authors as MMEs, massive die-offs or UMEs, as these events occurred in the Mediterranean Sea where these subpopulations are under threat. These included a dolphin morbillivirus outbreak in sperm whales (*Physeter macrocephalus*)

resulting in the death of three adults and one foetus, and a pilot whale morbillivirus outbreak resulting in 21 deaths of long-finned pilot whales (*Globicephala melas*). The Mediterranean sperm whale subpopulation is considered *Endangered* with a decreasing population trend (Notarbartolo di Sciara, Frantzis, Bearzi, & Reeves, 2006) based on current, unsustainable, observed mortality levels (Rendell & Frantzis, 2016). While IUCN data are limited for the Mediterranean long-finned pilot whale subpopulation (Cañadas, 2012), fewer than 250 individuals exist and the population has declined by 26.2% over 5 years since the outbreak (Verborgh et al., 2016). Additionally, studies predict an 85% chance that this subpopulation will become extinct within the next 100 years (Verborgh et al., 2016). As such, even the death of a few individuals in these species greatly increased their risk of extirpation.

While our results implicate SST anomalies and habitat breadth as two important variables associated with the occurrence of ID MMEs in marine mammals, the source of infection and the transmission dynamics of the majority of these disease outbreaks remain elusive. Without historical baseline data on many of these species, it is hard to discern whether pathogens have been historically present within these populations, or they have recently emerged or been introduced. Furthermore, MMEs are most often associated with multiple stressors (Fey et al., 2015; Kock et al., 2018). Thus, the disease agents reported as being implicated in MMEs here, may have only been a compounding factor. Concurrent infections (Kuiken et al., 2006), malnutrition, biotoxins (Aguilar & Raga, 1993) and/or heavy metal presence may also contribute to MMEs in marine mammal species. Indeed, concurrent infections were discovered during infectious disease outbreaks resulting in MMEs in Caspian seals (Kuiken et al., 2006), New Zealand sea lions (Duignan, Wilkinson, & Alley, 2003), common bottlenose dolphins (NOAA, 2015), California sea lions (Norman et al., 2008) and harbour seals (Krog et al., 2015; Supporting Information 1). Changes in pathogen incubation period, dispersal rates or virulence, unrelated to host life-history characteristics, may also play an important role in ID MMEs in marine mammals (Nunn, Thrall, Stewart, & Harcourt, 2008). For example, sexual recombination among *T. gondii* isolates in the marine environment has already resulted in the emergence of a hypervirulent strain resulting in sea otter mortality (Grigg & Sundar, 2009).

Increased and improved marine mammal surveillance, together with infectious disease surveillance, is critical for long-term population health monitoring of marine mammals. Increasing SSTs and infectious disease outbreaks are not the only risk climate change brings to marine life, with changes in precipitation, as well as indirect effects from nonlinear and amplifying effects, also having significant impacts on marine organisms and ecosystems (Marcogliese, 2016). Researching the effects of these additional climate and environmental variables on infectious disease emergence, as well as anthropogenic effects, such as habitat loss and fragmentation, will also provide essential data for developing early warning frameworks, forecasting models, species- and location-specific adaptive management plans (Burge et al., 2014), and most importantly, for mitigating the impacts of climate change on these species. Due to the large number of marine mammals with no data or extensive data gaps, and a lack of resources to carry out detailed surveillance on all species, conservation efforts and funds will need to be prioritized. However, modelling efforts should not only focus on keystone marine mammal species that already have long-term population data sets and are highly susceptible to climate change (Silber et al., 2017), but also lesser studied populations, particularly in regions where anthropogenic threats are great yet mitigation efforts are lacking. The 18 marine mammal species already known to have suffered from ID MMEs are also important species in which monitoring and adaptive management plans should be developed.

There is undeniable evidence that the oceans and the biota inhabiting them will be significantly influenced by climate change (Howes, Joos, Eakin, & Gattuso, 2015). For all involved, survival will depend on their ability to adapt to these new and changing conditions. While we have limited control over environmental variables, a reduction in ID MMEs in marine mammals may be achieved by minimizing non-climatic stressors on these populations (Burge et al., 2014). By managing pollution, terrestrial run-off and habitat loss, exposure to pathogens likely to cause infectious disease outbreaks may be reduced.

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CONFLICT OF INTEREST

We have no competing interests.

AUTHOR CONTRIBUTION

C.E.S. compiled the data, conducted the data analyses, interpreted the results, and drafted and revised the manuscript. K.A.A. developed the study concept, conducted critical revision of the manuscript and provided funding for the study. Both authors gave final approval for publication.

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SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section.

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