A framework for understanding how and why animals die

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Glossary:

- **Outcome:** The final, observable endpoint of a causal process, such as death or survival.
- **Driver:** An intrinsic or extrinsic factor that initiates or modifies causal pathways leading toward an outcome.
- **How:** The immediate, observable mechanism by which death occurs.
- **Why:** An upstream driver or condition that increases vulnerability to mortality, shaping risk prior to the final fatal event.
- **Phenomenon:** An emergent pattern or event arising from the interaction of multiple drivers, mechanisms, and contexts.
- **Vulnerability:** The state-dependent susceptibility of an individual to mortality, produced by interactions between intrinsic states and the extrinsic environment.

The Foundation. Mortality is a fundamental demographic process that shapes both populations and ecological communities. Yet, *how* and *why* animals die is just as important as the simple fact of *whether* or *not* they do. A richer understanding about drivers of death across taxa is needed to advance ecological theory and to improve conservation practice [1]. Both require identifying the causal pathways that lead to death; misattributing mortality sources, such as emphasizing predator control when declines are actually driven by food limitation or habitat loss, can lead to ineffective interventions and incorrect inference. These gaps are especially important as climate change and human disturbance alter both vulnerability drivers and mortality outcomes.

Natural selection operates through mortality that depends on both the animal's intrinsic state and its environmental context. The interaction between these elements determines an individual's vulnerability to mortality, and is therefore a central feature of life-history theory. Despite this importance, we still lack a clear understanding of not only *how* most animals die (the immediate, observable event that causes death), but also *why* they were vulnerable to death in the first place. Most research focuses on either *how* or *why* individually, but very few studies examine both. This lack of integration obscures the ecological processes that lead to mortality and can hide opportunities for intervention. Moreover, this lack of integration limits our ability to synthesize across studies and uncover broad macroecological patterns in the causal pathways leading to mortality. We argue that there is a need to conceptually distinguish and identify upstream drivers (*whys)* from downstream mortality events (*hows*) and to examine their interaction in causal pathways. Clarifying the conceptual framework within which animal mortality is detected and understood has the potential to reveal new insights in this fundamental eco-evolutionary process, and to provide a mechanistic basis for understanding and addressing the drivers of biodiversity loss. Here, we provide this conceptual framework for understanding the *why* and *how* of mortality by mapping linkages between the two across space and time.

The Puzzle. Although current research often focuses on determining *how* animals die (e.g., carcass necropsies to determine cause of death) or *why* animals die (e.g., mark-recapture to estimate variables associated with apparent survival or mortality), theory and emerging tools demonstrate that studying either without the other gives an incomplete understanding. For example, a recent global synthesis on animal mortality found that predation and human harvest overwhelmingly dominated mortality in biologging studies, whereas starvation was seldom identified as the cause of death [2]. However, theory [3–7], simulation models [8] , and empirical studies [9,10] consistently show that individuals in poorer condition engage in state-dependent riskier behaviors, making them disproportionately vulnerable to predation or harvest. In these cases, poor body condition plays a key role in *why* an animal died, even though starvation rarely appears as the causal *how* of death. More generally, the full causal pathways leading to death in animals are rarely characterized, yet doing so could fundamentally change how mortality is interpreted by revealing how deeper processes shaping individual vulnerability affect the final causes of mortality.

The Point. Mortality is more than an endpoint; it is the product of multiple, sometimes interacting, drivers. Morbidity, which includes states of reduced health or impaired function that precede death, influences the critical pathways leading to death. Synthesizing both the factors predisposing animals to death and then identifying the actual cause of death moves us toward a clearer, more comprehensive framework for understanding mortality both now and in the future. Doing so requires moving beyond simply either categorizing the final cause of death or identifying upstream causes, and instead mapping the causal pathways leading to mortality. Mapping more complete causal pathways offers a unifying perspective for integrating diverse approaches to understanding how and why animals die by revealing the *whys* and *hows* that unfold across space and time.

There are many factors that influence *why* and *how* animals die (Figure 1), and the strengths, categorizations, and orders of influence of these drivers vary widely with scale and context. Our aim is to describe the structure of possible causal pathways linking drivers to mortality, rather than to assign mortality to single individual causes. Existing conceptual frameworks, including consumptive and non-consumptive effects [11], costs of reproduction [12], and carry-over effects [13], provide crucial insights into specific components of mortality risk. Here, we integrate these perspectives to build a more complete picture of how interacting drivers can ultimately lead to death, which we hope will facilitate new avenues of research.

The Framework. We can gain a richer understanding of mortality by developing a conceptual framework and terminology for describing mortality (Figure 2). Drawing from the literature on causal path diagrams [14], we propose a framework for describing causal pathways ending in mortality. Causal mortality pathways have four components: *whys*, *hows*, chain structures, and attributes of drivers (*whys*) and links. First, we make a clear distinction between *why* and *how* mortality occurs, which highlights the value of tracing causal chains rather than focusing solely on either drivers (*whys*) or outcomes (*hows*). The *how* refers to the immediate, observable event that causes death, whereas the *why* captures the upstream drivers that create vulnerability in the first place. Determining both is essential for a full understanding, yet the mapping between them is usually only hypothesized. Second, hypothesized causal chains can include different structures including sequential, additive, or interactive (Figure 2), and determining which structure applies is an important step in analyzing the influence of multiple drivers. Third, causal chains include both driver attributes (e.g., internal/external, biotic/abiotic) and link attributes (e.g., acute/chronic and overlapping/distant). For example, long-term energetic stress of an

ungulate caused by unfavorable environmental conditions can interact with an individual's body condition (*why*) to increase susceptibility to an acute lethal event, such as predation (*how*), illustrating a sequential causal chain with chronic, distant drivers. Driver and link attributes are intended to be flexible tools to support conceptual brainstorming and hypothesis generation as well as to guide data collection and analysis. We anticipate many additional system-specific attributes will help clarify how interacting factors across space and time culminate in mortality. Together, these four elements emphasize that understanding animal mortality requires identifying both the immediate cause of death (the *how*), and also identifying the structure, attributes, and pathways that connect underlying drivers (the *whys*) to the final outcome.

The Examples. Distinguishing *why* animals die from *how* they die matters because the immediate cause of death often does not reveal the underlying drivers of vulnerability. A separate but related challenge is that the same immediate *how* of death can arise from very different underlying *whys*, creating multiple pathways to similar endpoints. Although studies that explicitly test the full causal chain leading to death would be ideal, they are exceedingly rare, given the logistical challenges involved; as a result, inference often requires integrating evidence across multiple studies. Below, we illustrate several causal chains that include one or more drivers (*whys*) influencing mortality as well as the final cause of death (*how*) to show how inference about mortality can be incomplete or incorrect when only part of the causal pathway is observed (Figure 3).

A dead white-tailed deer at a wolf kill site, for instance, might be classified as predation, yet if the deer prey was in poor condition or diseased, this understanding of the *how* of death is incomplete and does not include these deeper *whys* (Figure 3A) [15]. Elephant seals provide a parallel example: individuals that fail to return to haul-out beaches are often assumed to be killed by predators [9], yet emerging evidence shows that foraging success is tightly linked to oceanographic conditions that shape vulnerability via state-dependent risk-taking long before any predation event occurs (Figure 3B) [16]. Without incorporating animals' energetic state and state-dependent foraging, and their interaction, we risk over-attributing mortality to predators while underestimating the role of environmental conditions and resource dynamics, and misinterpreting demographic responses to environmental change. These distinctions have real management consequences, underscoring the need to recognize when vulnerability is shaped by resource environments, by predators, or by both acting together.

Conversely, revealing the drivers of mortality rates (*whys*) without following the causal chain to identify *how* an animal dies results in a similarly incomplete picture of vulnerability and ecological process. For example, a growing body of work on migratory birds shows that climatic conditions during the nonbreeding season affect subsequent survival [17,18], mediated by individual body condition and migration phenology [19,20]. Yet in these cases, the immediate cause of death remains unknown, with hypotheses ranging from starvation to compensatory increases in risk-taking that elevate exposure to predation or collisions with human infrastructure. Given the catastrophic decline of North American avifauna over the past half century [21], resolving the full causal pathways linking environmental drivers to specific causes of death is an urgent conservation priority.

Similar challenges arise when environmental change alters animal movement and exposure to anthropogenic hazards. For example, reduced water availability associated with drought can increase animal movement across roads, leading to elevated vehicle collisions, as shown in long-term amphibian roadkill studies where mortality increased

during dry conditions as individuals moved farther to locate surface water (Figure 3C) [22]. In migratory birds, urbanization and associated light pollution alter nocturnal flight behavior and concentration patterns during migration, substantially increasing collision mortality with buildings, even though the immediate cause of death is often classified simply as impact trauma (Figure 3D) [23]. In Yellowstone, the immediate cause of death for many bison is human removal by management agencies, yet the underlying drivers include increasing population size and severe winters that push animals beyond park boundaries in search of forage, thereby increasing exposure to culling intended to reduce disease transmission risk (Figure 3E) [24]. In both cases, human-caused mortality represents the *how*, while environmentally driven movement and resource limitation constitute the deeper *why*.

Environmental context can also mediate mortality through interactions with predators and disease. For example, top predators can displace mesopredators into human-dominated landscapes, where mortality rates increase roughly threefold, primarily from vehicles and other human causes [25]. Body condition further interacts with disease to determine survival outcomes: bats affected by white-nose syndrome ultimately die from starvation because the pathogen disrupts hibernation and accelerates fat depletion, forcing winter foraging in subfreezing conditions [26], yet individuals entering hibernation with larger fat reserves are less likely to die (Figure 3F) [27]. In marine systems, changes in oceanographic conditions have also shifted the seasonal timing of baleen whale migrations in central California, causing humpback and blue whales to arrive earlier and remain longer in productive coastal waters. This shift increases temporal overlap with pot and trap fisheries, amplifying entanglement risk and, in some cases, leading to death through chronic energy depletion and starvation (Figure 3G) [28,29]. Resource-driven foraging shifts can similarly route animals onto hazardous food sources, as illustrated by Gyps vultures feeding on livestock carrion contaminated with veterinary diclofenac, resulting in acute renal failure and widespread mortality that drove rapid population collapse (Figure 3H) [30]. Taken together, these cases illustrate that interacting ecological and physiological processes often operate well before death, underscoring the importance of tracing full causal pathways.

Additional examples, not illustrated in Figure 3, further underscore these hazards. In some systems, starvation can dominate mesopredator or herbivore mortality in the absence of top predators, whereas predator reintroduction or recovery shifts mortality toward predation while increasing food availability and reducing starvation. In long-lived animals, tooth wear can limit foraging efficiency late in life, shifting prey choice or foraging location and exposing individuals to new predation or anthropogenic risks. Even seemingly stochastic events such as avalanches may reflect deeper vulnerabilities caused by climate or land-use change that alter food availability and push ungulates into high-risk terrain. Together, these examples demonstrate that understanding mortality requires linking proximate causes with the deeper ecological, physiological, and life-history processes that shape vulnerability.

The Approaches. To identify the cause of mortality (the *how*), researchers must first identify animals that have died, and then determine the cause of death. Finding animals that have died is possible through four approaches that differ in generality, precision, and biases (Figure 4).

1) Mass mortality events - Detect carcasses through satellite images [31] and citizen science/reports in rare cases where animals are large and/or accessible enough to be found.
2) Cause-specific discovery - Find and study individual animals that have died from a specific cause (e.g., harvest, bycatch [32], window or vehicle strikes). This approach is

biased toward accessible carcasses, often in terrestrial environments, that persist long enough to be located before removal by scavengers.

3) Following stationary animals - Examine causes of death, including predator identity and spatial and temporal patterns of mortality, through focal monitoring of nestlings or eggs in bird nests [33], young mammals in dens, or stationary animals (e.g. marine invertebrates). This is limited to a subset of taxa and a portion of the life history of the species.
4) Transmitting biologgers - Identify the place, timing, and cause of death. Some transmitting biologgers can also directly inform the cause of death (the *how*). For example, location loggers on prey [34] or predators [35], proximity loggers on predators and prey, as well as accelerometers, video cameras, acoustic recorders [36], temperature loggers [37,38] or heart rate sensors [39] on prey, can detect predator encounters or movement and physiological signatures consistent with starvation, disease, heat stress, or human disturbance.

In all four approaches for finding dead animals, necropsies on the carcasses can sometimes identify a cause (*how*) of death [40]. However, only approach (4), transmitting biologgers, can provide a relatively general estimate of the relative frequency of different causes of mortality within a population and across individual lifetimes [2]. Unfortunately, transmitting biologgers remain relatively expensive and are often large or heavy, which can limit the range of taxa that can be studied, and their use requires animal handling and sometimes more invasive procedures such as implanted loggers. However, costs and device size are rapidly decreasing, expanding taxonomic scope and enabling much larger sample sizes without increasing animal disturbance or risk of harm [41].

To identify factors associated with mortality (the *whys*), one approach is to compare the traits, behaviors, or environments of animals that died with those that survived. Location transmitting biologgers provide fine scale information on the environments and movement behaviors of animals. These data can capture conditions at the immediate time and place of death, representing acute and overlapping drivers, as well as longer term histories preceding death or survival, representing chronic drivers that may be overlapping or temporally distant. Environmental variables derived from remote sensing, such as land use or land cover [42], buildings, roads, and food availability including NDVI [43], measures of predator presence or abundance using eDNA [44], acoustic recordings especially in marine systems [45], camera traps [46], vehicle traffic [47], and other factors that influence movement, predation risk, or foraging can be linked to individuals in space and time using location transmitting biologgers. These methods are varied in their resolution, need for validation, and ability to capture illicit human activities. In addition, traits measured at handling (for mark-recapture or biologging) or necropsy [48] such as age, size, body condition, genetic inbreeding or mutation load [49], epigenetics [50], pathogen infection [51], immune function, or diet inferred from stable isotope analysis [52], as well as data from additional onboard sensors, can be compared between animals that died and those that survived to identify factors influencing mortality (*why*).

Another approach for identifying *whys* or contributors to mortality are mark-recapture studies [53]. Mark-recapture approaches provide insight into the *whys* of mortality, including individual traits, as well as environmental factors that vary in space or time such as climate, habitat, and resource limitation, but cannot resolve *how* animals die because mortality is never directly observed. Mark-recapture studies also can't link the fine scale environmental dynamics described above to individual deaths. As a result, mark-recapture studies can quantify only a limited subset of *whys* at the individual level (constant traits measured at

marking/recapture), and another set at the population level on a coarser time scale (i.e. the recapture interval).

Different empirical methods and approaches tend to capture different parts of the causal chain leading to animal mortality, resulting in either unobserved *whys* or *hows* depending on the approach used (Figure 4). Most approaches cluster around either pre-mortem inference of *why* or post-mortem reconstruction of *how*, leaving a critical gap in approaches that follow individuals prospectively through the full causal chain, to capture the transition from vulnerability to event to outcome. This gap makes it far harder to understand the processes by which mortality occurs, particularly when multiple interacting drivers are involved.

The Future. Going forward, the key challenge is not only generating more data, but determining which links in mortality causal chains remain poorly understood and require stronger conceptual, empirical, or integrative approaches. In Box 1, we provide a starting point for clarifying where inference about mortality breaks down, and where future work is most needed. By examining the full causal chains leading to animal death, we can better understand the hidden drivers of population change. Initial brainstorming efforts that draw out hypothetical causal chains can clarify the links to study, help distinguish organismal from environmental sources of variation, and determine when and where critical connections occur. Such *a priori* work can clarify inferential frameworks, reveal new targets for study, guard against spurious inference, and facilitate the choice of analytical methods [54,55]. Empirical studies can then produce data-informed causal chains, and comparisons of the chain structures can be used as a basis for describing and comparing mortality processes across taxa and contexts, and identify priorities for future research. Together, these tools will clarify the mechanisms that shape mortality in wild animals and strengthen our ability to detect vulnerability early enough to intervene effectively.

Acknowledgements. The authors wish to thank the Beltran Lab, Justine Smith, and Chris Wilmers for helpful conversations, and Conner Hale and Carmella Re for research support. This work was funded by the Packard, Sloan, and National Science Foundations, and generous gifts from the Elysea and Lind Family Charitable Funds.

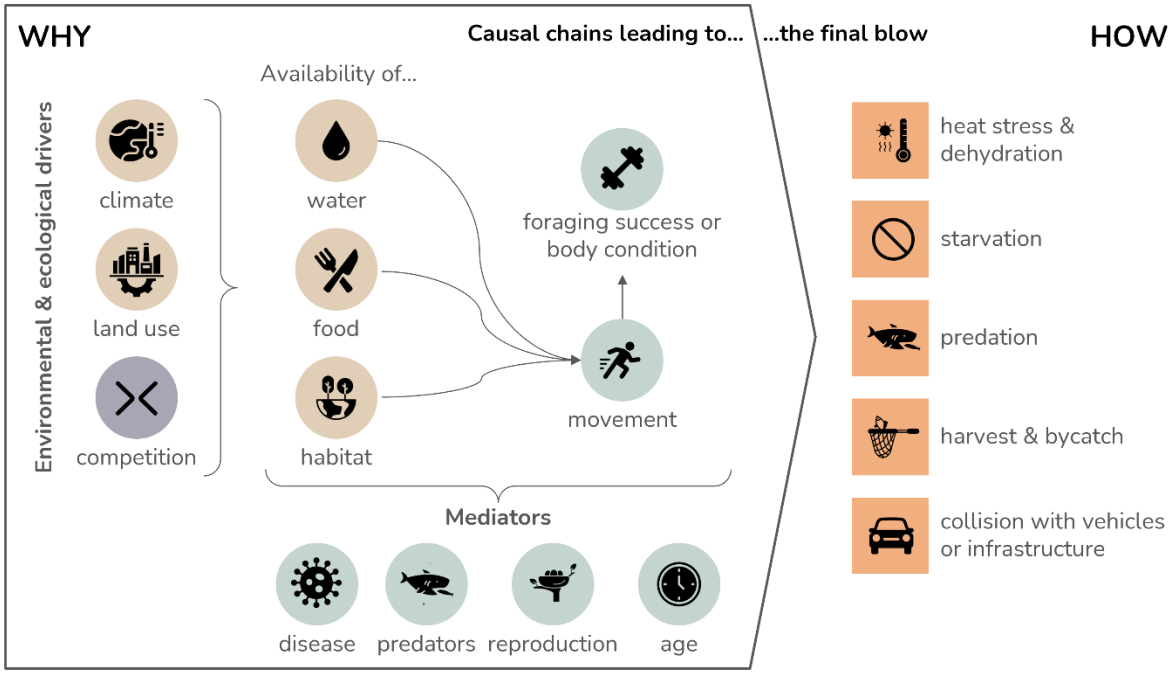

Figure 1. Conceptual web linking potential drivers (*whys*) of mortality to proximate causes of death (*hows*). Environmental and ecological drivers influence mortality indirectly through interacting mediators such as movement, body condition, disease, and life history state, culminating in diverse proximate causes of death. The shape, strength, and ordering of these causal pathways remain incompletely understood, but conceptualizing these pathways could spark hypotheses and guide future research into how multiple drivers combine to produce mortality outcomes in wild animals.

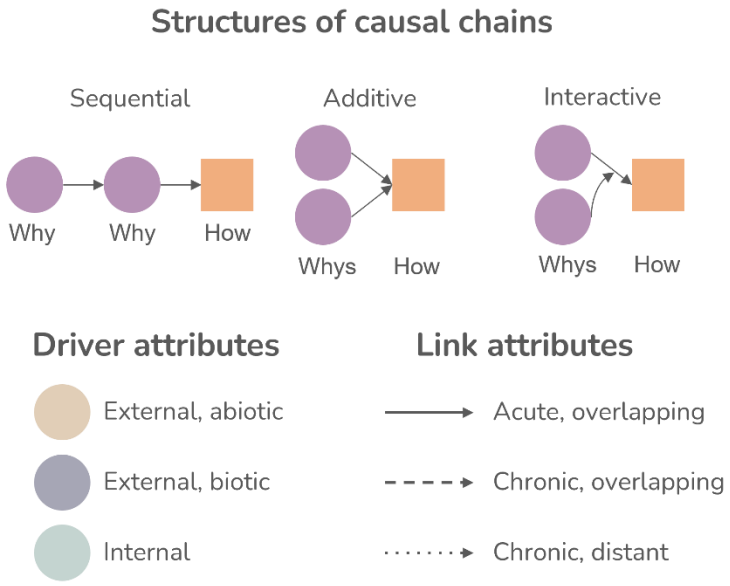

Figure 2. Simplified structures and attributes of causal chains linking drivers to mortality outcomes. Highly idealized examples illustrate sequential, additive, and interactive causal chains connecting proximate mechanisms (hows) to underlying drivers (whys). A standardized language including symbols and arrow styles denoting driver and link attributes can facilitate comparison, synthesis, and cumulative inference across studies.

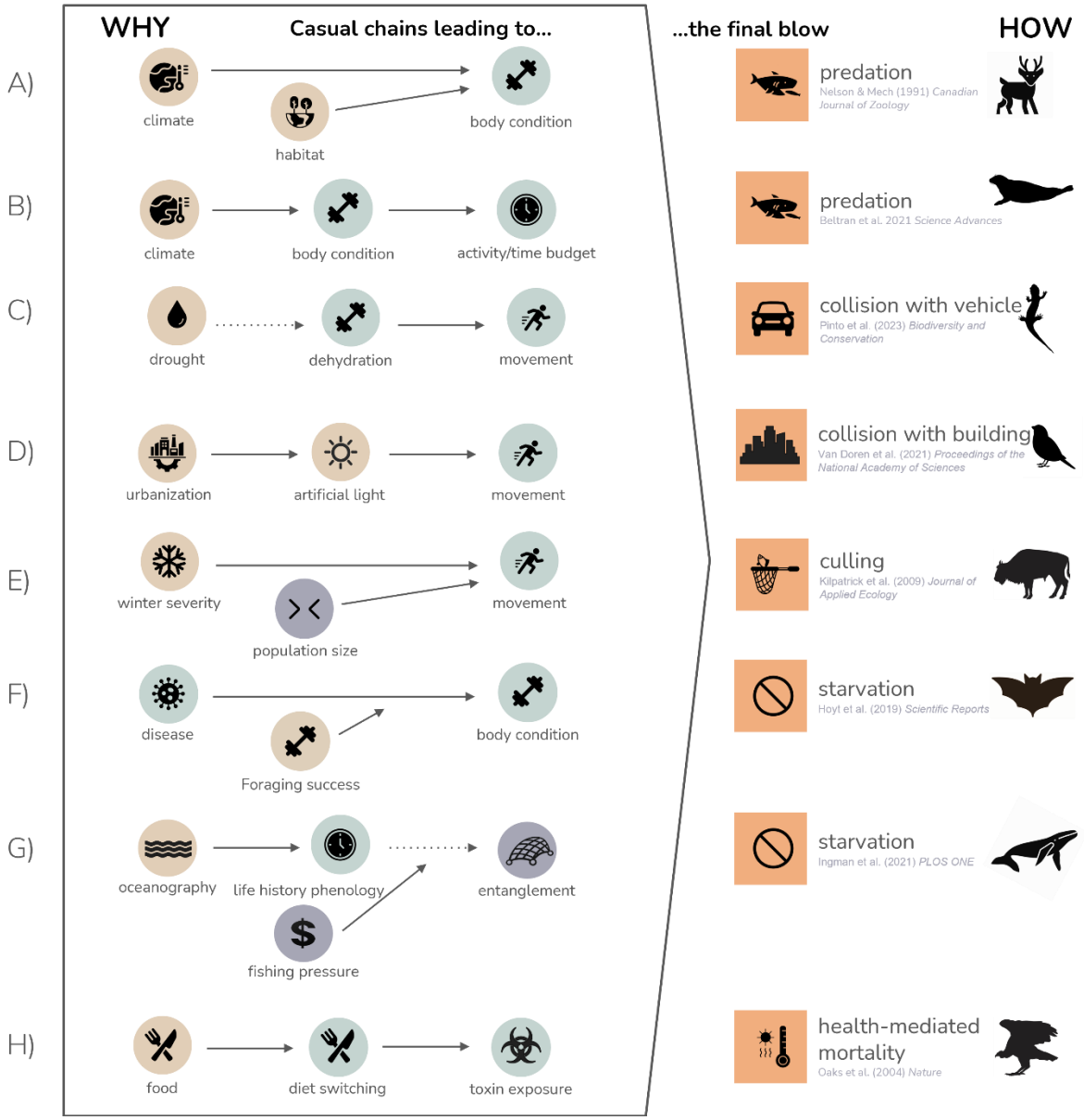

Figure 3. Causal chains linking underlying drivers (*whys*) to the final cause of mortality (*how*), modified from published studies [9,15,22–24,26,28,30]. For example, Causal Pathway A shows how two external abiotic drivers (climate and habitat) influence internal state (body condition) of white-tailed deer which ultimately increases the likelihood of a mortality *outcome* (predation). The examples emphasize that distal drivers often contribute to mortality indirectly through intermediate physiological and behavioral processes, and that the final cause of death may obscure the underlying drivers that shaped risk. Missing information on either *whys* or *hows* can therefore lead to incomplete inference.

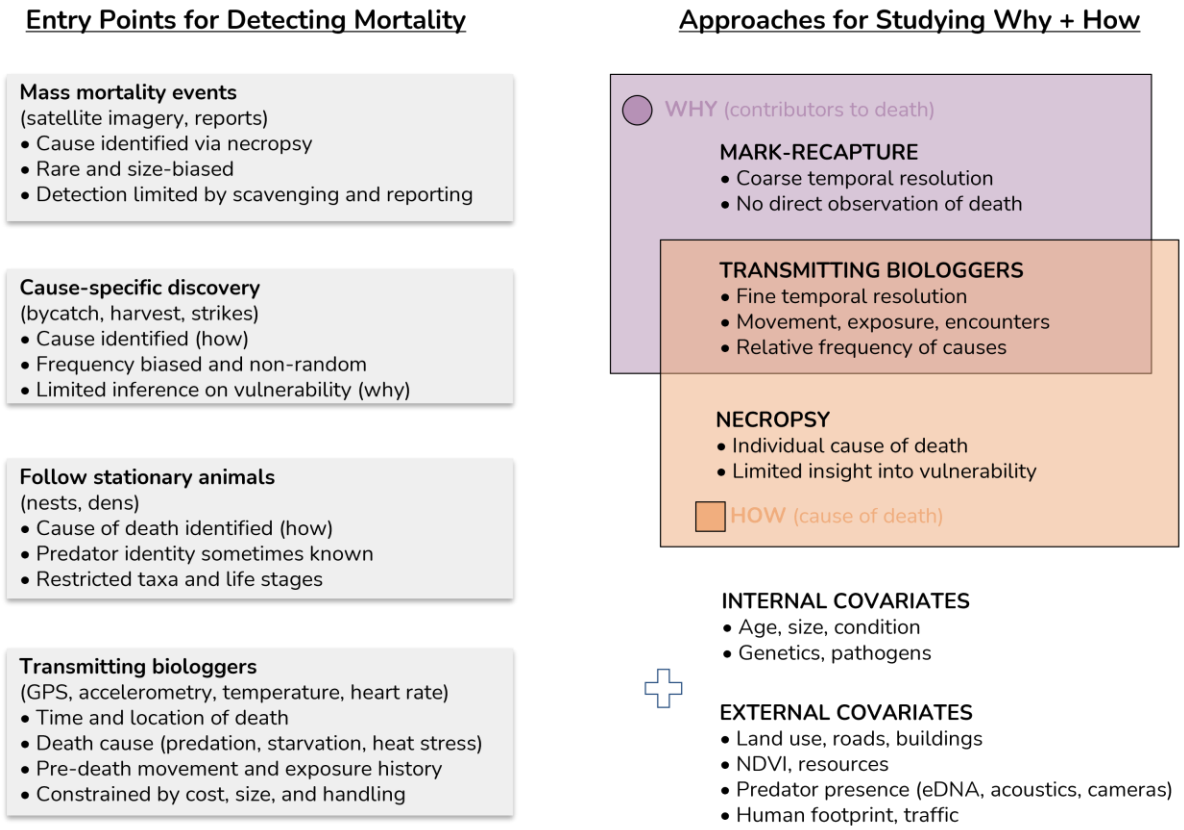

Figure 4. Approaches for detecting (left) and understanding (right) wildlife mortality. Integrating multiple approaches across scales/whys/hows is often required to move from documenting mortality events to understanding their causes.

- Integrate data from mark-recapture, biologging, and individual state measurements to connect temporal indicators of condition, movement, foraging, and physiology to subsequent survival and mortality outcomes [56].
- Improve estimates of starvation thresholds [57].
- Quantifying how risk and hazard landscapes vary across both space and time (e.g. seasons and time of day) [58].
- Clarifying trade-offs between life-history timing and mortality processes [59].
- Incorporate consistent individual behavioral differences, such as personality or risk-taking tendencies, which can mediate both why and how mortality occurs and can generate testable hypotheses about variation in vulnerability [60].
- Leverage recent work on cumulative adversities [61] to understand how multiple stressors often contribute to mortality.
- Expand research beyond large, harvested, terrestrial adults into smaller taxa and life stages where mortality mechanisms are less understood [62].
- Identify the hows and whys of mortality for ecosystem sentinels to understand environmental change [63].
- Identify truly stochastic mortality sources that lack clear *whys* [64]
- Quantify multi-generational impacts, such as offspring loss driven by parental exposure to predators, humans, or reduced food availability.

Box 1. Key gaps and priorities for understanding mortality causal chains.
This non-exhaustive set of examples highlights conceptual and empirical needs for linking individual condition, behavior, spatial context, life history, and environmental drivers to survival and mortality outcomes, and for identifying where inference remains limited by incomplete causal chains.

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