EDITORIAL

Physiological processes through which heatwaves threaten fauna biodiversity

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In recent years, with the intensification of global warming, heatwaves have significantly increased in frequency, extent, and intensity. These heatwaves, along with secondary disasters such as droughts and wildfires, have severe impacts on natural ecosystems.[1] High-intensity heatwave events can trigger mass mortality among fauna, causing detrimental impacts on biological communities. However, the specific mechanisms by which heatwaves threaten fauna biodiversity remain largely unclear.

The physiological homeostasis of individuals is crucial for determining functional stability and fitness under heatwave conditions, and thus forms the basis for understanding the threat of heatwaves to biodiversity. To comprehensively understand the threats of heatwaves to fauna biodiversity, it is essential to elucidate the processes and underlying physiological mechanisms through which heatwaves suppress individual fitness. Here, we summarize some effects of heatwaves on physiological homeostasis and fitness, which can potentially serve as a framework to examine the impacts and approximate mechanisms of heatwaves on animals (Figure 1).

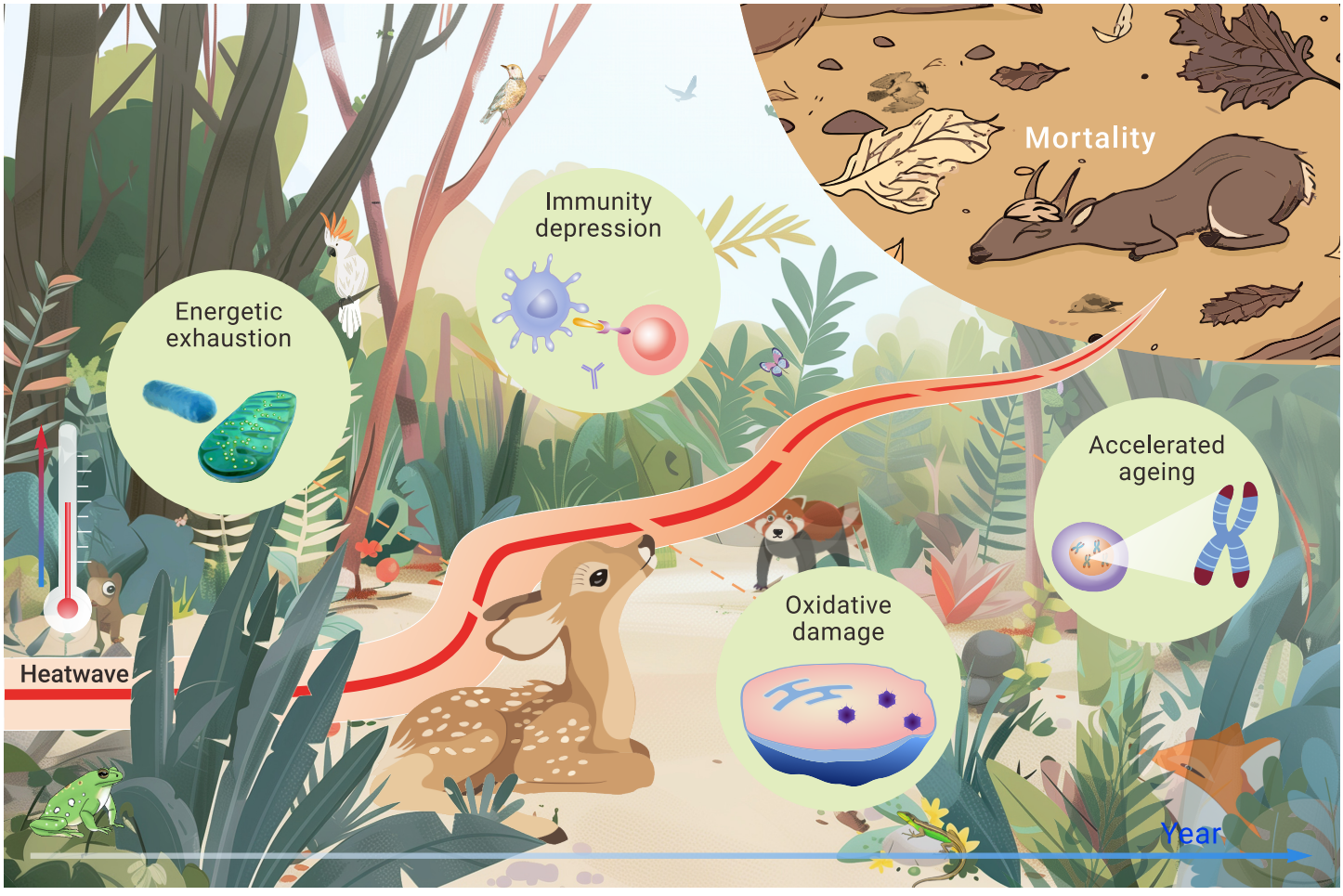

Figure 1. Potential physiological processes through which heatwaves threaten fauna biodiversity.

DEPRESSING SURVIVAL AND FUNCTIONS

Heatwaves can be lethal as predicted by thermal tolerance ranges. Animals exhibit specific temperature tolerance ranges within which various physiological processes are influenced by temperature. If the temperatures imposed by heatwaves exceed the threshold of heat tolerance, these conditions can be lethal, potentially killing animals within a short time frame, ranging from minutes to hours. To examine the lethal effects of heatwaves on animals, the critical thermal maximum (CT_{max}) and thermal safety margin (the difference between CT_{max} and the highest environmental temperature) are widely employed.

Heatwaves are also detrimental by depressing animal fitness-related functions, as predicted by the thermal performance curve (TPC).[2] Ectotherms are particularly sensitive to environmental temperature fluctuations since their body temperature and all fitness-related functions are affected by external thermal environments. In this context, ectothermic physiological and behavioral functions increase with body temperature up to the optimal temperature

(T_{opt}) and then sharply decline after T_{opt}. In contrast, endotherms typically maintain a constant core body temperature through metabolic control, endocrine regulation, and other physiological processes. However, endotherms have narrow temperature thresholds, and under the impact of heatwaves, their physiological functions can be disrupted. This disruption can affect muscle and nervous systems, consequently reducing their fitness and even survival. Accordingly, the TPC for fitness-related traits, including locomotion, food assimilation, and growth, are widely employed to determine the effects of heatwaves on fitness.[2]

ENERGETIC EXHAUSTION

Heatwaves can disrupt the metabolism of animals, hindering their ability to allocate resources and energy for growth, reproduction, and survival under harsh conditions, ultimately leading to reduced fitness and other long-term detrimental effects. Metabolic rate provides insights into the overall energy demands and utilization of animals, offering a valuable means to assess metabolic processes. Metabolic rates are thermally dependent and increase significantly as the thermal environment approaches extreme high temperatures (e.g., during heatwaves), as predicted by the allometric growth curve. Consequently, the energetic expenditure for fundamental functions, including maintaining basic life activities, predation, digestion, immunity, and nervous response, increases steeply under heatwaves.

Therefore, when faced with the stress of heatwaves, animals must carefully respond to temperatures by adjusting their metabolic rate. Correspondingly, metabolic traits at multiple biological hierarchies, such as mitochondrial structures (e.g., membrane components) and functions (e.g., mitochondrial respiration), and metabolic enzyme activities [e.g., hexokinase (HK), citrate synthase (CS), succinate dehydrogenase (SDH), cytochrome C oxidase (COX), and ATPase], are robust indicators for assessing energetic status under heatwaves.[3]

OXIDATIVE DAMAGE

Sustained exposure to heatwaves can accumulate oxidative damage by perturbing the delicate balance between the generation of reactive oxygen species (ROS) and the antioxidant defense system. The accumulation of the oxidative damage in lipids (e.g., MDA), proteins (e.g., carbonylated protein), and DNA molecules (e.g., 8-Hydroxyguanine) is widely documented to induce the structural and functional damage of cell membranes, enzymes, and genetic information and transmission, respectively. Consequently, oxidative damage leads to depressed immunity, physiological dysfunctions, accelerated ageing, and even mortality. Key enzymes involved in antioxidant defense and oxidative repair, such as superoxide dismutase (SOD) and catalase (CAT), are able to eliminate ROS, and repair the damage to biomolecules, thereby sustaining physiological homeostasis at subcellular level. Accordingly, the indices for oxidative damage, antioxidant defense and oxidative repair mentioned above can serve as indicative biomarkers for evaluating the extent of oxidative damage to cellular components under heatwaves.

IMMUNITY DEPRESSION

The immune response in animals is a highly energetically demanding process, sensitive to perturbations induced by heatwaves.[4] Disruption of metabolic equilibrium under heat stress leads to a reduced allocation of energy toward immune functions, resulting in compromised immune system functionality. Additionally, the excessive accumulation of ROS can disrupt the functioning of immune cells and tissues, impairing their ability to mount a prompt and effective response to external pathogens. The combined effects of energy depletion and ROS accumulation contribute to the suppression of the overall immune capacity in animals, facilitating the evasion of viral pathogens through the host's innate and acquired immune defenses. Consequently, the heightened risk of viral infections in animals destabilizes their survival prospects under heatwaves.

Regarding innate immunity, physical barriers such as the skin and mucous membranes are susceptible to damage under thermal stress (e.g., heatwaves), diminishing their barrier function. The response intensity of phagocytic cells and humoral factors to external pathogens can be assessed through indicators including white blood cell counts, PHA skin tests, acid phosphatase (ACP) and tumor necrosis factor-α (TNF-α). In the context of acquired immunity, parameters such as lymphocyte subpopulations, alkaline phosphatase (AKP), immunoglobulins (Ig), and interleukin-6 (IL-6) serve as valuable biomarkers. Monitoring these immunological parameters provides valuable insights into the degree of immunity depression in animals exposed to heatwaves.

ACCELERATED AGEING

Heatwaves may decrease fitness, increase mortality rates, and shorten the potential lifespans of animals by accelerating ageing. Increasing research has found several detrimental effects of heatwaves on ageing in animals, such as shortened telomere length, cell cycle arrest, mitochondrial dysfunction, and chronic inflammation.[5] Theoretically, ageing is a complex network involving multiple biological hierarchies at the cellular, tissue, organ, and whole-body levels in animals. The immunity depression, oxidative damage, and energetic depletion mentioned above can accelerate ageing, and ageing is demonstrated to aggravate immune deficiency, oxidative damage, and energetic expenditure through a vicious cycle.

Drawing upon recent conceptual frameworks on ageing hallmarks and biomarkers under climate change, several phenotypes and related biomarkers can be employed to detect the detrimental effects of heatwaves on animals: telomere length, epigenetic alterations (e.g., DNA methylation, abnormal histone modifications, loss of heterochromatin, and deregulated RNA modifications), cell cycle arrest (e.g., P53/P21^{CIP1} and P16^{INK4a}/RB), mitochondrial dysfunction (e.g., mitochondrial disintegration), and the senescence-associated secretory phenotype (SASP) [e.g., interleukin-6 (IL-6), IL1-β, TNF-α, and interleukin-8 (IL-8)].

Currently, heatwaves are progressively becoming the new norm during summer, threatening the fitness and even survival of animals, consequently influencing the overall equilibrium of entire ecosystem. Under the stress of heatwaves, animals may experience functional disability, metabolic exhaustion, oxidative damage, immunity depression, and accelerated ageing. Against the backdrop of frequent extreme weather occurrences, the imperative to conserve the biodiversity has never been more pressing. Therefore, gaining a comprehensive understanding of the physiological and biochemical mechanisms by which heatwaves impact animals is essential for the preservation of biodiversity and the sustainability of ecosystems.

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DECLARATION OF INTERESTS

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