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The effect of stress hormones on reproductive function in this era of prevailing climatic change

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Abstract

In an era defined by escalating climate change, the interplay between environmental stressors and reproductive health emerges as a critical challenge, with stress hormones like cortisol and catecholamines acting as pivotal mediators. This review elucidates how climate-induced stressors, rising temperatures, extreme weather, food scarcity, and habitat loss disrupt the hypothalamic-pituitary-gonadal axis, impairing gamete quality, suppressing fertility, and altering reproductive behaviors across humans and wildlife. Through intricate physiological pathways, including HPA-HPG axis crosstalk and epigenetic modifications, chronic stress hormone elevation undermines reproductive success, threatening human fertility, food security, and biodiversity. From heat-stressed dairy cows to vulnerable human populations in tropical regions, the cascading effects of climate change amplify reproductive dysfunction, with profound implications for population dynamics and ecosystem stability. By synthesizing cutting-edge research, this paper highlights the urgency of integrating climate adaptation, public health interventions, and conservation strategies to mitigate these impacts. It calls for interdisciplinary efforts to address research gaps, particularly in transgenerational effects, to safeguard reproductive health and ensure sustainable futures in a warming world.

Keywords: Climate Change; Stress Hormones; Reproductive Function; Cortisol; Hypothalamic-Pituitary-Gonadal Axis; Fertility; Biodiversity; Epigenetic Effects

1. Introduction

The escalating impacts of climate change, including rising temperatures, extreme weather events, and resource scarcity, pose significant challenges to physiological homeostasis in both humans and animals. These environmental stressors activate stress hormone pathways, notably the hypothalamic-pituitary-adrenal (HPA) axis, which regulates cortisol and catecholamine release, influencing multiple physiological systems, including reproduction. Concurrently, reproductive function, governed by the hypothalamic-pituitary-gonadal (HPG) axis, is critical for species survival and population stability but is highly sensitive to stress-induced disruptions. This review synthesizes current knowledge on how climate change-induced stress hormones impair reproductive health, exploring mechanisms, implications, and mitigation strategies in the context of global environmental change.

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1.1. Stress Hormones and Their Role in Physiological Responses

Stress hormones, primarily cortisol and catecholamines (e.g., adrenaline, noradrenaline), are central to the body's response to environmental and physiological challenges. The HPA axis orchestrates the release of cortisol, a glucocorticoid that modulates metabolism, immune function, and stress adaptation. According to Sapolsky et al. [1], chronic activation of the HPA axis leads to elevated cortisol levels, which can disrupt homeostatic balance and contribute to pathological outcomes. In a classic study by Charmandari et al. [2], glucocorticoid receptor signaling was shown to mediate stress responses at the cellular level, influencing gene expression across multiple tissues. This systemic reach underscores the potential for stress hormones to affect reproductive physiology, particularly under chronic stress conditions induced by environmental changes.

Catecholamines, released via the sympathetic-adrenal-medullary axis, facilitate acute stress responses, such as the "fight-or-flight" reaction. Findings from McEwen et al. [3] indicate that prolonged catecholamine elevation, often triggered by repeated stressors, can lead to cardiovascular and metabolic dysregulation, indirectly impacting reproductive organs. For instance, chronic stress in mammals has been linked to altered gonadal function through sympathetic nervous system overactivation, as noted in a study by Tilbrook et al. [4]. These findings suggest that both acute and chronic stress hormone responses create a physiological environment that may compromise reproductive success.

The interplay between stress hormones and environmental stressors is particularly relevant in the context of climate change. Rising global temperatures and extreme weather events, as documented by Masson-Delmotte et al. [5], act as potent stressors, triggering sustained HPA axis activation. Such chronic stress can lead to allostatic load, a concept described by McEwen [6], where cumulative stress effects impair physiological resilience. This framework is critical for understanding how climate-induced stressors may exacerbate reproductive dysfunction through prolonged hormonal imbalances.

In humans and animals, stress hormone dysregulation has been associated with altered reproductive behaviors and outcomes. For example, a study by Wingfield et al. [7] on avian species demonstrated that elevated corticosterone levels under environmental stress suppress reproductive behaviors, such as mating and parental care. This evidence highlights the need to explore how stress hormones, amplified by climate change, disrupt reproductive physiology across species, setting the stage for deeper investigation in subsequent sections.

1.2. Importance of Reproductive Function for Species Survival

Reproductive function is fundamental to species survival, ensuring genetic continuity and population stability. The HPG axis regulates key reproductive hormones, including gonadotropin-releasing hormone (GnRH), luteinizing hormone (LH), follicle-stimulating hormone (FSH), and sex steroids (e.g., estrogen, testosterone). According to Plant et al. [8], the HPG axis orchestrates gametogenesis, ovulation, and sexual behavior, with disruptions leading to infertility or reduced reproductive success. In humans, fertility challenges linked to stress have been documented, with a study by Louis et al. [9] showing that psychosocial stress correlates with delayed ovulation and reduced conception rates.

In wildlife, reproductive success is equally critical for maintaining biodiversity, particularly in ecosystems stressed by climate change. A seminal study by Bronson [10] highlighted that environmental stressors, such as food scarcity, disrupt HPG axis function in mammals, leading to suppressed reproduction. For instance, in small mammals, chronic stress reduces litter size and offspring survival, as reported by Sheriff et al. [11]. These findings underscore the vulnerability of reproductive systems to external pressures, with implications for population dynamics in changing environments.

Climate change exacerbates these challenges by altering habitats and resource availability, further stressing reproductive systems. According to Parmesan et al. [12], shifts in temperature and precipitation patterns disrupt breeding seasons in birds and amphibians, often mediated by stress hormone elevations. For example, heat stress in fish has been shown to impair gonadal development, as evidenced by Pankhurst et al. [13], who found reduced oocyte quality in heat-stressed salmon. These disruptions threaten biodiversity, as species with low reproductive output struggle to maintain viable populations.

The cascading effects of reproductive dysfunction extend beyond individual species to ecosystems and human societies. In agricultural systems, stress-induced reproductive failure in livestock, as noted by Dobson et al. [14], impacts food security, a concern amplified by climate change. Similarly, human populations in climate-vulnerable regions face reproductive health challenges, with studies like those by Rylander et al. [15] linking heat stress to adverse pregnancy outcomes. These interconnected impacts highlight the urgency of understanding stress-reproduction interactions in the context of climate change.

1.3. Climate Change as a Global Stressor

Climate change introduces a suite of stressors that challenge physiological and ecological systems worldwide. Rising global temperatures, documented by the IPCC [5], increase heat stress, which activates the HPA axis and elevates cortisol levels. A study by Hansen [16] demonstrated that heatwaves, increasingly frequent due to climate change, induce physiological stress in humans and animals, with measurable impacts on endocrine function. In tropical regions, where temperature extremes are pronounced, these effects are particularly severe, as noted in research by Mora et al. [17], who projected increased human health risks under warming scenarios.

Beyond heat, climate change drives indirect stressors, such as food and water scarcity, habitat loss, and extreme weather events. According to Godfray et al. [18], food insecurity resulting from climate-driven agricultural disruptions heightens stress in human populations, with potential endocrine consequences. In wildlife, habitat degradation disrupts breeding cycles, as evidenced by Walther et al. [19], who reported altered reproductive phenology in migratory birds due to changing climatic conditions. These stressors collectively amplify HPA axis activation, creating a feedback loop that exacerbates physiological strain.

The synergistic effects of multiple climate-related stressors compound their impact on reproductive health. A study by Brook et al. [20] found that combined stressors, such as temperature extremes and resource scarcity, lead to greater reproductive suppression in coral reef fish than single stressors alone. Similarly, in humans, chronic stress from climate-induced displacement has been linked to reproductive health challenges, as reported by McMichael et al. [21]. These findings emphasize the complexity of climate change as a multifaceted stressor with far-reaching endocrine and reproductive consequences.

Addressing these challenges requires an integrated understanding of how climate change modulates stress responses and reproductive outcomes. The literature suggests that vulnerable populations—both human and non-human—are disproportionately affected, as highlighted by studies on climate impacts in low-resource settings [22]. By examining these interactions, this review aims to elucidate the mechanisms linking climate-induced stress hormones to reproductive dysfunction, paving the way for targeted interventions.

2. Stress Hormones and Their Physiological Mechanisms

Stress hormones, including cortisol and catecholamines, are critical mediators of the body's response to environmental and physiological challenges, with profound implications for reproductive function in the context of climate change. The hypothalamic-pituitary-adrenal (HPA) axis regulates cortisol release, while the sympathetic-adrenal-medullary (SAM) axis controls catecholamine secretion, orchestrating adaptive responses to stressors such as heat, food scarcity, and habitat disruption. Understanding the physiological mechanisms of these hormones is essential to elucidate how climate-induced stressors disrupt reproductive health across species. This section explores the molecular and systemic pathways of stress hormones, their chronic effects, and their relevance to environmental stressors.

2.1. Hypothalamic-Pituitary-Adrenal (HPA) Axis and Cortisol Dynamics

The HPA axis is the cornerstone of the stress response, coordinating the release of glucocorticoids, primarily cortisol in humans and corticosterone in many animals. According to Chrousos [23], activation of the HPA axis begins with the release of corticotropin-releasing hormone (CRH) from the hypothalamus, which stimulates adrenocorticotropic hormone (ACTH) secretion from the pituitary, ultimately triggering cortisol production in the adrenal glands. Cortisol modulates metabolism, immune function, and stress adaptation, but chronic elevation disrupts physiological homeostasis. A seminal study by Herman et al. [24] demonstrated that glucocorticoid receptors in the brain and peripheral tissues mediate these effects, influencing gene expression and cellular function.

Chronic HPA axis activation, as occurs under prolonged environmental stress, leads to sustained cortisol elevation, contributing to allostatic load. McEwen [25] defined allostatic load as the cumulative wear-and-tear on physiological systems due to repeated or chronic stress, with cortisol playing a central role. For instance, studies on humans exposed to chronic psychosocial stress, such as those by Cohen et al. [26], show that elevated cortisol levels impair neuroendocrine regulation, potentially affecting reproductive hormone signaling. This is particularly relevant in climate change contexts, where stressors like heatwaves and resource scarcity persistently activate the HPA axis.

In wildlife, similar patterns are observed. A study by Romero et al. [27] on marine iguanas found that prolonged environmental stress, such as El Niño-induced food shortages, increased corticosterone levels, leading to suppressed immune and reproductive functions. These findings highlight the conserved nature of HPA axis responses across species. Additionally, research by Tsigos and Chrousos [28] indicates that cortisol's feedback inhibition on the HPA axis can become dysregulated under chronic stress, exacerbating physiological imbalances that may impair reproductive processes.

The molecular mechanisms of cortisol action involve glucocorticoid receptor binding, which regulates transcription of genes involved in metabolism and inflammation. According to Oakley and Cidlowski [29], these receptors are expressed in reproductive tissues, suggesting direct pathways through which cortisol may disrupt gonadal function. In the context of climate change, where chronic stressors are increasingly prevalent, understanding these mechanisms is critical for predicting reproductive outcomes in stressed populations.

2.2. Catecholamines and the Sympathetic-Adrenal-Medullary (SAM) Axis

The SAM axis governs the rapid release of catecholamines—adrenaline and noradrenaline—in response to acute stressors, facilitating immediate physiological adjustments. Goldstein [30] explains that catecholamines increase heart rate, blood pressure, and energy mobilization, preparing organisms for "fight-or-flight" responses. However, as noted in a study by Kvetnansky et al. [31], repeated or prolonged activation of the SAM axis, as seen in chronic stress scenarios, leads to systemic dysregulation, including cardiovascular strain and altered neuroendocrine signaling. This chronic activation is relevant to climate-induced stressors, such as extreme weather events, which demand repeated acute responses.

Catecholamines indirectly influence reproductive function by modulating blood flow and energy allocation. A study by Spicer [32] found that noradrenaline, acting through adrenergic receptors in ovarian tissue, alters follicular development in mammals, potentially reducing ovulation rates. In males, chronic catecholamine elevation has been linked to reduced testosterone production, as reported by Hardy et al. [33], who observed suppressed Leydig cell function in stressed rodents. These findings suggest that SAM axis activation under climate-related stressors could impair reproductive physiology.

In ecological contexts, catecholamine responses are critical for survival but can compromise long-term reproductive success. For example, research by Boonstra et al. [34] on Arctic ground squirrels showed that frequent predator-induced stress increased catecholamine levels, reducing energy allocation to reproduction and lowering litter success. Climate change exacerbates such stressors by altering predator-prey dynamics and habitat stability, amplifying SAM axis activation.

The interplay between catecholamines and cortisol further complicates stress responses. According to Young et al. [35], simultaneous activation of the SAM and HPA axes under chronic stress creates a synergistic effect, amplifying physiological strain. This interaction is particularly relevant in climate-vulnerable environments, where multiple stressors (e.g., heat, food scarcity) converge, potentially magnifying reproductive disruptions through catecholamine-mediated pathways.

To provide a comprehensive overview of how stress hormones mediate physiological responses relevant to climate-induced stressors, Table 1 summarizes the key stress hormones, their primary physiological effects, and their implications for reproductive function, drawing from studies cited in this review.

Table 1 Stress Hormones and Their Physiological Effects

Stress Hormone	Axis	Primary Physiological Effects	Impact on Reproduction	Relevant Climate Stressor	Reference
Cortisol	HPA	Modulates metabolism, immune function, and stress adaptation; chronic elevation leads to allostatic load	Suppresses GnRH, LH, and FSH; impairs gamete quality and fertility	Heat stress, food scarcity	Chrousos [23], McEwen [25], Cohen et al. [26]
Corticosterone	HPA	Regulates stress responses in non-human species; suppresses immune and reproductive functions	Reduces breeding behaviors, delays reproduction	Temperature extremes, habitat loss	Romero et al. [27], Wingfield et al. [7]
Adrenaline	SAM	Increases heart rate, blood pressure, and energy	Diverts energy from reproduction, alters	Extreme weather events	Goldstein [30], Lane et al. [65]

		mobilization for acute stress	follicular development		
Noradrenaline	SAM	Facilitates "fight-or-flight" responses; chronic elevation causes cardiovascular strain	Reduces ovulation rates, impairs testosterone production	Predation stress, habitat disruption	Spicer [32], Hardy et al. [33]

2.3. Relevance to Climate Change-Induced Stress

Climate change introduces a range of stressors that activate both the HPA and SAM axes, with cascading effects on reproductive health. Rising temperatures, a hallmark of climate change, directly stimulate stress hormone release. A study by Cramer et al. [36] demonstrated that heat stress in humans increases cortisol and catecholamine levels, disrupting thermoregulation and energy balance. In livestock, heat stress has been shown to elevate cortisol, reducing reproductive efficiency, as reported by Wolfenson et al. [37]. These findings underscore the direct link between climate-induced thermal stress and stress hormone dysregulation.

Indirect stressors, such as food and water scarcity, further exacerbate stress hormone responses. Research by Godfray et al. [38] highlights how climate-driven agricultural disruptions lead to nutritional stress, triggering HPA axis activation in human and animal populations. In wildlife, habitat loss due to climate change increases stress hormone levels, as evidenced by a study on amphibians by Hayes et al. [39], which linked habitat degradation to elevated corticosterone and reduced reproductive output. These indirect effects amplify the physiological burden of climate change.

The synergistic impact of multiple climate-related stressors intensifies stress hormone responses. According to Brook et al. [40], combined stressors, such as temperature extremes and habitat alteration, produce greater physiological impacts than single stressors, with stress hormones serving as key mediators. For example, a study by Pankhurst [41] on fish found that combined heat and hypoxia stress increased cortisol levels, impairing gonadal development more severely than either stressor alone. These findings highlight the need to consider multi-stressor interactions in climate change research.

Chronic stress hormone elevation under climate change conditions creates a feedback loop that sustains physiological disruption. As noted by Wingfield [42], prolonged stress in birds leads to a “stress phenotype” characterized by high glucocorticoid levels and suppressed reproductive behaviors. This phenotype is increasingly prevalent in climate-stressed ecosystems, emphasizing the urgency of understanding how stress hormones mediate reproductive impacts in the current era of environmental change.

3. Reproductive Function and Its Regulation

Reproductive function is a cornerstone of species survival, governed by the hypothalamic-pituitary-gonadal (HPG) axis, which orchestrates the production of gametes and sex hormones critical for fertility and population maintenance. This axis integrates environmental and physiological cues, making it highly susceptible to disruptions from stress hormones, particularly in the context of climate change. Understanding the regulation of reproductive processes in humans and animals, and their vulnerability to stressors, is essential for assessing the broader implications of climate-induced stress on reproductive health. This section explores the mechanisms of the HPG axis, the roles of key reproductive hormones, and the evidence for stress-related disruptions in reproductive function.

3.1. The Hypothalamic-Pituitary-Gonadal (HPG) Axis and Hormonal Regulation

The HPG axis is the primary regulator of reproductive function, coordinating the release of hormones that drive gametogenesis, ovulation, and sexual behavior. According to Herbison [43], the hypothalamus secretes gonadotropin-releasing hormone (GnRH), which stimulates the anterior pituitary to release luteinizing hormone (LH) and follicle-stimulating hormone (FSH). These gonadotropins, in turn, regulate gonadal production of sex steroids, such as estrogen and testosterone, which are essential for reproductive processes. A seminal study by Tsutsumi et al. [44] demonstrated that pulsatile GnRH secretion is critical for maintaining HPG axis function, with disruptions leading to impaired fertility in mammals.

In females, LH and FSH orchestrate follicular development, ovulation, and corpus luteum formation, as detailed by Richards et al. [45]. Estrogen and progesterone, produced by the ovaries, regulate the menstrual cycle and support pregnancy. In males, LH stimulates Leydig cells to produce testosterone, while FSH supports spermatogenesis in Sertoli cells, as described by Walker and Cheng [46]. These tightly regulated processes are sensitive to external stressors, including those amplified by climate change, which can disrupt GnRH pulsatility and downstream hormone production.

Stress hormones, particularly cortisol, can suppress HPG axis activity by inhibiting GnRH release. A study by Whirledge and Cidlowski [47] found that glucocorticoid receptors in hypothalamic neurons mediate this suppression, reducing LH and FSH secretion. This mechanism is particularly relevant in climate-stressed environments, where chronic stressors like heat and food scarcity elevate cortisol levels, potentially impairing reproductive hormone signaling.

The HPG axis also integrates environmental cues, such as temperature and photoperiod, which are increasingly disrupted by climate change. Research by Kriegsfeld et al. [48] on rodents showed that altered environmental signals disrupt GnRH neuron activity, leading to mistimed reproductive cycles. These findings highlight the vulnerability of the HPG axis to climate-related stressors, underscoring the need to explore how stress hormones mediate these effects in diverse species.

3.2. Species-Specific Reproductive Strategies and Environmental Sensitivity

Reproductive strategies vary across species, shaped by evolutionary adaptations to environmental conditions, yet all are susceptible to stress-induced disruptions. In mammals, seasonal breeders rely on environmental cues like photoperiod to time reproduction, as noted by Bronson [49]. For example, a study by Paul et al. [50] on hamsters demonstrated that disruptions in day length, a phenomenon linked to climate change, alter melatonin signaling, suppressing GnRH and reducing reproductive success. Such sensitivity to environmental changes makes seasonal breeders particularly vulnerable to climate-driven stressors.

In non-mammalian species, such as birds and fish, reproductive strategies are equally dependent on environmental stability. According to Wingfield et al. [51], birds use temperature and food availability as cues for breeding, with stress hormones like corticosterone disrupting nest-building and egg-laying under adverse conditions. Similarly, a study by Pankhurst and Munday [52] found that elevated sea temperatures, a consequence of climate change, impair gonadal development in coral reef fish, reducing fecundity. These species-specific responses highlight the broad impact of environmental stressors on reproductive function.

Amphibians, often considered sentinels of environmental change, exhibit acute sensitivity to climate-induced stressors. Research by Blaustein et al. [53] showed that altered precipitation patterns and temperature fluctuations disrupt amphibian breeding cycles, with stress hormones mediating reduced clutch sizes. These findings are critical in the context of climate change, as amphibians face habitat loss and thermal stress, amplifying HPG axis disruptions.

The interplay between species-specific reproductive strategies and stress hormone effects underscores the need for comparative studies. As noted by Zera and Harshman [54], understanding how stress hormones differentially impact reproductive physiology across taxa can inform conservation strategies for species at risk from climate change. This comparative approach is vital for predicting population-level impacts in diverse ecosystems.

3.3. Evidence of Stress-Induced Reproductive Disruption

Stress-induced disruptions to reproductive function are well-documented across species, with stress hormones playing a central role. In humans, psychosocial stress has been shown to impair fertility by altering HPG axis dynamics. A study by Lynch et al. [55] found that elevated cortisol levels in women undergoing fertility treatment were associated with lower ovulation rates and reduced pregnancy success. Similarly, research by Nepomnaschy et al. [56] demonstrated that cortisol spikes in early pregnancy increase the risk of miscarriage, highlighting the sensitivity of reproductive processes to stress.

In livestock, stress from environmental factors like heat and crowding reduces reproductive efficiency. According to Nardone et al. [57], heat stress in dairy cows elevates cortisol, suppressing LH surges and impairing ovulation, which leads to lower conception rates. These findings are particularly relevant in climate change scenarios, where rising temperatures exacerbate heat stress in agricultural systems, threatening food security.

Wildlife studies further illustrate the impact of stress on reproduction. A classic study by Moberg [58] on mammals showed that chronic stress, mediated by glucocorticoids, reduces testosterone levels in males and disrupts estrous cycles in females, leading to population declines. In marine species, such as sea turtles, thermal stress from warming oceans has been linked to skewed sex ratios and reduced hatchling success, as reported by Jensen et al. [59]. These disruptions, driven by stress hormone elevations, threaten biodiversity in climate-impacted ecosystems.

The cumulative evidence suggests that stress hormones act as a critical link between environmental stressors and reproductive dysfunction. As climate change intensifies stressors like heat, drought, and habitat loss, the HPG axis becomes increasingly vulnerable, as highlighted by Acevedo-Rodriguez et al. [60]. These findings emphasize the need to integrate stress physiology and reproductive biology to address the challenges posed by a changing climate.

4. Climate Change as a Stressor

Climate change introduces a complex array of environmental stressors that profoundly impact physiological systems, including the stress hormone pathways that influence reproductive function. Rising temperatures, extreme weather events, and resource scarcity—hallmarks of the current era of climate change—activate the hypothalamic-pituitary-adrenal (HPA) and sympathetic-adrenal-medullary (SAM) axes, elevating cortisol and catecholamine levels. These stressors disrupt homeostasis and exacerbate reproductive challenges across species, threatening biodiversity and human health. This section examines the direct and indirect effects of climate change as stressors, their physiological consequences, and their synergistic impacts on stress hormone responses.

4.1. Direct Stressors: Heat Stress and Extreme Weather Events

Heat stress, driven by rising global temperatures, is a primary direct stressor associated with climate change, triggering robust stress hormone responses. According to Anderson et al. [61], prolonged exposure to high temperatures activates the HPA axis, increasing cortisol levels in humans and animals, which disrupts thermoregulation and metabolic balance. In a study by Hansen et al. [62], heatwaves were shown to elevate cortisol in urban populations, leading to physiological strain that can impair reproductive hormone signaling. This is particularly concerning in tropical and subtropical regions, where temperature extremes are becoming more frequent and severe.

In livestock, heat stress significantly impacts reproductive efficiency. Research by Roth et al. [63] demonstrated that heat-stressed dairy cows exhibit elevated cortisol levels, which suppress luteinizing hormone (LH) surges, reducing ovulation rates and fertility. Similarly, in wildlife, heat stress disrupts breeding behaviors. A study by du Plessis et al. [64] on desert birds found that extreme heat increased corticosterone levels, leading to delayed breeding and reduced clutch sizes. These findings highlight the direct link between thermal stress and reproductive dysfunction mediated by stress hormones.

Extreme weather events, such as hurricanes and floods, further amplify stress responses. According to Lane et al. [65], acute stressors from extreme weather events trigger catecholamine release via the SAM axis, causing immediate physiological adjustments that can divert energy from reproduction. In marine species, such as corals, temperature-induced stress events like bleaching elevate cortisol-like compounds, impairing gametogenesis, as reported by Tarrant et al. [66]. These direct stressors underscore the pervasive impact of climate change on stress physiology.

The chronic nature of these direct stressors, driven by ongoing climate change, creates a sustained physiological burden. As noted by Diffenbaugh and Field [67], the increasing frequency and intensity of heatwaves and extreme weather events amplify HPA axis activation, leading to prolonged cortisol elevation. This chronic stress response is likely to exacerbate reproductive impairments, necessitating further research into adaptive mechanisms in affected populations.

4.2. Indirect Stressors: Food and Water Scarcity and Habitat Loss

Indirect stressors, such as food and water scarcity and habitat loss, are significant consequences of climate change that activate stress hormone pathways. Food insecurity, driven by climate-induced agricultural disruptions, elevates cortisol levels in human populations. A study by Myers et al. [68] found that reduced crop yields due to drought and heat stress contribute to nutritional stress, triggering HPA axis activation and potentially impairing reproductive health. In vulnerable communities, this stress is compounded by socioeconomic factors, amplifying physiological impacts.

In wildlife, food scarcity disrupts reproductive cycles through stress hormone-mediated pathways. Research by Kitaysky et al. [69] on seabirds showed that food shortages, linked to ocean warming, increased corticosterone levels, reducing breeding success and chick survival. Similarly, water scarcity, a growing issue in arid regions, induces stress responses that affect reproduction. A study by McKechnie and Wolf [70] on desert-dwelling mammals found that dehydration stress elevated cortisol, suppressing reproductive behaviors and fecundity.

Habitat loss, driven by deforestation, urbanization, and climate-induced ecosystem shifts, further exacerbates stress responses. According to Fahrig [71], habitat fragmentation increases stress hormone levels in wildlife by limiting access to breeding sites and resources. For example, a study by Janin et al. [72] on amphibians demonstrated that habitat degradation due to climate change increased corticosterone levels, leading to reduced reproductive output. These indirect stressors create a cascade of physiological effects that disrupt HPG axis function.

The interplay between food scarcity, water shortages, and habitat loss creates a complex stress landscape. As highlighted by Tilman et al. [73], the combined impact of these indirect stressors amplifies physiological stress, with stress hormones acting as key mediators. In the context of climate change, these indirect effects are critical drivers of reproductive challenges, particularly in species and populations already under pressure.

4.3. Synergistic Effects of Multiple Climate-Related Stressors

Climate change often involves multiple stressors acting simultaneously, producing synergistic effects that intensify stress hormone responses and reproductive impacts. According to Crain et al. [74], synergistic interactions between stressors, such as heat and food scarcity, result in greater physiological disruption than single stressors alone. In fish, a study by Munday et al. [75] found that combined heat and ocean acidification stress increased cortisol levels, leading to more severe reductions in reproductive success than either stressor independently.

In terrestrial species, synergistic stressors also amplify reproductive challenges. Research by Conradie et al. [76] on reptiles showed that combined heat and drought stress elevated corticosterone, disrupting gonadal development and reducing clutch viability. Similarly, in human populations, the combination of heat stress and socioeconomic stressors, such as poverty, exacerbates cortisol elevation, as reported by Watts et al. [77]. These synergistic effects highlight the complexity of climate change impacts on stress physiology.

The synergistic nature of climate stressors has significant implications for population dynamics. A study by Brook et al. [78] emphasized that multiple stressors can push species toward extinction by compounding reproductive suppression. For example, in pollinators like bees, combined heat and pesticide exposure increase stress hormone levels, reducing queen fertility, as noted by Potts et al. [79]. These findings underscore the need for holistic approaches to studying climate change impacts.

The synergistic interplay of climate stressors amplifies reproductive and biodiversity threats beyond individual impacts, as seen in projections for livestock and wildlife populations. Figure 1 maps these interactions globally, demonstrating how combined heat and scarcity could exacerbate fertility declines and ecosystem instability by century's end.

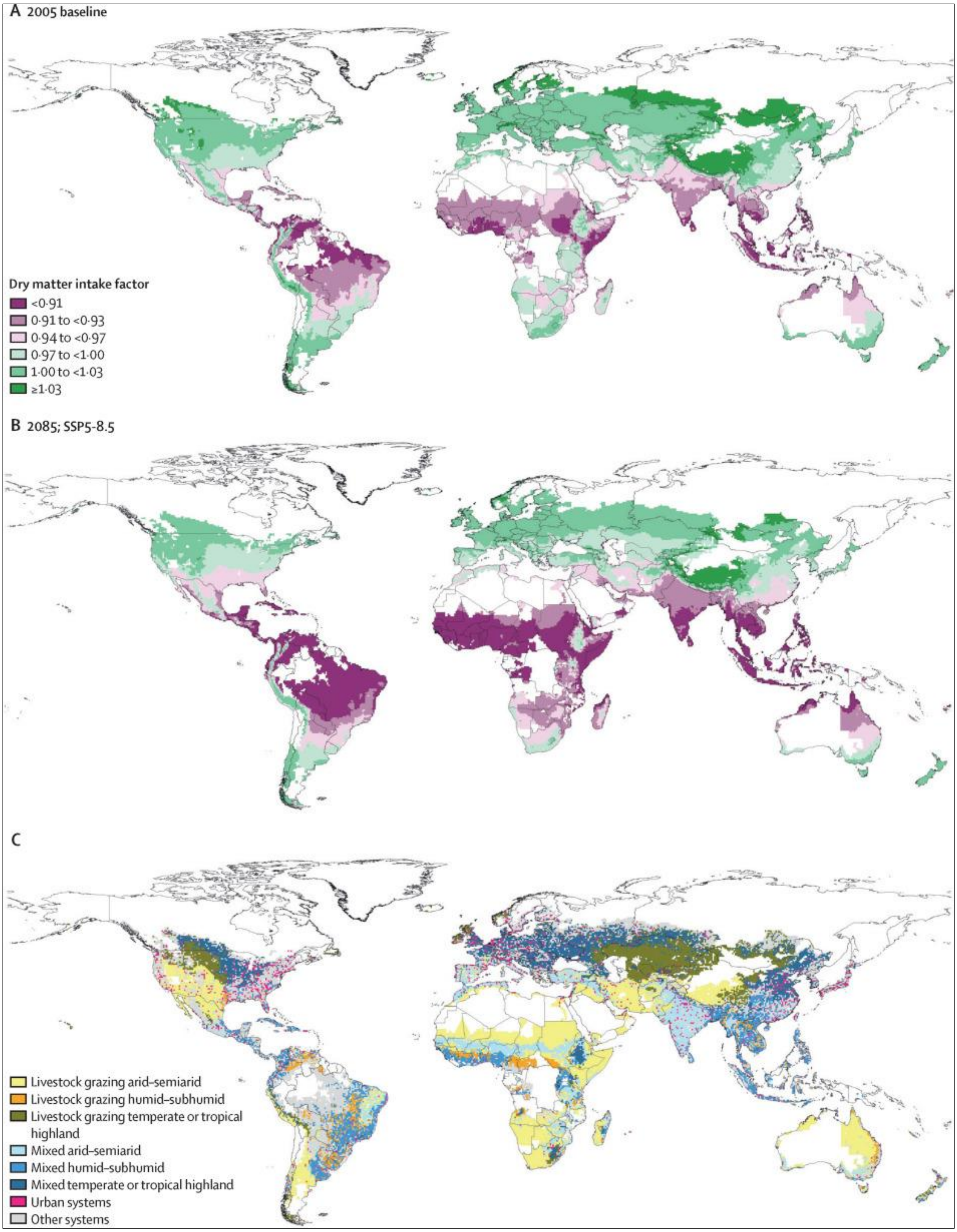

Figure 1 Synergistic Interactions Among Multiple Stressors. Thornton et al [81]

Addressing synergistic stressors requires integrated research and mitigation strategies. As noted by Fischer and Lindenmayer [80], understanding the combined effects of climate stressors on stress hormones and reproduction is critical for developing effective conservation and public health interventions. This perspective is essential for predicting and managing the long-term consequences of climate change on reproductive health across ecosystems.

To illustrate the diverse ways in which climate change acts as a stressor, Table 2 presents a detailed comparison of direct and indirect climate-induced stressors, their effects on stress hormone pathways, and associated reproductive outcomes, synthesizing evidence from the literature reviewed.

Table 2 Climate-Induced Stressors and Their Effects on Stress Hormones

Stressor Type	Specific Stressor	Stress Hormone Affected	Physiological Response	Reproductive Outcome	Species Affected	Reference
Direct	Heat Stress	Cortisol, Corticosterone	Increased HPA axis activation, thermoregulatory disruption	Suppressed LH surges, reduced gamete quality	Humans, Dairy Cows, Birds	Hansen et al. [62], Roth et al. [63], du Plessis et al. [64]
Direct	Extreme Weather Events	Adrenaline, Noradrenaline	SAM axis activation, energy diversion	Impaired gametogenesis, delayed breeding	Corals, Mammals	Lane et al. [65], Tarrant et al. [66]
Indirect	Food Scarcity	Cortisol, Corticosterone	HPA axis activation, nutritional stress	Reduced breeding success, lower litter sizes	Seabirds, Small Mammals	Myers et al. [68], Kitaysky et al. [69]
Indirect	Water Scarcity	Cortisol	Dehydration stress, HPA axis activation	Suppressed reproductive behaviors	Desert Mammals	McKechnie and Wolf [70]
Indirect	Habitat Loss	Corticosterone	Increased stress from limited breeding sites	Reduced reproductive output	Amphibians	Janin et al. [72], Fahrig [71]
Synergistic	Heat + Food Scarcity	Cortisol, Corticosterone	Amplified HPA axis activation	Severe reduction in reproductive success	Fish, Reptiles	Munday et al. [75], Conradie et al. [76]

5. Mechanisms Linking Stress Hormones to Reproductive Dysfunction

Climate change amplifies stress hormone production, particularly cortisol and catecholamines, which disrupt reproductive function through complex physiological pathways. The interplay between the hypothalamic-pituitary-adrenal (HPA) and hypothalamic-pituitary-gonadal (HPG) axes is central to these disruptions, with stress hormones suppressing reproductive hormone signaling and impairing gamete quality, fertility, and reproductive behaviors. These mechanisms are particularly pronounced in the context of climate-induced stressors, such as heat, food scarcity, and habitat loss, which exacerbate stress responses across species. This section explores the molecular and physiological pathways linking stress hormones to reproductive dysfunction, focusing on HPA-HPG axis crosstalk, gamete quality, sex-specific impacts, and epigenetic effects.

5.1. HPA-HPG Axis Crosstalk and Reproductive Suppression

The interaction between the HPA and HPG axes is a primary mechanism through which stress hormones impair reproductive function. According to Whirledge and Cidlowski [47], elevated cortisol levels inhibit gonadotropin-releasing hormone (GnRH) secretion in the hypothalamus, reducing luteinizing hormone (LH) and follicle-stimulating hormone (FSH) release from the pituitary. This suppression disrupts gonadal steroidogenesis, leading to decreased estrogen and testosterone production. A seminal study by Tilbrook et al. [4] demonstrated that chronic stress in sheep reduces GnRH pulsatility, resulting in impaired ovulation and spermatogenesis, a mechanism likely exacerbated by climate-related stressors like heat and resource scarcity.

The intricate crosstalk between the HPA and HPG axes is central to understanding stress-mediated reproductive suppression, as elevated glucocorticoids directly inhibit key reproductive hormones. Figure 1 illustrates this interaction, highlighting how environmental stressors disrupt GnRH pulsatility and downstream gonadal function, consistent with observations in mammals under chronic stress.

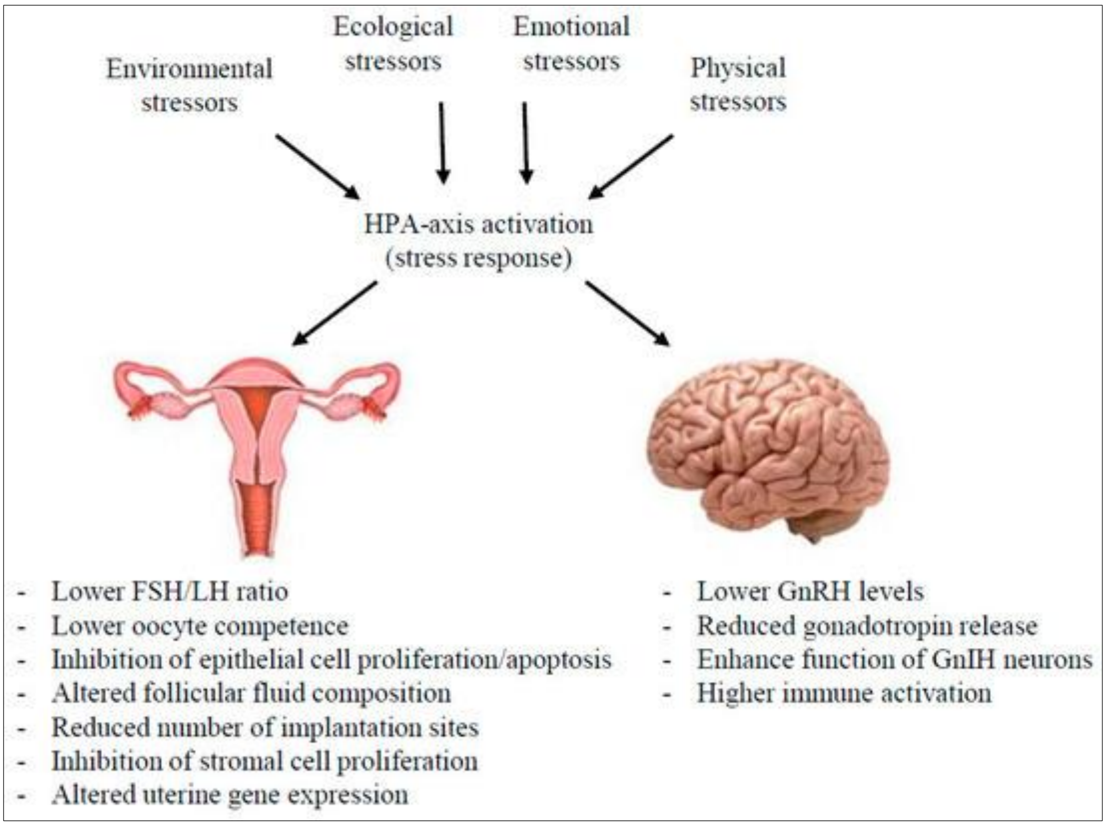

Figure 2 Diagram of HPA-HPG Axis Interactions. Joseph et al. [82]

Cortisol's inhibitory effects are mediated by glucocorticoid receptors expressed in hypothalamic and pituitary tissues. Research by Oakley and Cidlowski [29] showed that these receptors modulate gene expression, downregulating GnRH and gonadotropin synthesis under stress. In humans, this crosstalk has been linked to fertility challenges. For instance, Lynch et al. [55] found that women with elevated cortisol due to psychosocial stress exhibited disrupted LH surges, reducing ovulation rates. These findings highlight how climate-induced stress, which sustains HPA axis activation, can suppress HPG axis function.

In wildlife, similar mechanisms are observed. A study by Wingfield et al. [7] on birds showed that corticosterone elevation under environmental stress suppresses GnRH, leading to delayed breeding and reduced reproductive success. This is particularly relevant in climate change scenarios, where stressors like temperature extremes and habitat loss, as noted by Diffenbaugh and Field [67], chronically elevate glucocorticoids. The conserved nature of HPA-HPG axis crosstalk across species underscores its role as a critical pathway for reproductive dysfunction.

The chronicity of climate-related stressors amplifies these effects, creating a feedback loop that sustains reproductive suppression. As described by McEwen [25], prolonged HPA axis activation leads to allostatic load, impairing neuroendocrine regulation and exacerbating HPG axis dysfunction. This mechanism is critical for understanding how climate change, through sustained stress hormone elevation, threatens reproductive health in both human and animal populations.

5.2. Effects on Gamete Quality and Fertility Outcomes

Stress hormones directly impact gamete quality, reducing fertility across species. In females, cortisol disrupts oocyte maturation and ovulation by altering follicular development. A study by Roth et al. [63] on dairy cows found that heat stress-induced cortisol elevation impaired oocyte quality, leading to lower fertilization rates and embryo viability. Similarly, in fish, Pankhurst and Munday [52] reported that elevated cortisol due to ocean warming reduced oocyte size and quality, decreasing reproductive output in coral reef species. These findings are critical in the context of climate change, where thermal stress is increasingly prevalent.

In males, stress hormones impair spermatogenesis and testosterone production. Research by Hardy et al. [33] demonstrated that chronic stress in rodents, mediated by catecholamines and cortisol, reduced sperm motility and count by suppressing Leydig cell function. In humans, a study by Louis et al. [9] linked psychosocial stress to decreased sperm quality, with cortisol disrupting testosterone synthesis. These effects are amplified in climate-stressed environments, where multiple stressors converge to elevate stress hormone levels.

Fertility outcomes are further compromised by stress-induced disruptions in embryo implantation and pregnancy maintenance. According to Nepomnaschy et al. [56], elevated cortisol in early human pregnancy increases miscarriage risk by altering uterine receptivity and placental function. In wildlife, similar patterns are observed; for example, Sheriff et al. [11] found that stress-induced corticosterone elevation in small mammals reduced litter size and offspring survival. These impacts highlight the cascading effects of stress hormones on reproductive success.

Climate change exacerbates these effects by intensifying stressors that impair gamete quality. As noted by Pankhurst [41], combined stressors like heat and hypoxia in aquatic systems amplify cortisol-mediated reproductive disruptions, reducing population viability. Understanding these mechanisms is essential for predicting fertility outcomes in climate-vulnerable species and human populations.

5.3. Sex-Specific Impacts of Stress Hormones

Stress hormones exert sex-specific effects on reproductive function, reflecting differences in HPG axis regulation between males and females. In females, cortisol disrupts the menstrual cycle by suppressing GnRH and LH, leading to anovulation or irregular cycles. A study by Whirledge and Cidlowski [47] found that glucocorticoid receptors in ovarian tissue mediate cortisol's inhibitory effects on estrogen production, impairing follicular development. In climate-stressed populations, such as those exposed to heatwaves, these effects are pronounced, as reported by Rylander et al. [15], who linked heat stress to adverse pregnancy outcomes in women.

In males, stress hormones primarily affect testosterone production and spermatogenesis. Research by Moberg [58] showed that chronic glucocorticoid elevation in male mammals reduces testosterone levels by inhibiting Leydig cell activity, leading to decreased libido and sperm quality. In humans, Hansen [16] noted that heat stress, a common climate-related stressor, elevates cortisol and catecholamines, further suppressing male reproductive function. These sex-specific responses are critical for understanding differential vulnerabilities in climate-impacted environments.

Sex-specific effects also manifest in reproductive behaviors. In birds, Wingfield et al. [51] found that corticosterone elevation under environmental stress reduces female egg-laying and male courtship behaviors, with females showing greater sensitivity due to higher energy demands. Similarly, in amphibians, Blaustein et al. [53] reported that females are more affected by stress-induced reproductive suppression due to their higher investment in gamete production. These differences highlight the need for sex-specific analyses in climate change research.

The interaction of climate stressors and sex-specific physiology complicates reproductive outcomes. As noted by Acevedo-Rodriguez et al. [60], understanding these differences is crucial for developing targeted interventions, particularly in species where one sex is more vulnerable to climate-induced stress. This perspective is vital for addressing the disproportionate impacts of climate change on reproductive health.

5.4. Epigenetic and Transgenerational Effects

Chronic stress hormone elevation can induce epigenetic changes that affect reproductive function across generations. According to Bale [73], glucocorticoids alter DNA methylation patterns in reproductive tissues, influencing gene expression related to HPG axis function. In rodents, a study by Rodgers et al. [74] showed that maternal stress during pregnancy increased cortisol in offspring, leading to reduced fertility through epigenetic modifications in GnRH genes. These findings suggest that climate-induced stress could have long-lasting reproductive impacts.

In humans, epigenetic effects of stress are increasingly recognized. Research by Nepomnaschy et al. [56] indicated that maternal cortisol elevation due to environmental stressors alters placental gene expression, potentially affecting offspring reproductive health. In climate-vulnerable populations, where chronic stressors like food scarcity and heat are prevalent, these epigenetic changes could exacerbate fertility challenges, as noted by Myers et al. [68].

Wildlife studies also reveal transgenerational effects. A study by Sheriff et al. [11] on small mammals found that stress-induced corticosterone elevation in parents led to reduced reproductive success in offspring through epigenetic inheritance. In marine species, Jensen et al. [59] reported that thermal stress in sea turtles altered epigenetic markers, affecting sex determination and reproductive outcomes in subsequent generations. These transgenerational effects amplify the long-term consequences of climate change.

The epigenetic impact of stress hormones underscores the need for longitudinal studies in climate change research. As highlighted by Acevedo-Rodriguez et al. [60], understanding how stress-mediated epigenetic changes affect reproductive function across generations is critical for predicting population resilience in a warming world. This perspective emphasizes the far-reaching implications of climate-induced stress on reproductive health.

6. Implications and Mitigation Strategies

The interplay between climate change-induced stress hormones and reproductive dysfunction has profound implications for human health, biodiversity, and ecosystem stability. Elevated cortisol and catecholamine levels, triggered by stressors such as heat, food scarcity, and habitat loss, disrupt the hypothalamic-pituitary-gonadal (HPG) axis, leading to reduced fertility and population declines across species. These effects threaten food security, human reproductive health, and the survival of vulnerable species in a warming world. This section explores the implications of these disruptions for human and ecological systems and discusses mitigation strategies, including climate adaptation, public health interventions, and conservation efforts, to address the challenges posed by climate-induced stress.

6.1. Implications for Human Health

Climate change-induced stress hormones contribute to reproductive health challenges in humans, particularly in vulnerable populations. Elevated cortisol levels, driven by stressors like heatwaves and food insecurity, impair fertility by suppressing GnRH, LH, and FSH secretion, as noted by Whirledge and Cidlowski [47]. A study by Lynch et al. [55] found that women experiencing chronic stress, such as that induced by climate-related socioeconomic pressures, have lower ovulation rates and conception probabilities. These effects are particularly pronounced in low-resource communities, where access to reproductive healthcare is limited, as highlighted by Watts et al. [77].

Adverse pregnancy outcomes are another significant concern. Research by Rylander et al. [15] demonstrated that heat stress increases cortisol levels, contributing to higher rates of preterm birth and miscarriage in climate-vulnerable regions. In addition, Nepomnaschy et al. [56] reported that elevated cortisol in early pregnancy disrupts placental function, increasing the risk of pregnancy loss. These findings underscore the need for targeted interventions to protect reproductive health in populations exposed to climate stressors.

The broader societal implications include potential population declines in regions heavily impacted by climate change. According to Myers et al. [68], food scarcity and nutritional stress, exacerbated by climate-driven agricultural disruptions, can amplify cortisol-mediated reproductive suppression, particularly in developing nations. This could lead to demographic shifts, with long-term consequences for economic and social stability in affected areas.

Addressing these implications requires integrating reproductive health into climate change adaptation frameworks. As noted by Gemmill et al. [21], public health strategies must prioritize monitoring and supporting fertility in climate-stressed populations. This includes improving access to reproductive healthcare and addressing the socioeconomic stressors that amplify physiological stress responses.

6.2. Implications for Biodiversity and Ecosystems

Climate-induced stress hormones threaten biodiversity by reducing reproductive success in wildlife, disrupting population dynamics and ecosystem balance. In birds, elevated corticosterone levels due to temperature extremes and food shortages suppress breeding behaviors, as reported by Wingfield et al. [51]. Similarly, Pankhurst and Munday [52] found that thermal stress in fish impairs gonadal development, reducing fecundity and threatening population viability. These disruptions can lead to declines in species abundance, with cascading effects on ecosystems reliant on these species.

In amphibians, habitat loss and altered precipitation patterns increase corticosterone levels, reducing clutch sizes and larval survival, as noted by Blaustein et al. [53]. This is particularly concerning for amphibians, which are already facing global declines due to climate change, as highlighted by Hayes et al. [39]. Similarly, Jensen et al. [59] reported that warming oceans disrupt sea turtle reproduction by altering sex ratios and reducing hatchling success, threatening long-term population stability.

The loss of reproductive capacity in keystone species can destabilize ecosystems. For example, Potts et al. [79] noted that stress-induced reproductive suppression in pollinators like bees, driven by combined heat and pesticide exposure, reduces pollination services, impacting plant reproduction and food production. These findings emphasize the interconnectedness of reproductive health and ecosystem function in the context of climate change.

The synergistic effects of multiple stressors amplify these biodiversity losses. According to Brook et al. [78], combined stressors such as habitat loss and temperature extremes exacerbate reproductive suppression, pushing species toward extinction. Conservation strategies must address these synergistic impacts to protect biodiversity and maintain ecosystem services in a changing climate.

6.3. Mitigation Strategies and Research Directions

Mitigating the impact of climate-induced stress on reproductive function requires a multifaceted approach, including climate adaptation, public health interventions, and conservation efforts. Climate adaptation strategies, such as heat stress management, can reduce physiological stress. For instance, Wheeler et al. [36] suggest that urban planning to mitigate heat island effects and improve access to cooling can lower cortisol levels in human populations. In agriculture, Nardone et al. [57] advocate for heat-tolerant livestock breeds and improved cooling systems to enhance reproductive efficiency under warming conditions.

Public health interventions are critical for addressing human reproductive health challenges. As recommended by Watts et al. [77], stress reduction programs, such as mindfulness and nutritional support, can mitigate cortisol elevation in climate-vulnerable communities. Additionally, Gemmill et al. [21] emphasize the importance of reproductive health monitoring in regions affected by climate stressors, ensuring early intervention for fertility issues. These strategies can help protect human populations from the reproductive impacts of climate change.

Conservation efforts are essential for preserving biodiversity. Fischer and Lindenmayer [80] propose protecting critical breeding habitats to reduce stress hormone elevation in wildlife. For example, restoring wetlands can support amphibian reproduction, as noted by Blaustein et al. [53]. Assisted reproductive technologies, such as those used for endangered species, can also enhance reproductive success, as discussed by Acevedo-Rodriguez et al. [60]. These efforts are vital for mitigating the impact of climate stressors on vulnerable species.

Future research should focus on filling knowledge gaps, particularly regarding longitudinal and multi-species studies. As highlighted by Pankhurst [41], understanding the long-term effects of chronic stress on reproduction across taxa is essential for predicting population resilience. Additionally, investigating epigenetic mechanisms, as explored by Rodgers et al. [74], can provide insights into transgenerational impacts. Interdisciplinary research integrating endocrinology, ecology, and public health will be critical for developing effective mitigation strategies.

7. Conclusion

Climate change, through its multifaceted stressors such as rising temperatures, extreme weather events, food scarcity, and habitat loss, profoundly impacts reproductive function by elevating stress hormones like cortisol and catecholamines. These hormones disrupt the hypothalamic-pituitary-gonadal axis, impairing gamete quality, suppressing fertility, and altering reproductive behaviors across humans and wildlife. The chronic activation of the hypothalamic-pituitary-adrenal and sympathetic-adrenal-medullary axes under climate-induced stress creates a cascade of physiological effects, from molecular disruptions in hormone signaling to transgenerational epigenetic changes, threatening population stability and biodiversity. The evidence underscores the vulnerability of reproductive systems to environmental stressors, highlighting the urgent need to address these impacts in the context of a rapidly warming world.

The implications of these disruptions are far-reaching, affecting human health, food security, and ecosystem resilience. In human populations, particularly in climate-vulnerable regions, stress-induced reproductive challenges contribute to infertility, pregnancy complications, and potential demographic shifts. In wildlife, reduced reproductive success exacerbates biodiversity loss, destabilizing ecosystems and threatening species survival. These interconnected challenges emphasize the critical link between environmental change, stress physiology, and reproductive health, underscoring the need for integrated approaches to mitigate the impacts.

Mitigation strategies, including climate adaptation measures, public health interventions, and conservation efforts, offer pathways to alleviate the reproductive burden of climate change. Protecting breeding habitats, implementing heat stress management, and enhancing reproductive health monitoring can help safeguard both human and ecological systems.

However, significant research gaps remain, particularly in understanding long-term and transgenerational effects across diverse species. Interdisciplinary efforts combining endocrinology, ecology, and public health are essential to develop effective solutions. By prioritizing these strategies and fostering global collaboration, we can address the challenges posed by climate-induced stress on reproductive function, ensuring sustainable futures for both human societies and natural ecosystems.

Compliance with ethical standards

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The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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