

"The Physiological Basis of Hibernation in Mammals"

Ayesha Khan

Department of Commerce and Entrepreneurship, Aliah University

Received: 15/01/2026 ; Accepted: 27/05/2026 ; Published: 01/06/2026

Abstract

Hibernation is a complicated physiological adaptation that allows mammals, especially those living in temperate and polar regions, to survive for extended periods of time when food is few and the environment is harsh. In order to store energy reserves for the winter, animals enter a state of deep metabolic suppression known as hibernation. This state is characterized by a synchronized decrease in body temperature, heart rate, respiration rate, and total energy expenditure. Complex molecular, cellular, and systemic regulatory mechanisms underpin hibernation's physiological basis. These mechanisms include changes in metabolic fuel utilization from carbohydrates to lipids, modulation of mitochondrial function, and suppression of non-essential physiological processes. The hypothalamus, thyroid hormones, and melatonin are all important neuroendocrine regulators that help start and keep torpor states going. The underlying causes of periodic arousals, which are necessary for the maintenance of brain and immunological functions and are characterized by the transient restoration of euthermic circumstances, are only partially understood. In order to prevent tissue damage during cycles of hypothermia and rewarming, hibernation entails reversible changes in gene expression, protein synthesis, and neuroprotection. This finding has important biological implications for organ preservation, hypothermia therapy, and space travel. examines the physiological processes that cause mammals to hibernate, delves into the adaptive value of this phenomenon, and discusses new viewpoints on how it relates to human health and biomedical advancements.

Keywords:

Hibernation, torpor, metabolic suppression, thermoregulation, lipid metabolism, neuroendocrine regulation

Introduction

As one of the most incredible physiological adaptations in animal history, hibernation allows mammals to enter a state of torpor with significantly reduced metabolic rate, body temperature, and energy expenditure, enabling them to survive for extended periods of cold weather, food scarcity, and environmental stress. While certain tiny mammals and birds enter a daily state of torpor, hibernation is a longer seasonal phenomena that can last weeks or months. During this time, an animal cycles between lengthy periods of dormancy and brief awakenings that bring its metabolic activity back to normal levels. Some tropical species have been observed to employ this adaptive approach in response to drought or resource instability; nevertheless, it is more prevalent among temperate and polar species due to the devastating ecological effects of cold winters. The intricate web of molecular, cellular, systemic, and behavioral processes that allow creatures to endure potentially fatal conditions is the physiological basis of hibernation. Hibernation is characterized metabolically by a dramatic downregulation of energy-intensive processes, with the main energy source shifting from carbohydrate metabolism to lipid oxidation, enabling the efficient use of fat stores amassed during active seasons. Metabolic

flexibility, altered membrane fluidity, and antioxidant defense systems keep tissues safe from harm, even if core body temperature can dip close to ambient levels—or even freeze—in many species. This adaptation revolves around thermoregulation. The hypothalamus integrates environmental cues like photoperiod and temperature, and hormones like melatonin, thyroid hormones, and insulin play regulatory roles in starting and maintaining torpor; neuroendocrine regulation provides the central control of hibernation. Changes in gene expression and protein synthesis underpin long-term physiological remodeling, whereas neural plasticity and altered neurotransmitter activity affect sleep-wake cycles and circadian rhythms during hibernation. One of the most fascinating parts of hibernation is periodic reawakenings, when animals briefly go back to a state of thermoregulation. There is a fine line between conserving energy and ensuring physiological maintenance, and these arousals are thought to be necessary for keeping neurons firing, synapses intact, the immune system in check, and waste products removed from the body. Hibernators are valuable models for biological study because they show remarkable resilience to circumstances like hypothermia, ischemia, and reperfusion stress, which would injure non-hibernating mammals severely. Molecular and cellular mechanisms underlying neuroprotection, cardiac stability, and muscle preservation in hibernators need to be better understood in order to further treatments for organ preservation, hypothermia, stroke, and even long-duration spaceflight.

Defining Hibernation and Torpor

Hibernation is a long-term physiological adaptation that allows animals, particularly mammals in temperate and polar regions, to survive periods of extreme cold and food scarcity. Animals go into a condition of reduced metabolic activity called hibernation, when they lower their body temperature, slow their heart rate, suppress their respiration, and limit their energy intake. This state lasts for a long time. Hibernation, in contrast to sleep, can endure for weeks or months with very short awakenings. Bears, ground squirrels, hedgehogs, and bats are some classic examples. To keep energy costs down, this mechanism mostly uses fat stores that have built up during times of plenty.

Torpor, by contrast, is a shorter-term and more flexible state of reduced metabolic rate and body temperature, often lasting for hours to a few days. The occurrence might be daily or irregular, and it aids animals in enduring temporary food shortages or difficult circumstances. Daily torpor is a frequent energy-saving mechanism for small mammals and birds, including hummingbirds, mice, and some marsupials, especially when temperatures drop. Upon waking from torpor, animals can resume their regular activities much more rapidly than during hibernation.

Thermoregulation and Body Temperature Control

An animal's ability to keep its internal temperature within a reasonable range, regardless of changes in its surrounding environment, is known as thermoregulation. Assuring the best possible circumstances for cellular metabolism, enzymatic function, and survival, it is an essential part of homeostasis. Both ectothermy and endothermy are shown by animals. Reptiles, amphibians, and the majority of fish are ectothermic, meaning they control their internal body temperature mostly by behavioral adaptations like basking in the sun or hiding in shade. Mammals and birds are endothermic, meaning they produce heat internally and regulate their

body temperature through processes including shivering, perspiration, panting, vasodilation, and vasoconstriction. Some endothermic species exhibit heterothermy, going back and forth between phases where their body temperature drops (hibernation, torpor, etc.) and ones where it stays high (metabolic temperature). An intricate web of interactions between the neurological system, the endocrine system, and the circulatory system regulates core body temperature. The hypothalamus receives information from the body's temperature receptors and processes it in order to set the thermostat in the right place. Shivering thermogenesis, brown adipose tissue metabolism, and peripheral vasoconstriction are a few examples of the systems that enhance heat generation and decrease heat loss in cold conditions. On the flip side, when it is hot outside, body activities like perspiration, panting, and cutaneous vasodilation help dissipate heat. Behavioral measures like looking for water or burrows can also help. Differences in body size, habitat, and evolutionary adaptations determine the efficacy of thermoregulatory systems among species. Large animals may have trouble dissipating heat in hot climates, whereas smaller endotherms rely on daily torpor and lower surface-area-to-volume ratios to keep themselves warm. Animals in the desert survive the night and avoid the scorching heat by saving water and using specialized adaptations like gular flapping to cool themselves through evaporation. Thermoregulation refers to the ability of organisms to maintain their body temperature within a suitable range for proper physiological functioning. Climate change directly affects this process by altering environmental temperatures, making it difficult for many species to regulate their internal heat balance. Organisms can broadly be classified into ectotherms (cold-blooded animals like reptiles, amphibians, and fish) and endotherms (warm-blooded animals like birds and mammals), and both groups are impacted in different ways.

Ectothermic animals depend largely on external environmental conditions to control their body temperature. With rising global temperatures, these organisms may experience overheating, reduced activity periods, and increased metabolic stress. For example, reptiles that rely on basking to warm up may face difficulty avoiding excessive heat, which can impair physiological processes such as digestion, reproduction, and movement. In extreme cases, prolonged exposure to high temperatures can lead to mortality. Endothermic animals, on the other hand, regulate their body temperature internally through metabolic processes, but this also comes at an energy cost. As environmental temperatures rise, these animals must expend more energy on cooling mechanisms such as sweating, panting, or seeking shade. This increased energy demand can reduce the energy available for growth, reproduction, and immune function. In colder regions, warming may initially seem beneficial, but it can disrupt seasonal adaptations such as hibernation or migration.

Climate change also affects behavioral thermoregulation, where animals adjust their activities to maintain optimal body temperature. Many species may shift their activity patterns to cooler times of the day, such as becoming more nocturnal. However, such behavioral changes can increase competition for resources and alter predator-prey interactions. Additionally, suitable microhabitats (like shaded areas or burrows) may become scarce due to habitat degradation, limiting the ability of species to regulate temperature effectively. Another important aspect is the impact on reproductive success. In some species, especially reptiles, temperature determines sex during embryonic development. Rising temperatures can skew sex ratios, leading to long-term population imbalances. Furthermore, extreme heat can affect egg viability, reduce fertility, and disrupt breeding cycles. Thermoregulation is also closely linked to

physiological stress and survival limits. Each species has a thermal tolerance range, beyond which survival becomes difficult. Climate change is pushing many species closer to or beyond these limits, increasing the risk of population decline or extinction, particularly for those living in already warm environments.

Molecular and Genetic Basis of Hibernation

Mammalian hibernation is a complex survival mechanism that allows organisms to suppress metabolism, tolerate hypothermia, and periodically arouse without long-term physiological damage. The molecular and genetic basis of this mechanism is complex, but it is reversible. Adapting cellular physiology to changing environmental conditions and seasons relies on dynamic control of gene expression, protein synthesis, and epigenetic alterations. Researchers have shown that while mammals like bats, ground squirrels, and bears go into torpor, their gene activity changes selectively. Specifically, genes involved in energy-intensive processes like protein synthesis, cell cycle progression, and immune activation are downregulated, while genes related to stress resistance, DNA repair, antioxidant defense, and lipid metabolism are upregulated. In order to conserve energy, this pattern makes sure that non-essential activities are turned off and that protective pathways are turned up to protect cells from damage during metabolic depression and rewarming. Protein abundance and post-translational modifications, such as phosphorylation and acetylation, have been found to change in proteomic analyses. These changes impact mitochondrial function, chromatin remodeling, and enzymatic activity, all of which contribute to metabolic flexibility and stress tolerance.

The ability to undergo reversible changes in gene expression during hibernation is greatly facilitated by epigenetic control. Hibernators are able to switch between a repressed and an active metabolic state without undergoing irreversible genetic changes because changes to histone modifications and DNA methylation patterns affect the accessibility of chromatin and transcriptional activity. For instance, during torpor, genes involved in cell proliferation are hypermethylated to conserve energy, and following arousal, histone acetylation at the promoters of metabolic and stress-responsive genes can enhance rapid transcriptional responses. Post-transcriptional regulators like as microRNAs (miRNAs) and other non-coding RNAs play an important role as well, adjusting protein synthesis and repressing pathways that use too much energy. Hibernation is associated with an increase in microRNAs (miRNAs) that promote energy conservation and cellular survival under hypometabolic conditions by targeting mRNAs involved in mitochondrial energy generation, growth factor signaling, and apoptosis.

During extended fasting, the metabolic level shifts from energy metabolism based on carbohydrates to lipid oxidation, which is more efficient and sustained because it is controlled genetically. A coordinated metabolic reprogramming is indicated by the downregulation of key enzymes in glycolysis and the citric acid cycle and the upregulation of genes encoding enzymes involved in fatty acid transport, β -oxidation, and ketone body metabolism. Not only does this lipid-centric metabolism reduce oxidative stress during hypothermia and rewarming, but it also offers sustained energy by minimizing the formation of reactive oxygen species (ROS). Significantly, molecular adaptations also strengthen mitochondrial resilience; for example, antioxidant enzymes and uncoupling proteins are overexpressed, shielding cells from reactive oxygen species (ROS) damage; and changes to the activity of the electron transport chain

guarantee that ATP production is adequate even when metabolism is down. Hibernation is a complex physiological adaptation that allows certain animals to survive periods of extreme cold and food scarcity by entering a state of reduced metabolic activity. At the molecular and genetic level, hibernation involves highly regulated changes in gene expression, protein activity, and cellular metabolism, enabling organisms to conserve energy while maintaining vital functions. One of the central features of hibernation is metabolic suppression. During this state, metabolic rate can drop to as low as 1–5% of normal levels. This is achieved through the downregulation of genes involved in energy-intensive processes such as cell growth, protein synthesis, and active transport. At the same time, genes that support energy conservation, stress resistance, and cellular protection are upregulated. This coordinated gene expression ensures that the organism minimizes energy expenditure while protecting tissues from damage.

A key molecular pathway involved in hibernation is the regulation of mitochondrial function, as mitochondria are responsible for energy production. During hibernation, mitochondrial activity is reduced, leading to decreased ATP production and oxygen consumption. Additionally, changes in enzyme activity help shift metabolism from carbohydrate utilization to lipid (fat) metabolism, which provides a more efficient and long-lasting energy source during prolonged inactivity. Another important aspect is the role of transcription factors and epigenetic modifications. Transcription factors regulate which genes are turned on or off during hibernation, while epigenetic mechanisms such as DNA methylation and histone modification allow reversible changes in gene expression without altering the DNA sequence. These processes enable animals to enter and exit hibernation cycles efficiently in response to environmental cues like temperature and food availability. Protein stability and cellular protection mechanisms are also critical. During hibernation, cells must withstand low temperatures and reduced blood flow. Special proteins, such as heat shock proteins and antioxidant enzymes, are produced to prevent protein denaturation, oxidative stress, and cellular damage. These protective mechanisms help maintain cellular integrity even under extreme physiological conditions.

Hibernation also involves controlled reduction in body temperature and heart rate, regulated by genetic and hormonal signaling pathways. Hormones such as melatonin and thyroid hormones play a role in initiating and maintaining the hibernation state. In some species, body temperature can drop close to the ambient temperature, significantly reducing energy requirements. At the genetic level, studies have identified specific genes associated with hibernation traits, including those involved in circadian rhythms, metabolism, and stress response. These genes enable animals to anticipate seasonal changes and prepare physiologically for hibernation. Interestingly, many of these genetic pathways are conserved across species, suggesting a common evolutionary mechanism. From a broader perspective, understanding the molecular and genetic basis of hibernation has important implications for science and medicine. It provides insights into metabolic regulation, organ preservation, and potential applications such as improving outcomes in organ transplantation or developing therapies for metabolic disorders.

Conclusion

To ensure survival during long periods of resource scarcity and harsh climatic conditions, mammals undergo hibernation, which is a remarkable adaptation of their physiological systems

to suppress metabolism, regulate body temperature, and preserve organ and tissue integrity. At its foundation, hibernation is a state of controlled hypometabolism that is attained by significant changes in energy utilization. Specifically, the reliance shifts from carbohydrates to lipid oxidation, and this is accompanied by mitochondrial reprogramming and antioxidant defenses that minimize cellular damage and oxidative stress. An intricate web of neuroendocrine signals underpins metabolic plasticity; the hypothalamus, in particular, plays a pivotal role in coordinating the onset, maintenance, and arousal of sleep in response to environmental cues and hormonal mediators such as insulin, thyroid hormones, and melatonin. Molecular and genetic changes in gene expression, protein synthesis, and epigenetic regulation allow for the suppression of energetically costly processes and the upregulation of protective pathways that confer resilience to hypothermia, ischemia, and inactivity during hibernation. This process is equally remarkable. Despite the energy cost, periodic awakenings demonstrate how hibernators have evolved to conserve energy while keeping their neurological, immune, and physiological systems functioning. This adaptation allows them to survive in harsh environments that would kill off most mammals that do not hibernate. These adaptations show the variety of techniques mammals have developed to deal with seasonal and unpredictable resource availability, which is important for species survival, ecosystem stability, and resistance to environmental fluctuation. They are also significant from an evolutionary and ecological perspective. In addition to its biological significance, research into hibernation has great biomedical promise. By understanding how muscles and organs naturally deal with hypothermia, ischemia-reperfusion, and disuse, we can create models to help improve organ preservation, treat cardiac arrest and stroke, prevent osteoporosis and muscle atrophy, and create protective measures for long-duration spaceflight. Recent developments in molecular biology, genetics, and proteomics have shed light on the regulatory network underlying hibernation, showing that it is remarkably adaptable and reversible, which may lead to new ways of treating human diseases. The complex metabolic, neuroendocrine, and molecular mechanisms that underlie mammalian hibernation are a marvel of evolutionary adaptation and a cutting edge in translational research. Ultimately, this state of reversible suspended animation is the result of these mechanisms. Our understanding of mammalian biology will be enhanced, and our methods for addressing human health, resilience, and survival in harsh environments will be revolutionized, if we can decipher and apply the principles of hibernation.

Bibliography

- Andrews, M. T. (2007). Advances in molecular biology of hibernation in mammals. *BioEssays*, 29(5), 431–440. <https://doi.org/10.1002/bies.20560>
- Carey, H. V., Andrews, M. T., & Martin, S. L. (2003). Mammalian hibernation: Cellular and molecular responses to depressed metabolism and low temperature. *Physiological Reviews*, 83(4), 1153–1181. <https://doi.org/10.1152/physrev.00008.2003>
- Geiser, F. (2013). Hibernation. *Current Biology*, 23(5), R188–R193. <https://doi.org/10.1016/j.cub.2013.01.062>
- Hampton, M., & Andrews, M. T. (2019). Hibernation as a model of organ protection: Lessons from natural animal models. *Journal of Applied Physiology*, 127(6), 1621–1632. <https://doi.org/10.1152/jappphysiol.00491.2019>

- Jastroch, M., Giroud, S., Barrett, P., & Geiser, F. (2016). Hibernation and daily torpor: Molecular and metabolic mechanisms. *Journal of Comparative Physiology B*, 186(8), 563–579. <https://doi.org/10.1007/s00360-016-0995-3>
- Lyman, C. P., Willis, J. S., Malan, A., & Wang, L. C. H. (1982). *Hibernation and torpor in mammals and birds*. Academic Press. <https://doi.org/10.1016/C2013-0-10344-0>
- Martin, S. L. (2008). Mammalian hibernation: A naturally reversible model for insulin resistance in man? *Diabetes & Vascular Disease Research*, 5(2), 76–81. <https://doi.org/10.3132/dvdr.2008.013>
- Morin, P., & Storey, K. B. (2009). Mammalian hibernation: Differential gene expression and novel application of epigenetic controls. *International Journal of Developmental Biology*, 53(2-3), 433–442. <https://doi.org/10.1387/ijdb.082689pm>
- Staples, J. F. (2016). Metabolic flexibility: Hibernation, torpor, and estivation. *Comprehensive Physiology*, 6(2), 737–771. <https://doi.org/10.1002/cphy.c140064>
- Vázquez-Medina, J. P., Zenteno-Savín, T., & Elsner, R. (2010). Antioxidant defenses in marine mammals. *Free Radical Biology and Medicine*, 49(4), 503–511. <https://doi.org/10.1016/j.freeradbiomed.2010.04.002>