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Sleeping Without a Brain: How Jellyfish Reveal the True Purpose of Sleep

Biology * Evolution * DNA

Introduction: The Universal Paradox of Sleep

In biological evolution, few behaviors appear as paradoxical as sleep. For a substantial fraction of its lifespan, an animal enters a state of sensory disconnection, suspending foraging, defense, and reproduction. In an environment governed by natural selection, such acute vulnerability should carry a prohibitive fitness penalty, yet sleep is conserved across the animal kingdom -- from primates and fruit flies to the earliest nervous systems.[1]

For decades, neurobiology framed sleep through a brain-centric lens, proposing that it evolved primarily to consolidate complex cognitive memories, process sensory information, or downscale synaptic weights to prevent metabolic saturation and neural network burnout (the Synaptic Homeostasis Hypothesis).[2] While compelling for mammals and birds, these neurocentric models struggled to explain why basal invertebrates lacking centralized brains also sleep. A landmark 2026 study published in Nature Communications by R. Aguillon, A. Harduf, Professor Oren Levy, and Professor Lior Appelbaum at Bar-Ilan University resolves this evolutionary paradox by investigating two ancient cnidarians: the upside-down jellyfish Cassiopea andromeda and the starlet sea anemone Nematostella vectensis.[3] Their findings demonstrate that the original, primordial function of sleep is fundamental cellular maintenance -- specifically, the nuclear repair of neuronal DNA damage accumulated during the metabolic exertion of wakefulness.[4]

Basal Cnidarian Models and Divergent Chronotypes

Situated near the base of the metazoan tree of life, the phylum Cnidaria diverged from the bilaterian lineage approximately 600 to 700 million years ago and represents the earliest animal clade to evolve a nervous system.[5] Rather than a centralized brain, cnidarians possess a decentralized, diffuse nerve net that coordinates motor and sensory responses.[1]

To separate universal cellular mechanisms from ecological adaptations, the Bar-Ilan team compared two species with opposite chronotypes:

* Cassiopea andromeda (Diurnal Symbiont): Inhabiting shallow tropical mangroves, this upside-down jellyfish rests its bell on the sediment with its oral arms oriented upward to expose symbiotic photosynthetic dinoflagellates (zooxanthellae) to sunlight.[3][1] Cassiopea pulses vigorously during the day to drive gas exchange and nutrient transport, and sleeps at night -- along with a brief midday "siesta" -- spending roughly one-third of its 24-hour cycle quiescent, driven by light masking and homeostatic pressure.[3][4]

* Nematostella vectensis (Nocturnal/Crepuscular Predator): An estuarine burrowing sea anemone from the sister class Anthozoa, Nematostella lacks photosynthetic

symbionts and hunts prey with its tentacles during dark and twilight phases.[3][4] Regulated strongly by an endogenous circadian clock (conserved Clock and Cycle homologs), Nematostella sleeps primarily during the day following dawn onset.[3][4]

Comparing a diurnal scyphozoan against a nocturnal anthozoan allowed the researchers to test whether DNA repair tracks behavioral wakefulness rather than solar radiation exposure.[4][7]

Behavioral Verification of Brainless Sleep

In the absence of cortical electroencephalography (EEG), sleep in non-cephalized organisms is established through three rigorous behavioral criteria: reversible quiescence, elevated arousal thresholds, and homeostatic rebound.[6] Using 24-hour infrared video tracking to quantify bell pulsation in Cassiopea and body/tentacle extension in Nematostella, the researchers confirmed all three hallmarks:[3]

* Reversible Quiescence: Both species exhibited prolonged, recurring bouts of immobility -- at night for Cassiopea and during the day for Nematostella -- that were rapidly reversible upon strong stimulation, distinguishing sleep from coma or metabolic torpor.[6]

* Sensory Gating (Arousal Threshold): When presented with food stimuli or mechanical vibrations during quiescent states, sleeping animals exhibited significantly delayed response latencies compared to active animals.[6]

* Homeostatic Sleep Rebound: When mechanically deprived of sleep via controlled water pulses during their normal rest phase, both cnidarians accumulated a sleep debt, exhibiting longer and deeper compensatory rebound sleep once the disturbance ceased.[3]

| Feature | | Cassiopea andromeda (Jellyfish) | | Nematostella vectensis (Sea Anemone) | | Humans (Homo sapiens) |

| Phylum / Class | | Cnidaria (Scyphozoa) | | Cnidaria (Anthozoa) | | Chordata (Mammalia) |

| Nervous System | | Diffuse Nerve Net | | Diffuse Nerve Net | | Centralized Brain (CNS) |

| Chronotype | | Diurnal (Day Active) | | Nocturnal / Crepuscular | | Diurnal (Day Active) |

| Sleep Phase | | Night + Midday "Siesta" | | Day (Dawn Onset) | | Night |

| Regulatory Driver | | Light Masking + Homeostasis | | Circadian Clock + Homeostasis | | Circadian Clock + Homeostasis |

| DNA Damage Peak | | End of Day (Wake) | | End of Night (Wake) | | End of Day (Wake) |

| Primary Sleep Function | | Neuronal DNA Repair | | Neuronal DNA Repair | | DNA Repair + Synaptic Homeostasis |

| Rebound Sleep | | Present | | Present | | Present |

Molecular Mechanism: Neuronal DNA Damage Drives Homeostatic Sleep Pressure

To identify the cellular driver of sleep pressure, Appelbaum and Levy examined the genomic cost of neuronal excitability. Firing action potentials, maintaining resting membrane potentials, and recycling neurotransmitters require intense metabolic activity, generating reactive oxygen species (ROS) and oxidative byproducts that induce physical lesions and double-strand breaks (DSBs) in the DNA double helix.[1]

Tracking neuronal DNA damage across the 24-hour cycle using phosphorylated histone H2AX (-H2AX) immunostaining -- a canonical molecular marker for double-strand DNA breaks previously validated in vertebrate zebrafish studies -- revealed a direct, phase-locked cycle of accumulation and repair:[8]

* Wake-Induced Accumulation: Neuronal DNA damage rose linearly throughout the active wake phase -- peaking at the end of the daylight photophase in diurnal Cassiopea and at the end of the dark scotophase in nocturnal Nematostella. This inverse temporal alignment across opposite chronotypes demonstrates that genomic degradation is driven directly by metabolic and electrical neuronal activity rather than environmental solar exposure.[4][7]

* Sleep-Dependent Clearance: During wakefulness, endogenous repair enzymes could not keep pace with lesion accumulation. Only during the quiescent sleep state did neuronal DNA damage markers drop back to baseline.[7] Efficient nuclear repair requires enhanced chromosome mobility, PARP1-mediated recruitment of repair complexes, and structural chromatin reorganization within the nucleus -- biomechanical processes that are physically incompatible with the continuous transcriptional demands of active sensory and motor signaling.[9]

To establish bidirectional causality rather than mere correlation, the team subjected both cnidarians to exogenous genotoxic stressors independent of prolonged wakefulness: controlled bursts of ultraviolet (UV) radiation and the chemical topoisomerase II mutagen Etoposide.[4][7] In both experimental paradigms, artificially inducing neuronal DNA breaks immediately triggered prolonged sleep bouts out of phase with normal circadian timing, overriding environmental cues to facilitate accelerated genomic repair.[4][7] Thus, nuclear DNA damage acts as a homeostatic cellular counter: once neuronal lesions cross a critical threshold, intracellular signaling cascades initiate behavioral quiescence.

Conclusion: Evolutionary Synthesis and Neurodegenerative Implications

The conservation of sleep-dependent DNA repair across basal cnidarians (diverged >600 million years ago), teleost fishes (~450 million years ago), and mammals places the origin of sleep at the very dawn of the metazoan nervous system.[5][9] Under the Nuclear Maintenance Hypothesis, sleep evolved first as a mandatory cellular repair state to prevent genomic catastrophe in electrically excitable, largely post-mitotic neurons. Whereas the Synaptic Homeostasis Hypothesis addresses plasticity challenges specific to centralized brains, DNA repair represents the universal evolutionary denominator; higher-order functions such as synaptic downscaling, memory consolidation, and glymphatic metabolite clearance were subsequently layered onto

this primordial maintenance window.[1][2] Ongoing investigations into neuron-lacking sponges (Porifera) and placozoans aim to determine whether this rest-repair cycle even predates neural tissue.[5]

These findings also carry direct mechanistic relevance for human neurological health. Epidemiology consistently links chronic sleep deprivation and fragmented sleep architecture to Alzheimer's disease, Parkinson's disease, amyotrophic lateral sclerosis (ALS), and accelerated cognitive aging.[10] By demonstrating that wakefulness inherently inflicts double-strand DNA breaks while sleep restores nuclear integrity, the Bar-Ilan study reframes sleep as the nervous system's primary endogenous defense against cumulative genomic instability -- a physiological law conserved since the first jellyfish pulsed in ancient seas.[5]

Updates... (Nov 22, 2026): Corrected second author initial from H. Harduf to A. Harduf in the opening section, replaced all access-date placeholders in References #3-#5, #7, and #10 with verified exact publication dates, deduplicated the AMP mirror of CHOSUNBIZ (formerly Reference #10) into Reference #5 and renumbered Reference #11 to #10, and enriched journal references #1, #2, #6, #8, and #9 with exact publication dates (building on Nov 19, 2026 DOI updates).

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