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Article

The Sentinel Sleep Theory: The Biological Function of REM Sleep Unveiled

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Abstract: The biological function of rapid eye movement (or REM) sleep—one of the great mysteries of neuroscience—remains unknown. Here, I demonstrate that the biological function of REM sleep is to heighten brain alertness, significantly reducing the high vulnerability of deep sleep. Every organism with a nervous system must undergo deep sleep: a necessity that comes with substantial vulnerability. Deep sleep, by lowering alertness, compromises the organism's safety, putting its survival at risk. Therefore, REM sleep is a necessary adaptation for any organism that must sleep. My primary goal here is to present a comprehensive conceptual framework—supported by extensive empirical evidence—to connect numerous disparate empirical findings under a unified theory of the biological function of REM sleep. Additionally, I also provide a historical narrative to explain its origin and subsequent evolution. The theory I developed to explain the function of REM sleep is not only widely corroborated but has also resisted numerous attempts at refutation. This allows me to claim that I solved this great enigma of neuroscience.

Keywords: REM sleep; N-REM sleep; theory; scientific theory; biological function; evolution; evolutionary biology; evolutionary origin; historical narrative

1. INTRODUCTION

Why is it so difficult for scientists to describe what Rapid Eye Movement sleep is? The primary reason is that they still do not understand the biological function of this sleep state. If you do not know the function of a mechanism, you are limited to describing its physical and behavioral aspects. Part of the difficulty is also due to REM sleep being many things: a brain state, a behavior, a dreaming state, as well as a paradoxical state (Blumberg et al., 2020). I aim to describe REM sleep beyond its physiological, neurophysiological, neurochemical, neurobiological, and behavioral aspects. To do so, I will need to resolve the enigma of its function. Only then can I describe it more broadly from this resolution, including the evolutionary reason for its existence. This is my objective here. I will begin by summarizing some of its fundamental characteristics.

Scientists classify REM sleep as a sleep state because arousal thresholds increase in this state (Andrillon and Kouider, 2020; Ermis et al., 2010), causing the organism to stop responding behaviorally to the external environment in the same way it does during wakefulness (Tainton-Heap et al., 2021). Indeed, the arousal thresholds of mammals can be as high during REM sleep as they are during N-REM sleep (Andrillon and Kouider; Dillon and Webb, 1965; Ermis et al., 2010; Siegel and Langley, 1965; Tainton-Heap et al., 2021). During REM sleep, the sleeping organism (with an elevated arousal threshold) exhibits neural activity analogous to that of wakefulness (Blumberg et al., 2020; Tainton-Heap et al., 2021). The physiology during REM sleep is so similar to wakefulness that the electroencephalogram (EEG) shows electrical activity almost indistinguishable from that occurring in the brain during wakefulness (Bear et al., 2016, p. 659). This is why REM sleep was originally termed paradoxical sleep (Tainton-Heap et al., 2021).

Especially in mammals and birds, both the REM sleep period and the deep sleep period—called non-REM (or N-REM) sleep—are marked by specific and easily distinguishable physiological changes (Rattenborg et al., 2019; Yamazaki et al., 2020). The physiological changes that occur during the REM period contrast with those of the N-REM period by exhibiting a comparatively higher frequency (Purves et al., 2004, p. 671). Unsurprisingly, the REM period increases energy expenditure

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(Mignot, 2008). After all, metabolic activity, blood pressure, and respiratory and heart rates rise to levels that appear as if the organism is awake (Mignot, 2008, Purves et al., 2004, p. 671; Yamazaki et al., 2020).

During REM sleep, brain metabolism increases by about 20% due to the higher intensity of neural activity, making it clear that the brain does not rest in this state (Bear et al., 2016, p. 660; Peever and Fuller, 2017). Considering that the reverberation of neural patterns during sleep is energetically more costly than neuronal silencing (Kandel et al., 2013, p. 1157) and that REM sleep causes a significant energy expenditure, this indicates that REM sleep plays a critical role. After all, non-random elimination is prolific in eliminating waste. Nothing so costly lasts for several million years unless it serves an important function—a frequently neglected evolutionary consequence (McFadden, 2022, p. 268).

Many scientists tried to uncover the function of REM sleep. None of them succeeded. Their proposals were not unanimously accepted, both because they are incapable of explaining an abundance of disparate facts pertaining to the domain of REM sleep and because they are inconsistent with the evidence, or at least with parts of it. Here are some of the various hypotheses already proposed: *learning* (Moruzzi and Eccles, 1966); *sentinel function* (Snyder, 1966); *psychological health* (Kollar et al., 1969); *reverse learning* (Crick and Mitchison, 1983); *energy regulation* (Siegel, 2005); *sensorimotor integration* (Hong et al., 2009); and *defensive activation of the visual cortex* (Eagleman and Vaughn, 2021). Despite these attempts (and others not listed), the question "What is the biological function of REM sleep?" remains unsolved and stands as one of the major enigmas of neuroscience—indeed, of science (Akre, 2024; Bear et al., 2016, p. 666; Kandel et al., 2021, p. 1080; Peever and Fuller, 2016; Siegel, 2011).

In 1966, Frederick Snyder published an article titled "Toward an Evolutionary Theory of Dreaming." In it, the author presented the "sentinel hypothesis" to try to explain the function of REM sleep. Although this concept was later developed (e.g., Vertes, 1986), the sentinel function of REM sleep remained a *hypothesis*. My goal in this article is to develop this concept into a *theory* of the function of REM sleep. This highlights the disparity between my work and Snyder's. The contribution I aim to make is to solve the mystery of the primary function of REM sleep, not to propose or elaborate a hypothesis, as Snyder did. As he himself stated, his article "is intended merely as a first and uncertain step toward an evolutionary theory of dreaming." With this, I hope to demonstrate that, despite the hypothesis being Snyder's, the theory is mine.

As not all scientists are scrupulous with terminology and fail to distinguish between the terms "hypothesis" and "theory," this needs to be considered to avoid accusations of misconduct when I assert that the theory is mine. There is an abyss between a hypothesis and a theory (Dawkins, 2010a, pp. 9-10; Gazzaniga et al., 2016, pp. 37-38; Nelson and Cox, 2013, p. v). Therefore, I prefer to define these terms.

A *hypothesis* refers to the equivalent of a conjecture or speculation. It also refers to a specific and easily testable prediction formulated based on a theory, concept, or knowledge. In the case of a hypothesis formulated from a theory, testing it serves to substantiate the theory or to refute it totally or partially (Dawkins, 2010a, pp. 9-10; Gazzaniga et al., 2016, pp. 37-38; dos Reis, 2016, pp. 24-25; Nelson and Cox, 2013, p. v; Sagan, 1996, pp. 172-173, 208; Sokal and Bricmont, 2016, p. 67).

The typical characteristics and purposes of hypotheses are: (1) to present a provisional solution to a specific problem; (2) to present an explanation or prediction of a limited nature (which is opposed to the generalized nature of a theory); (3) to be logically consistent and in accordance with current scientific knowledge; (4) to be testable through its empirical consequences or by logical or mathematical means (Gazzaniga et al., 2016, pp. 37-38; dos Reis, 2016, p. 21; Nelson and Cox, 2013, p. v; Walton, 2008, p. 214). Once proposed, the fate of a hypothesis is to be confirmed or refuted by scientific research. Scientists formulate hypotheses with this objective (Gazzaniga et al., 2016, pp. 37-38; dos Reis, 2016, p. 21).

A *theory* refers to a set or system of interconnected assertions or concepts that explain or justify an extensive group of disparate facts or phenomena belonging to a specific domain (e.g., all the facts collected about REM sleep). A theory encompasses hypotheses, facts, and laws (when applicable) to

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explain a multitude of previously collected evidence and to propose a series of specific predictions about future events—a crucial characteristic of a good scientific theory (Dawkins, 2010a, pp. 9-10; Gazzaniga et al., 2016, p. 37; Serway and Jewett, 2014, p. 2; Weiskopf, 2024). What makes a scientific theory good is much more its ability to generate testable hypotheses than its empirical foundation (Gazzaniga et al., 2016, p. 37). The more testable hypotheses a theory encompasses in its conceptual body, the better it is.

Now that I defined these two important terms, it is clear that the work Snyder did in his 1966 article is far from being considered a theory. As we have seen, he himself stated that his proposal was merely a hypothesis. It is the disparity between hypothesis and theory that allows me to assert that the sentinel sleep theory is mine, being a contribution to the hypothetical concept proposed by Snyder. Therefore, in addition to considering the rigorous distinction between "hypothesis" and "theory" (typical of the philosophy of science context), I suggest that you also carefully read Snyder's article to perceive the disparity between his work and mine, as well as the contributions of each.

My scientific contribution is to present a comprehensive conceptual framework—supported by extensive empirical evidence—that will turn the sentinel *hypothesis* into the *theory* of sentinel sleep. To demonstrate the validity and robustness of my theory, I drew on a substantial body of evidence that corroborates it and, more importantly, showed that numerous attempts to refute it failed. This made my article long., It could not be otherwise. To prove that I solved one of the main enigmas of science, I need this length. Both to thoroughly present and delve into the facts and arguments that support the theory, as well as to demonstrate the failed attempts to refute it.

2. THE SENTINEL SLEEP THEORY

To present my conclusions concerning the biological function of REM sleep, I must first engage in a necessary digression. I need to address the importance of N-REM sleep first. There is still no consensus on the function (or functions) of N-REM sleep. Despite this, it is evident that it serves an essential biological function. N-REM sleep is not merely a dispensable luxury; it is strictly necessary for the brain, for the body, and for the survival of the organism (Cirelli and Tononi, 2008; Jaggard et al., 2021; Kandel et al., 2021, p. 1097; Mignot, 2008; Urry et al., 2020, p. 1094). For the brain to function normally, sleep is a necessary condition (Kandel et al., 2021, p. 1097).

A defining characteristic of this behavioral state is the marked reduction in alertness to the immediately surrounding environment (Anafi et al., 2019; Capellini et al., 2008; Ramón et al., 2004; Rattenborg and Ungurean, 2023). Something that clearly distinguishes the state of sleep from the state of wakefulness is the reduced responsiveness to environmental stimuli (Capellini et al., 2008; Nath et al., 2017; Rattenborg and Ungurean, 2023). Sleep undermines attention and, eventually, suspends consciousness (in those who possess it) (Damasio, 2003, p. 202; Damasio, 2012, pp. 240-241; Ramón et al., 2004). As the brain is gradually subjected to deeper sleep (stage 3 of N-REM sleep), its alertness mechanisms are inactivated. When in the deepest stage of sleep, the brain exhibits the greatest inactivation of its alertness mechanisms (e.g., in the brainstem, anterior cingulate cortex, and thalamus) (Dang-Vu et al., 2010; Jan et al., 2009; Kandel et al., 2013, p. 1141; Moyne et al., 2022; Ramón et al., 2004). However, this inactivation is not total. Even during N-REM sleep, the brain (albeit mildly) monitors the surrounding environment for potential dangers and can respond differentially to specific prominent stimuli (e.g., unfamiliar sounds) (Gazzaniga et al., 2016, p. 146; Moyne et al., 2022).

During wakefulness, the organism readily responds to exteroceptive stimuli intercepted by some "sensory portal" (a term used by Damasio [2012] that I will borrow here). During N-REM sleep, however, exteroceptive stimuli need to be more intense for the organism to respond to them (Moyne et al., 2022; Rattenborg and Ungurean, 2023). Therefore, from an adaptive perspective, sleep could seem illogical, effectively a contradiction. The greater neural inactivation characteristic of N-REM sleep—where firing rates and energy use reach their lowest levels during the day—certainly constitutes a substantial risk to the survival of the organism. After all, greater neural inactivation equals greater vulnerability (Anafi et al., 2019; Bear et al., 2016, p. 659; Capellini et al., 2008; Gazzaniga

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et al., 2016, p. 148; Libourel and Herrel, 2016; Ramón et al., 2004; Rattenborg and Ungurean, 2023). This is why sleeping animals are highly vulnerable to predation (Anafi et al., 2019).

If N-REM sleep did not serve a critical biological function, the central nervous system of countless species would have, over the course of evolution, overcome the need to undergo such a highly vulnerable mental and behavioral state (Anafi et al., 2019; Bear et al., 2016, pp. 662-663; Mignot, 2008). Therefore, *the fact that N-REM sleep persisted throughout evolution is due to its being strictly necessary* (even if we do not yet know exactly why).

Here, I set out to address the biological function of REM sleep, not that of N-REM sleep. Of the latter, only two characteristics are pertinent. The first is that it is present in all animal species with a nervous system, no matter how simple and decentralized it is (Cirelli and Tononi, 2008; Libourel and Herrel, 2016; Nath et al., 2017; Vyazovskiy and Harris, 2013; Zimmerman et al., 2008). The second is that it substantially reduces alertness to the surrounding environment, making the organism highly vulnerable to predation (Anafi et al., 2019). It is from this context—exacerbated vulnerability and the non-negotiable need for N-REM sleep—that we can understand the function of REM sleep. Here is a summary of my main conclusions regarding its function:

1. *The primary biological function of REM sleep is to reduce the vulnerability caused by N-REM sleep*. The brain being subjected to a state of deep sleep is necessary, but makes the organism substantially vulnerable, risking its survival. The REM period makes the brain more active—in a state of sleeping vigilance—to increase the organism's alertness to its surroundings, resulting in greater protection. After all, the greater the brain's alertness to the immediate environment, the higher the chances of the organism surviving when a sensory portal detects a sudden threat.
2. *The parameters of REM sleep depend on the organism's vulnerability*. The time invested in REM sleep is inversely proportional to body weight and muscle strength. Greater weight or muscle strength implies less time invested in REM sleep and vice versa. And not only is the total time of REM sleep affected. Its latency (i.e., the period between the onset of sleep and the occurrence of the first REM sleep episode) and its intensity are also affected. Furthermore, muscle strength and weight are not the only protection-related factors that affect REM sleep parameters. Generally, the better protected the organism is (lower vulnerability), the less time the brain will invest in REM sleep, and the longer its latency; the less protected the organism is (higher vulnerability), the more time the brain will invest in REM sleep, and the shorter its latency. The time the brain invests in the REM period, as well as the duration of each episode, latency to the first episode, and its density (or intensity), depend on the information provided by all varieties of mental mappings—interoceptive, proprioceptive, and exteroceptive (especially proprioceptive information).
3. *REM sleep is highly adaptive*. In the absence of what we happen to call "REM sleep," the crucial N-REM sleep would leave the organism highly vulnerable. When, by mere chance, a genetic mutation contributed to the emergence of an organism whose vulnerability due to N-REM sleep was reduced, non-random elimination (or natural selection) promptly favored this adaptive mutation. And given the high adaptive value of this novelty, it did not remain restricted to the lineage in which it originally debuted. It spread widely across various species.
4. *REM sleep is cyclical due to its protective function*. The function of REM sleep—to significantly reduce the vulnerability of N-REM sleep—reaches its full potential when it occurs periodically throughout N-REM sleep, rather than occurring only once.
5. *REM sleep evolved from a brief awakening from N-REM sleep*. The most plausible scenario regarding the evolutionary origin of REM sleep is that it emerged from an error. This error

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caused the organism to briefly wake up from deep sleep before its usual awakening, providing a limited but effective adaptive advantage. Consequently, this trait spread and, over the course of species evolution, became more complex. Eventually, this protective mechanism became REM sleep as we know it today.

Following the order in which the items above were presented, the sentinel sleep theory will be thoroughly explained, and its factual foundation demonstrated. I established a separate subsection for each of the five items above (2.1, 2.2, 2.3, 2.4, and 2.5), each addressing a specific part of the theory.

2.1. THE PRIMARY BIOLOGICAL FUNCTION OF REM SLEEP IS TO REDUCE THE VULNERABILITY CAUSED BY N-REM SLEEP

Before addressing the conceptual and factual foundations of the sentinel sleep theory, I must briefly discuss emotions and their importance in biological regulation. This context is crucial for a better understanding of the theory.

The central function of neurons and the brain composed of them is to assist the body in the intricate task of managing life (i.e., of administering the organism's survival) (Damasio, 2003, pp. 30, 194; Damasio, 2012, pp. 41, 64, 67; Moyne et al., 2022). In organisms equipped with a nervous system (which allows the body and any changes occurring within it to be mapped by the central nervous system), the most biologically valuable processes operating (automatically) to ensure the organism's life are *emotions* (Damasio, 2003, p. 34; Damasio, 2012, pp. 95-101; Damasio, 2019, pp. 56-65; Moyne et al., 2022; Wolpert, 2008). Some stimuli (whether from other animals, objects, or situations) can automatically trigger an emotional reaction. This is why many neuroscientists and psychologists describe them as *emotionally competent stimuli* or, equivalently, that they possess *emotional competence* (Caeiro et al., 2017; Clark et al., 2020; Damasio, 2003, p. 53; Kandel et al., 2013, p. 1079).

In short, emotions are the integration of all the automatic processes (many of which are independent of each other) involved in life regulation, and acquired over evolution (Damasio, 2012, pp. 55, 116; Damasio, 2015, p. 51; Kandel et al., 2013, p. 1079). These processes—which basically consist of complex sets of neural and chemical responses—are triggered whenever the brain receives an emotionally competent stimulus. The presence (real or recalled) of this biologically relevant stimulus (dangerous or valuable), from the internal or external environment, triggers automatic emotional responses (Damasio, 2003, p. 53; Damasio, 2015, p. 53; Kandel et al., 2013, p. 1079; Wolpert, 2008). These responses immediately result in altering—momentarily—the state of both the organism's body and the neural structures that map the body. Ultimately, emotional responses serve to place the organism—indirectly or directly—in a circumstance favorable to its self-preservation, survival, and well-being (Damasio, 2003, pp. 35, 53; Damasio, 2015, pp. 51-53; Gazzaniga et al., 2016, p. 416).

If the primary function of REM sleep is to provide the brain with a higher level of alertness to the immediately surrounding environment, contributing to the organism's survival, it is evident that there must be significant activation of neural regions involved in attention, threat detection, and emotional processing. And this activation must occur even if it lacks an obvious sense in this context (such as the primary visual cortex, as I will detail further). Before addressing neural activations that make sense, I will start by discussing the most obvious example of activation that (only superficially, as I will soon demonstrate) seems senseless in the context of sleep.

The primary visual cortex shows intense neural activation during REM sleep, similar to what occurs during the waking state (Bear et al., 2016, p. 670; Eagleman and Vaughn, 2021; Ribeiro, 2020, p. 136). The occipital lobes are almost exclusively dedicated to the sense of vision. The most prominent area of the occipital lobes is the *primary visual cortex*, whose function is to receive visual information from the eyes (Gazzaniga et al., 2016, p. 96). Considering that closed eyes during sleep prevent any visual input, what is the purpose of keeping the visual cortex active? This question led Eagleman and Vaughn (2021) to propose the hypothesis that the function of REM sleep is to activate the visual cortex to prevent neighboring neural regions from taking control of it. From the perspective of sentinel sleep theory, the reason why the visual cortex is intensely activated during REM sleep

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(analogous to activation during wakefulness) is that the eye is an obvious way to detect distant threats.

As a remote sensing "technology," the eye holds high survival value (Dawkins, 1997, p. 138; Mayr, 2004, p. 214). The adaptive solution we happen to call the "eye" provides the organism with the possibility of remote sensitivity. Instead of being forced to make physical contact with surrounding elements, an organism with vision can, for example, perceive a predator before colliding with it while being chased (Dawkins, 1997, p. 138).

Considering the high importance of the eye—during wakefulness—as a radar for threats, the intense activation of regions related to visual processing during REM sleep supports the sentinel function of REM sleep. It is due to the sentinel function that it makes sense for these regions to be substantially active during this sleep state. *The sentinel function also explains part of why rapid eye movements occur during REM sleep*. It is obvious to us, as conscious observers, that this activation is senseless. Closed eyes do not see and, therefore, are incapable of detecting threats. However, the automatic processes that regulate REM sleep are not conscious agents—nor are the evolutionary processes that shaped them. They are unaware that, although vision is excellent for perceiving threats during wakefulness, it does not operate during the organism's sleep.

In short, due to the protective function of REM sleep (providing greater alertness to the surrounding environment), the occipital cortex (due to its importance as a remote threat detector during wakefulness) ends up being substantially activated during this sleep state. I demonstrated that, according to sentinel sleep theory, the activation of regions involved in visual processing only superficially appears to be senseless. In general terms, any regions particularly responsible for attention and detecting dangerous stimuli play a fundamental role in REM sleep. It is due to their importance for survival that these regions are activated during REM sleep.

Now that I addressed this example of neural activation that superficially appears to be senseless, I will address the activation of brain structures that manifestly make sense from the perspective of REM sleep's protective function. One of them is the cingulate cortex—a structure that is part of the limbic system. REM sleep, like many attention paradigms, is positively correlated with increased activity in the cingulate cortex (Damasio, 2015, p. 212; Devinsky et al., 1995; Maquet et al., 1996; Paus et al., 1997; Schneider et al., 2020; Wu et al., 2017). The cingulate cortex plays a crucial role in processes associated with attention, emotional processing, autonomic and endocrine responses to emotions, and consciousness (Damasio, 2003, p. 59; Damasio, 2015, p. 212; Jumah and Dossani, 2022; Kandel et al., 2013, pp. 342, 495; Rolls, 2019).

The distinct subregions of the cingulate cortex and its extensive number of somatosensory input signals make it capable of potentially engendering the most integrated perception of the current state of the entire body of the organism at any moment; it is a center that integrates emotions, sensations, and actions (Damasio, 2003, p. 96; Damasio, 2015, p. 213; Jumah and Dossani, 2022). Therefore, it is not surprising that the anterior cingulate cortex is crucially involved in processing emotional states related to pain perception (Kandel et al., 2013, p. 545; Xiao and Zhang, 2018; Xiao et al., 2021). The fact that the anterior cingulate cortex plays a crucial role in pain processing is particularly relevant to my discussion. After all, physiological pain encloses a protective function (Xiao et al., 2021). Therefore, considering the protective function of REM sleep, it is crucial (and expected) that regions processing pain are activated during this sleep period.

Given that the cingulate cortex receives signals from major sensory portals, it is possible that it contributes to generating a neural pattern that maps, according to the appropriate causal sequence, the relationship between the appearance of a stimulus and the changes occurring in the body in response to it (Damasio, 2015, pp. 213-214). Upon being perceived, a stimulus can be easily communicated to the cingulate cortex via signals from the thalamus and direct signals from higher-order cortices in the lateral parietal, temporopolar, and inferotemporal regions (Damasio, 2015, pp. 213-214).

These characteristics make the cingulate cortex highly appropriate for the protective function exercised by REM sleep. The integrated perception of the body's state enabled by the cingulate cortex, as well as the pain processing carried out by this neural region, are very useful in the context of REM

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sleep. Since N-REM sleep is a state of high vulnerability, the increased neural activation of the cingulate cortex during REM sleep allows the brain to better analyze the organism's current state. Therefore, this structure crucially contributes to the protective role played by REM sleep.

Another brain structure whose activation makes sense from the perspective of the protective function of REM sleep is the amygdala. After all, it is a fundamental structure for detecting threats and triggering physiological and behavioral responses to danger. Additionally, it also plays a crucial role in both emotional processing and the regulation of the arousal state (Peever and Fuller, 2017; Purves et al., 2004, p. 687; Tang et al., 2005). Finally, the amygdala is so important for vigilance and attention that, when electrically stimulated in certain areas, it puts the brain into an even more intense state of vigilance and attention (Bear et al., 2016, p. 633; Davis and Whalen, 2001; Deboer et al., 1998).

Considering all these facts, as well as the fundamental role of emotions as the managers of life (Damasio, 2003, pp. 30-34; Damasio, 2012, pp. 41, 64, 67, 95-101; Damasio, 2019, pp. 56-65), and that arousal refers to the condition in which the organism is alert to the surrounding environment (Lee et al., 2022), it is entirely appropriate that the amygdala is involved (and with a prominent role) in REM sleep. Therefore, it is not surprising that the amygdala plays an important role in the regulation of REM sleep (Tang et al., 2005). In fact, I predict (based on the sentinel sleep theory) that the amygdala plays a central role in REM sleep, being one of the main structures that regulate this sleep state. Based on this theory, a strong correlation between REM sleep and the intense activation of the amygdala is expected.

Evidence supports this prediction: the amygdala is much more intensely activated during REM sleep than during wakefulness (Bear et al., 2016, p. 670; Corsi-Cabrera et al., 2016; Dang-Vu et al., 2010; Maquet et al., 1996; Nofzinger et al., 1997). The central importance of the amygdala to REM sleep is also evident when we analyze what happens when this structure is inhibited. Tetrodotoxin (a potent neurotoxin) can temporarily inhibit the action of neurons and tracts. When applied to the central nucleus of the amygdala, tetrodotoxin inhibits it. The consequences of this are revealing: a significant reduction in REM sleep duration and the number of REM episodes (Sanford et al., 2006; Tang et al., 2005). A scrutiny of the functions of the amygdala will allow me to demonstrate more clearly why it plays a central role in REM sleep.

The amygdala plays a crucial role—during wakefulness—in assessing the valence of received stimuli and, if negative, triggering the appropriate responses to ensure the organism's survival (Damasio, 2003, p. 58; Gazzaniga et al., 2016, p. 95; Pignatelli and Beyeler, 2019). The amygdala is particularly relevant to survival because it performs the function of receiving and learning about biologically relevant stimuli, especially emotionally competent stimuli with negative valence—exactly those crucial for survival. This is why activity in amygdala is more closely associated with the emotion of fear (Bear et al., 2016, pp. 626, 633; Damasio, 2003, p. 60; Gazzaniga et al., 2016, p. 95; Kandel et al., 2013, pp. 17, 1085; Pignatelli and Beyeler, 2019; Sah et al., 2003; Whalen et al., 2013). Part of the amygdala's function is to associate an external stimulus with its consequence for the organism, whether that consequence is positive (a reward) or negative (a punishment), encompassing all gradations between these extremes. Putting it another way, the amygdala also serves to assign valence (a biological value) to received sensory stimuli (Kandel et al., 2013, pp. 626, 1084; Pignatelli and Beyeler, 2019; Sah et al., 2003; Šimić et al., 2021).

Due to its sparse connections with cortical areas, the amygdala can influence the action of other neural regions; this is equivalent to saying that it can influence the action of other cognitive functions (e.g., modulate attention and perception) (Kandel et al., 2013, p. 1085). When the amygdala receives an emotionally competent stimulus (e.g., through neural projections from visual cortices), this stimulus is analyzed for its valence to determine the presence or absence of danger. If the valence of the stimulus is negative (i.e., if it consists of a threatening stimulus), the amygdala is activated. When this happens, it triggers the appropriate cascade of physiological and behavioral reactions (e.g., changes in heart rate, respiratory rate, pupil dilation, cutaneous blood flow, sweating, and facial muscle movements). It can accomplish all this by signaling to other neural regions (e.g., brainstem, hypothalamus, cingulate cortex, somatosensory cortices, and monoaminergic nuclei) and to the body (e.g., endocrine glands, viscera, and musculoskeletal system). This set of reactions is what we happen

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to call *emotions* (Asahina et al., 2003; Damasio, 2003, p. 58; Damasio, 2012, p. 119; Damasio, 2015, pp. 63-65; Gazzaniga et al., 2016, pp. 95, 404; Kandel et al., 2013, pp. 349, 1085, 1079; Ootsuka and Tanaka, 2015; Purves et al., 2004, p. 687; Whalen et al., 2013).

Physiological and behavioral reactions triggered by the amygdala serve the purpose of safeguarding the organism (Gazzaniga et al., 2016, pp. 95, 404; Kandel et al., 2013, p. 1085; Whalen et al., 2013). Therefore, it is particularly relevant that information from all sensory portals is projected to the amygdala, with each sensory portal having a distinct projection pattern. It is the interconnections within the amygdala that allow information from different sensory portals to be integrated (Bear et al., 2016, p. 632). All these characteristics of the amygdala (which I discussed in this paragraph and in the preceding ones) make it highly suitable for the protective function performed by REM sleep. It is thus not surprising that the amygdala plays a central role in the regulation of REM sleep. The sentinel function of REM sleep allows me to easily explain both the intense activation of the cortical amygdala during this sleep state and its distinctive regulatory role.

To prevent any careless scientist from misinterpreting my arguments, I want to emphasize the following. It might seem that I am employing circular reasoning when I claim, for example, that the distinctive activation of the amygdala during REM sleep corroborates the sentinel function of REM sleep. As if I were using the premise of the sentinel function of REM sleep to conclude that the amygdala being active during REM sleep corroborates the sentinel function. This would be a serious misinterpretation of my arguments. What I am actually using as a premise is the well-known fact that the amygdala performs a protective function *during wakefulness*. Consequently, its distinctive activation during REM sleep corroborates the sentinel function of REM sleep. There is no circularity here. And the same applies to the arguments I developed regarding the activation of the cingulate cortex and other neural regions during REM sleep.

In summary, for the sentinel function of REM sleep to be performed, it is necessary that the regions responsible—during wakefulness—for attention, vigilance, and emotional processing be activated during REM sleep. It is already well-documented in the scientific literature that limbic structures exhibit high neural activation during REM sleep (Caska et al., 2009; Peterson et al., 2002). Through Positron Emission Tomography (PET), Statistical Parametric Mapping (SPM), and neuroimaging studies, scientists demonstrated that numerous regions of the limbic system—emotion-related regions—are differentially active during REM sleep. The cingulate cortex (especially the anterior region), both amygdaloid complexes, the hippocampal formation, the striatum, and the left thalamus experience an increase in both blood flow and electroencephalographic activity during REM sleep (Braun et al., 1997; Goldstein and Walker, 2014; Maquet et al., 1996; Maquet, 2000).

Moreover, not only does the limbic system become prominently more active during the REM period. The paralimbic structures also exhibit high neural activation during this sleep period (Braun et al., 1997). The amygdalofugal pathways to the right parietal operculum, thalamic nuclei, entorhinal cortex, dorsal midbrain, pontine tegmentum, and anteroinferior portions of the insula are also notably activated during the REM period (Braun et al., 1997; Braun et al., 1998; Goldstein and Walker, 2014; Peterson et al., 2002). This heightened activation of the limbic system during REM sleep—the set of neural regions involved in emotional processing—as well as the paralimbic structures (also involved in emotion), is precisely what is predicted by the sentinel function of REM sleep.

A brief digression. The higher-order neural regions involved in emotional processing have traditionally been grouped under the label *limbic system* (Purves et al., 2004, p. 687; Sagan, 1978, p. 66). Despite the term "limbic system" still being widely used in discussions concerning the neural mechanisms responsible for emotions, it is important to note that there is no single emotional system (Bear et al., 2016, p. 625). Some neural structures undoubtedly involved in emotional processing (e.g., the anterior cingulate cortex, the amygdala, and the insula) also have other functions (Bear et al., 2016, p. 625; Šimić et al., 2021). In this case, therefore, there is no one-to-one correspondence between a neural region and a function (Bear et al., 2016, p. 625; Kandel et al., 2021, p. 1047).

Indeed, given the high biological value of emotions, any evolutionary biologist can easily perceive how the evolutionary strategy of a one-to-one correspondence between a neural region (or system) and an emotional function would, in all likelihood, be eliminated. After all, it is not an

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Evolutionarily Stable Strategy (ESS). It is biologically advantageous for emotional processing to be divided among various regions. This way, when one of them is compromised, the others can still perform the task.

I want to briefly highlight the thalamus. Functional neuroimaging demonstrates that during the N-REM period, the thalamus is inactivated. During the REM period, however, the thalamic nuclei are activated (Jan et al., 2009; Maquet, 2000). Among other functions, the thalamus is involved in attention and alertness (Perea Bartolomé and Ladera Fernández, 2004; Torrico and Munakomi, 2023; Tuttle et al., 2019). Damage to higher-order thalamic regions—such as the mediodorsal nucleus and the pulvinar nucleus—can result in severe attention deficits (Saalmann and Kastner, 2015).

To give an example: in the study by Exner and colleagues (2001), the scientists assessed certain cognitive aspects in patients with thalamic lesions. They compared three groups. One group (n = 15) with individuals who had focal thalamic infarction or hemorrhage was compared with two control groups: one (n = 15) with healthy individuals (i.e., without thalamic damage) and the other (n = 22) with individuals who had basal ganglia lesions. Exner and colleagues reported that individuals with thalamic lesions exhibited well-preserved intellectual and executive functions. However, among other findings, the scientists reported deficits in attention measures and psychomotor speed.

To continue with the factual foundation of the sentinel sleep theory, I will now analyze unihemispheric sleep. As will become evident, the fact that REM sleep almost *never* occurs in a brain undergoing unihemispheric sleep strongly supports my arguments about the protective function of REM sleep.

For certain animals, the environmental pressure against the brain being subjected to sleep in both cerebral hemispheres is so substantial that they ended up developing, through non-random elimination, unihemispheric sleep (Bear et al., 2016, p. 663; Purves et al., 2004, p. 661; Ribeiro, 2020, p. 132). Their brains can sleep using only one cerebral hemisphere at a time (Mascetti, 2016; Ribeiro, 2020, p. 131). In certain environments and niches, if the organism's brain were subjected to deep sleep in both hemispheres, the organism would face serious problems. Its survival would be severely compromised—either due to heightened vulnerability caused by the low levels of alertness characteristic of N-REM sleep or due to the need to maintain movement (Mascetti, 2016; Ribeiro, 2020, pp. 132-133).

Unihemispheric sleep allows only one hemisphere to undergo much-needed N-REM sleep. Putting it another way, unihemispheric sleep prevents both hemispheres from becoming significantly more inactive and, consequently, prevents the organism from becoming significantly more vulnerable (Kandel et al., 2013, p. 1141; Mascetti, 2016; Ribeiro, 2020, pp. 132-133). In unihemispheric sleep, the neural mechanisms involved in promoting the waking state predominate in one cerebral hemisphere (as indicated by desynchronized electroencephalographic activity with high-frequency and low-amplitude waves), while the neural mechanisms involved in promoting the deep sleep state predominate in the other (as indicated by low-frequency and high-amplitude waves) (Konadhode et al., 2016; Mascetti, 2016; Ribeiro, 2020, p. 137). Due to this evolutionary strategy, one hemisphere can lower its alertness (an imperative characteristic of N-REM sleep) while the other hemisphere ensures that vigilance and attention to the surrounding environment are maintained—preventing the organism from being subjected to substantial vulnerability.

For cetaceans (e.g., dolphins, belugas, orcas, porpoises, and whales), unihemispheric sleep constitutes the only form of sleep (Mascetti, 2016; Ribeiro, 2020, p. 131). This characteristic allows cetaceans to maintain constant movement, ensuring periodic surfacing for breathing (Mascetti, 2016; Ribeiro, 2020, p. 131). Repeated studies on cetaceans failed to find any amount of REM sleep in these animals (Lyamin et al., 2008; Lyamin et al., 2018). The fact that cetaceans lack REM sleep has been interpreted as evidence that the need for REM sleep is overridden if the brain maintains, in one of its hemispheres, elevated levels of electrical activity capable of sustaining continuous motor activity and a high level of alertness (Ribeiro, 2020, pp. 131-132). I argue, based on my theory, that this observation is entirely correct.

Since the function of REM sleep is to provide greater defense to the organism during the vulnerable N-REM sleep, its absence in cetaceans is further evidence in support of the sentinel sleep

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theory. After all, with the unilateral occurrence of N-REM sleep in these animals, there is sufficient neural activation to ensure consistent defense against any threats in the surrounding environment, making REM sleep unnecessary.

Unlike cetaceans, other animals have both unihemispheric and bihemispheric sleep. Birds are examples of this. A relevant fact for my discussion is that REM sleep occurs in them only when the brain is subjected to bilateral N-REM sleep: in birds, REM sleep is absent whenever unihemispheric sleep occurs (Mascetti, 2016; Rattenborg et al., 1999a; Rattenborg et al., 1999b; Ribeiro, 2020, p. 131). The same explanation I presented for cetaceans in the previous paragraph applies to birds. When a bird's brain is subjected to unihemispheric sleep, there is sufficient neural activation to ensure environmental vigilance. However, the same does not happen when the brain is subjected to bihemispheric N-REM sleep. That is why REM sleep is present in birds when they undergo bihemispheric N-REM sleep. I will discuss henceforth about another animal that has both bihemispheric and unihemispheric sleep.

The northern fur seal (*Callorhinus ursinus*) is a semiaquatic mammal: it can sleep both in seawater (where it spends most of its life) and on land (Lyamin et al., 2018). Lyamin and colleagues (2018) demonstrated that when the studied fur seals slept in water, REM sleep was either effectively suppressed or significantly reduced: from 80 minutes (when on a dry platform) to 3 minutes per day (when in water); a reduction of 96.4%. During the first three to seven days in water, no REM sleep was recorded in any of the fur seals; in one of the four fur seals, REM sleep occurred on only one of the eleven days of analysis. After undergoing this almost complete suppression of REM sleep and returning to sleep on the dry platform, the fur seals either exhibited minimal REM sleep rebound or no rebound at all. When the fur seals left the dry platform and returned to the water, bihemispheric sleep was replaced by unihemispheric sleep. While in seawater, their N-REM sleep was predominantly unihemispheric (94% of all N-REM sleep was unihemispheric in this condition). In comparison, when on the dry platform, unihemispheric N-REM sleep was reduced (61% of all N-REM sleep was unihemispheric in this condition). And again (as with birds), unihemispheric N-REM sleep was associated with the absence of REM sleep.

From the perspective of the sentinel sleep theory, the reason the fur seals did not exhibit REM sleep rebound (or exhibited minimal rebound) is due to the biological function of REM sleep. Since their brains were predominantly subjected to unihemispheric sleep while they remained in water, the fur seals were sufficiently protected. Their brains were sufficiently vigilant to the surrounding environment. Thus, REM sleep was dispensable. As I already stated, REM sleep is necessary only when N-REM sleep occurs in both hemispheres.

Regarding the minimal REM sleep rebound observed, it may be due to the following reason. The fur seals clearly enclose neural mechanisms that control REM sleep suppression, activated whenever unihemispheric sleep occurs. As I will elaborate further, REM sleep rebound constitutes a defense mechanism triggered whenever REM sleep is suppressed. It turns out that in this case there is conflicting information. On one hand, whenever unihemispheric N-REM sleep occurs, the organism is protected, making REM sleep dispensable. On the other hand, whenever unihemispheric N-REM sleep occurs, REM sleep is suppressed, making REM sleep rebound necessary. Therefore, the reason behind the minimal rebound observed may simply be because REM sleep was suppressed when the organism’s brain was subjected to unihemispheric N-REM sleep. However, since the organism was sufficiently protected by being subjected to N-REM sleep in only one hemisphere, the rebound was minimal instead of lasting as long as the suppression occurred. We must consider that non-random elimination may not have had time to eliminate this rebound when it makes no sense to have it. Therefore, it is expected that many animals will present minimal rebound even after their brain is subjected to unihemispheric N-REM sleep.

Another fact that corroborates my theory is the way organisms respond when awakened from REM sleep. When an organism (human or non-human) is awakened from REM sleep, it exhibits full alertness (an obvious adaptive advantage) (Kandel et al., 2013, p. 1157; Ribeiro, 2020, p. 127). The biological relevance of the REM period is evident from the fact that animals, when awakened during this period, respond more effectively and demonstrate better sensory and motor function compared

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to those awakened from N-REM sleep—who exhibit sensory, cognitive, and motor deficits that take several minutes to dissipate (Kandel et al., 2013, p. 1157; Ribeiro, 2020, p. 127). Additionally, another relevant fact is the habitual occurrence of spontaneous awakenings during or immediately after REM sleep; which led scientists to believe that the REM period serves to facilitate the transition from N-REM sleep to wakefulness (Ermann et al., 1993; Ficca et al., 2004; Klemm, 2011; Ribeiro, 2020, p. 127).

The aforementioned evidence clearly corroborates the sentinel sleep theory. If (as I am arguing) the biological function of the REM period is to reduce the vulnerability of N-REM sleep—especially through greater neural activation in regions related to vigilance and emotional processing—the heightened readiness demonstrated by organisms awakened from REM sleep is precisely what would be expected. This readiness is a consequence of the sentinel function of REM sleep.

To conclude this section, I will discuss what happens when REM sleep is suppressed. Organisms that undergo total REM sleep deprivation experience a vigorous compensatory return known as *REM sleep rebound*. This rebound is characterized by a subsequent increase in both the time the brain invests in the REM period and the intensity of this period, leading to more intense intrusive dreams (Kandel et al., 2013, p. 1157; Ribeiro, 2020, p. 77). REM sleep rebound is proportional to the duration of its suppression, but (which is particularly relevant to my arguments) the opposite is not true. Increasing N-REM sleep time also increases REM sleep, but it does not cause a subsequent "negative rebound" (Ribeiro, 2020, p. 169). The reason this negative rebound does not occur is obvious from the perspective of the sentinel sleep theory: doing so would compromise vigilance during sleep and, consequently, the organism's safety.

REM sleep rebound is due to its sentinel function. This biological mechanism that provides greater protection during sleep—the REM period—proved to be so fundamental throughout evolution that it is present in a vast number of distinct species. Due to its biological value, major or total suppression of this protective mechanism represents an abrupt increase in the organism's vulnerability during N-REM sleep. When the brain is subjected to major or total suppression of the REM period, it activates a defense mechanism: REM sleep rebound. If REM sleep is suppressed, the brain demands a subsequent compensatory investment in REM sleep to offset the heightened vulnerability it was exposed to during REM sleep suppression. *The evolutionary pressure to develop a protective sleep was so high that even this protective sleep has a protective mechanism: REM sleep rebound*. (See sections 2.3 and 2.5 for a deeper discussion of this evolutionary pressure.)

The sentinel function of REM sleep explains why its suppression (partial or total) does not result in neural or cognitive impairments for the organism. Contrary to what is claimed by many scientists (Bear et al., 2016, p. 665; Gazzaniga et al., 2016, pp. 150-151; Moruzzi and Eccles, 1966; Ribeiro, 2020, p. 130), the primary function of REM sleep is *not* to contribute to learning, but rather to provide greater protection to the highly vulnerable N-REM sleep. (See section 5, where I justify this assertion.)

This is why patients medicated with antidepressants can exhibit near-complete or complete REM sleep inhibition for years—an effect caused by practically all antidepressants (with some even interfering with the homeostatic regulation of REM sleep)—without showing any notable deficits in learning and the capacity to form new memories, while maintaining normal brain functionality (Bear et al., 2016, p. 665; Feriante and Araujo, 2023; Kandel et al., 2013, p. 1157; Matsuda et al., 2021; McCarthy et al., 2016; Nollet et al., 2019; Pagel and Parnes, 2001; Ribeiro, 2020, p. 171).

The greatest harm that REM sleep inhibition causes to an organism is the substantial increase in its vulnerability during sleep. Therefore, if you are in a safe place while sleeping, you do not have to worry about neural impairments if your REM sleep is suppressed. REM sleep suppression does not compromise any neural function other than the protective function it provides. Note that the preceding statements refer *exclusively* to REM sleep suppression. It is crucial to distinguish between the effects of exclusive REM sleep suppression and REM sleep suppression accompanied by N-REM sleep suppression. We must consider this distinction because it is common for scientists to also suppress N-REM sleep when studying REM sleep suppression (Lyamin et al., 2008).

2.2. THE PARAMETERS OF REM SLEEP DEPEND ON THE ORGANISM'S VULNERABILITY

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Although insufficient on its own, what usually serves as evidence to support an assertion concerning a cause is a correlation between two events (Carnielli and Epstein, 2019, p. 277; Weston, 2009, p. 41). Moreover, when one aims to demonstrate that *A* causes *B*, one also aims to demonstrate that it makes sense for *A* to cause *B*. The better the connection (or explanation) established between the cause and the effect, the stronger the argument will be (Weston, 2009, pp. 43-44). This is what I will attempt to do to demonstrate that increasing bodily protection causes specific and predictable changes in REM sleep parameters.

An obvious prediction of the sentinel sleep theory is that the brain of organisms with greater body fat or muscle strength will spend less time in the REM period. After all, greater weight or muscle strength leaves the organism more protected compared to its peers lacking this protection. An organism with lower body weight or lower muscle strength is more vulnerable compared to another organism of the same species with greater weight or muscle strength. This is why increasing muscle strength or weight should be accompanied by a reduction in the time the brain invests in REM sleep.

Furthermore, a longer latency to the first REM period is also predicted. In less vulnerable organisms—either due to a greater amount of body mass or greater muscle strength—the onset of the first REM period can delay beyond the usual time. Since the organism is better protected, more time can be dedicated to the fundamental N-REM sleep before transitioning to the sentinel stage. Here is the empirical basis for the preceding statements:

In the article by Driver and Taylor (2000), the authors reviewed the literature regarding the association between exercise and sleep. Using meta-analytic techniques, the authors demonstrated that long-duration, high-intensity exercises altered participants' sleep: N-REM sleep time increased (by 2 to 5 minutes), REM sleep time decreased (by 2 to 5 minutes), and REM sleep latency increased (by 10 minutes).

In the study by Myllymäki and colleagues (2011), the researchers analyzed the effects of intense exercise on sleep when performed within a period of three hours before bedtime. To do so, they evaluated a group of young adults ($n = 11$), monitored in two distinct situations: (1) after engaging in intense exercise before bed, and (2) after a day without exercise (control situation). Among other results, the researchers reported that REM sleep had an average duration of 88 minutes in the exercise situation and an average duration of 101.3 minutes in the no-exercise situation ($p = 0.155$).

From the perspective of sentinel sleep theory, the above-mentioned result indicates that engaging in high-intensity exercise—even for just one day and within three hours before sleep—is enough to virtually reduce the organism's vulnerability. The mere fact of exercising intensely before sleep causes the neural mechanisms that regulate REM sleep to interpret this action as a—small but significant—increase in the organism's protection. This supports my claim that proprioceptive information is particularly relevant to REM sleep parameters (e.g., to the time invested in it).

In the study by Driver and colleagues (1994), to evaluate whether the duration of physical exercise affects sleep immediately after practice, the researchers analyzed the sleep of a group of male endurance athletes ($n = 8$; age range: 23-42 years). Four distinct exercise conditions were analyzed: (1) a day without any specific exercise, (2) a day with a 15 km run, (3) a day with a 42.2 km run, and (4) a day when participants performed an exhaustive ultra-triathlon. The sleep parameters in the first three conditions were analogous. However, they were different in the fourth condition. Compared to the first three, the exercises in the fourth condition significantly increased wakefulness, reduced REM sleep time, and increased REM sleep latency. Here are some of the results presented: in the no-exercise condition, the average REM sleep latency was 90 minutes; in the ultra-triathlon condition, the average REM sleep latency was 186 minutes. In the no-exercise condition, the average amount of REM sleep was 72 minutes; in the ultra-triathlon condition, the average amount of REM sleep was 27 minutes.

In the study by Kitamura and colleagues (2021), the researchers evaluated the relationship between sleep parameters and body composition by comparing female athletes ($n = 19$) and male athletes ($n = 17$). Compared to the female athletes, the male athletes had greater muscle mass (82.4% muscle mass in men, 77.9% in women; $p = 0.008$). Among other results, male athletes spent less time in REM sleep compared to female athletes (21.1% in men, 26.2% in women; $p = 0.008$). This difference

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is exactly what would be expected due to the sentinel function. Since the men had greater muscle mass than the women, this made them more protected, thus requiring less time in REM sleep.

In the study by Hrozanova and colleagues (2020), to investigate the association between training load, mental stress, and sleep, the researchers recruited a group of junior athletes from schools specializing in endurance sports practices (n = 56; age range: 17-19 years). They reported that both mental stress and training load were associated with a subsequent significant reduction in REM sleep. This reduction was more intense when associated with an increase in training load. For each point added to the training load scoring scale, REM sleep significantly decreased. On the other hand, decreasing the training load increased the total time spent in both N-REM and REM sleep. Additionally, for each point added to the mental effort scoring scale, total sleep time, sleep efficiency, time spent in N1 and N2 stages, and REM sleep time also significantly decreased.

In the study by Hague and colleagues (2003), the researchers aimed to test how exercise impacts sleep. To do so, they chose to significantly reduce the intensity of physical activity in highly active individuals; which is why they recruited a group of trained athletes (n = 15) who maintained a daily exercise routine with varying intensity from moderate to high. The researchers evaluated two conditions: (1) a day when the participants did not perform their usual physical activities, and (2) a day when they did (control condition). Among other results, they observed that in the sedentary condition, REM sleep latency was reduced by an average of 24.0 minutes ($p < 0.05$), and REM sleep time increased by an average of 17.9 minutes ($p < 0.05$). When comparing the experienced conditions, they found no overall impact on total sleep time, sleep efficiency, or wakefulness after sleep onset ($p < 0.05$).

In the study by Chamorro and colleagues (2014), the researchers aimed to analyze the relationship between sleep parameters and being overweight. To do this, they recruited healthy 10-year-old children based on two groups: (1) normal weight (n = 37), and (2) overweight (n = 59). In the normal weight group, the average REM sleep time was 87.3 minutes (p = 0.05). In the overweight group, the average REM sleep time was 75.6 minutes (p = 0.05). In the normal weight group, the average REM sleep latency was 120.0 minutes (p = 0.05). In the overweight group, the average REM sleep latency was 138.7 minutes (p = 0.05). There was no statistically significant difference between the groups regarding the N3 stage (the deepest stage of N-REM sleep). The N3 values for the normal weight and overweight groups were, on average, 94.7 and 94.6 minutes, respectively.

The authors of the aforementioned study concluded that being overweight in childhood is associated with changes in total sleep duration, in N-REM sleep, and in REM sleep. Something particularly relevant to my theory is that *the amount by which REM sleep was reduced in overweight children was inversely proportional to body mass index*. In the study by Chamorro and colleagues (2014), the age of the children was almost identical in both groups, and sleep patterns were recorded under natural conditions. Therefore, as described by the researchers, these factors (which alter REM sleep) are unable to explain the discrepancy found in REM sleep between the two groups. This discrepancy is easily explained by the sentinel sleep theory.

In the study by Liu and colleagues (2008), the researchers analyzed the association between obesity and specific sleep stages in children and adolescents. The participants (n = 335; age range: 7-17 years) were divided into three groups: (1) non-overweight group (n = 241), (2) at-risk-of-overweight group (n = 49), and (3) overweight group (n = 45). Among other results, body mass was significantly related to REM sleep. Additionally, participants in the overweight group (compared to the non-overweight group) had longer REM sleep latency, lower REM sleep activity and density, and less REM sleep time. To determine which sleep stages (N1, N2, delta sleep, and REM sleep) were independently related to being overweight, the researchers conducted a multiple logistic regression analysis. The result was that only the reduction in REM sleep was independently and significantly related to being overweight (p = 0.03).

In the study by Theorell-Haglöw and colleagues (2010), the researchers evaluated the relationship between sleep stages, sleep duration, and central obesity in women. To do this, they selected a sample of women aged 20 to 70 years (n = 400) and divided them into two groups: (1) central obesity group (n = 182), and (2) non-central obesity group (n = 218). Based on the results, the

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researchers concluded that obesity was inversely proportional to the duration of both N-REM and REM sleep. In the obesity group, the average REM sleep time was 63.2 minutes ($p < 0.0001$); in the non-obesity group, the average REM sleep time was 77.5 minutes ($p < 0.0001$). The authors concluded that even after adjusting for confounding factors, the duration of both N-REM and REM sleep was inversely proportional to waist circumference and sagittal abdominal diameter.

In the study by Elrokhsi and colleagues (2020), the researchers investigated how different Body Mass Index (BMI) levels alter sleep. To do this, they selected a group of children with Type 1 Diabetes Mellitus (T1DM) ($n = 105$; average age: 13.54 years; 49.5% female). Of the total, 19% were obese, 22% were overweight, and 59% had a normal BMI (81% non-obese). As reported by the researchers, there was no statistically significant difference between obese and non-obese regarding N-REM sleep. However, compared to non-obese children, obese children had significantly less REM sleep time ($p = 0.022$); a statistical significance that remained even after the researchers adjusted the result for multiple covariates.

Considering the sentinel function of REM sleep, another prediction is that organisms exposed to an unknown environment (and therefore rich in sensory information) should show a significant increase in REM sleep time, as well as a shorter latency to the first REM episode. After all, *the unknown includes the possibility of danger*. This is equivalent to stating that an unfamiliar environment subjects the organism to greater vulnerability. As described by Kahneman (2011, p. 67):

To survive in a frequently dangerous world, an organism should react cautiously to a novel stimulus, with withdrawal and fear. Survival prospects are poor for an animal that is not suspicious of novelty. However, it is also adaptive for the initial caution to fade if the stimulus is actually safe.

When the organism rests in a familiar environment, the brain benefits from this familiarity, especially if the environment does not include (in recent experiences) a constant level of dangerousness. Under this condition, the brain can invest less time in the REM period and may even delay its onset slightly (longer latency). However, when the organism is in an unknown resting place, vigilance against any possible threats needs to be higher. This is why, whenever the organism is exposed to an unknown environment, the brain will invest more time in the REM period, and it will be more imperative that it does not delay its onset (shorter latency). *The possibility of danger demands a greater amount of REM sleep and a shorter latency to the first REM episode*.

It has been consistently demonstrated that exposing an animal to a rich sensory experience during wakefulness (e.g., being exposed to a new environment) significantly increases the time the brain invests in REM sleep and reduces REM sleep latency (in some cases, without altering total sleep time) (Borniger et al., 2018; Gutwein and Fishbein, 1980a; Gutwein and Fishbein, 1980b; Kiyono et al., 1981; Mirmiran et al., 1982; Nair et al., 2022; Smith, 1996; Tagney, 1973; van Gool and Mirmiran, 1986). This evidence corroborates the sentinel sleep theory and also implicates fear as an emotion capable of affecting REM sleep.

A not-so-obvious prediction of the sentinel sleep theory is that, besides body mass and muscle strength, any other factors that increase or decrease the organism's vulnerability will also affect REM sleep. After all, it is not only body mass and muscle strength that influence the organism's vulnerability: other factors can also make it more or less vulnerable. It is possible to extend the discussion beyond the obvious factors. This leads me to discuss stress and depression. I will start with depression.

A notable characteristic of depression is that it places the organism in a state of increased vulnerability—leaving it with low energy and greater fatigue (Arias et al., 2020; Gazzaniga et al., 2016, p. 620; Stahl, 2002; Targum and Fava, 2011; Wolpert, 2008). Therefore, according to the sentinel sleep theory, depression should cause the brain to invest more time in REM sleep, reduce the latency to the first REM episode, and increase the density (or intensity) of REM sleep. It may also cause the first REM episode to be longer. When the organism is more vulnerable (e.g., due to depression), the first REM episode may last longer precisely because of this vulnerability. Since N-REM sleep predominates at the beginning of sleep (Ribeiro, 2020, p. 143), this vulnerability combined with another vulnerability (e.g., depression) may result in a longer first REM episode. *When other factors remain unchanged, combined vulnerabilities produce more intense effects on REM sleep parameters*.

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All these predictions were consistently confirmed (although not under the context of my theory). Depressed patients exhibit a decrease in N-REM sleep, an increase in total REM sleep time, shorter REM sleep latency, a prolonged first REM episode, and greater intensity (or density) of REM sleep (especially in the first REM period) (Anderson and Bradley, 2013; Berger and Riemann, 1993; Gazzaniga et al., 2016, p. 620; Kishi et al., 2023; McCarley, 1982; Palagini et al., 2013; Riemann et al., 2020; Riemann and Berger, 1989; Schmid et al., 2008; Steiger et al., 2013; Steiger and Pawlowski, 2019; Suchecki et al., 2012; Wichniak et al., 2017). Now that I addressed depression, I will discuss stress.

Stress commonly impacts all body systems (e.g., cardiovascular, muscular, endocrine, nervous, respiratory, reproductive, and gastrointestinal systems). Regarding the cardiovascular system, acute stress increases heart rate, dilates the heart, intensifies heart muscle contractions, and reduces blood flow in organs that are not involved in rapid motor activity to redirect it to the large muscles—something particularly relevant in the context of fight or flight (Chu et al., 2022; Dhabhar, 2018).

Regarding the endocrine system, stress increases the production of hormones that activate the physiological responses to it—one of which is the cortisol (Chu et al., 2022). When the brain detects a stressful situation—whether recalled or actually present—it triggers a cascade of stress-related hormones that serve the purpose of preparing the body to fight or flee. This fight-or-flight response constitutes one of the primary survival mechanisms for an organism. Without this mechanism, a predator would be unable to capture its prey, and a prey would be unable to escape from its predator (Chand et al., 2021; Chu et al., 2022; Damasio, 2003, p. 53; Dhabhar, 2018). In short, the immediate result of stress is to favor, directly or indirectly, the survival of the organism.

A careless scientist might assume that it is incorrect for me to assert that stress makes the organism better protected (or less vulnerable). This scientist might argue that "as a prey, the stress during a fight-or-flight reaction indicates that I am being hunted, which is equivalent to saying that I am vulnerable." Thinking this way is incorrect. The vulnerability is due to the predator, not the stress. It is the stress that allows a prey to have some chance of successfully escaping from a predator. Without stress (and the other components of the fight-or-flight reaction), this would be impossible (Chand et al., 2021; Chu et al., 2022; Damasio, 2003, p. 53; Damasio, 2015, pp. 52-53; Dhabhar, 2018). Stress automatically provides a prey with an internal state whose purpose is to enable behavioral responses appropriate to the context of fleeing or fighting (e.g., increased heart rate, increased blood pressure, and increased blood flow directed to the arteries of large muscles), thereby increasing their chances of survival. Therefore, what is truly incorrect is to assert that stress does not contribute to reducing the organism's vulnerability.

Considering that stress (due to the physiological state that favors survival) reduces the organism's vulnerability, this implies that any organism under the influence of stress hormones will have its REM sleep affected. According to the sentinel sleep theory, stress should cause the brain to invest less time in REM sleep, increase the latency to the first REM episode, and increase the density of REM sleep. It may also cause the first REM episode to be (albeit subtly) shorter than the others. Given that N-REM sleep is highly important and that it predominates at the beginning of sleep (Ribeiro, 2020, p. 143), with the organism being better protected, the brain can dedicate less time to REM sleep and more time to N-REM sleep.

The reason it is expected that REM sleep density increases (rather than decreases) under the influence of stress is that stress leaves the organism prepared for a fight-or-flight response. This makes REM sleep more intense. Putting it another way, stress makes the organism more easily awakened during REM sleep because, among other effects, stress reduces the organism's vulnerability by increasing vigilance (Chand et al., 2021; Oken et al., 2006). Therefore, considering that both REM sleep and stress reduce the organism's vulnerability by increasing vigilance, the combination of both results in greater intensity of REM sleep. *Just as combined vulnerabilities produce more intense effects on REM sleep parameters, combined protections also do the same*.

Feinberg and colleagues (1987) already proposed that the density (or intensity) of REM sleep may be related to the level of arousal. Some data support this hypothesis (Barbato, 2023). Here I assert, based on the sentinel sleep theory, that REM sleep density is indeed directly related to arousal (or alertness, or vigilance, or attention). My argument is that REM sleep density is proportional to the

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level of alertness. In other words, REM sleep density equates to the organism's level of alertness. This implies that the organism will awaken if REM sleep density reaches a very high intensity (i.e., a threshold). I present henceforth some additional evidence that corroborates what I stated in this and the two preceding paragraphs.

When N-REM sleep predominates, cortisol levels reach their minimum; when REM sleep predominates, cortisol levels increase, approaching the cortisol levels associated with alertness during wakefulness—the peak is reached when the organism awakens (Ribeiro, 2020, pp. 143, 248). In the study by Feng and colleagues (2023), the REM sleep density in participants underwent a significant increase after being subjected to stressful situations; moreover, they were more likely to spontaneously awaken during sleep when under stress. In the research by Rodenbeck and Hajak (2001), the authors demonstrated that the number of spontaneous awakenings was correlated with cortisol levels. In the study by Barbato and colleagues (1994), the authors demonstrated that the propensity for spontaneous awakening is greater in REM sleep when it presents a high density. The same was demonstrated (especially in younger individuals) in the study by Ficca and colleagues (2004). In short, *most spontaneous awakenings are preceded by a high density of REM sleep* (Barbato, 2023).

Now that I demonstrated the evidence that corroborates my conclusion that REM sleep density equates to the brain's alertness level, I will present henceforth the evidence that corroborates my other assertions regarding the effect of stress on REM sleep.

Mental tension significantly reduces REM sleep time (Hrozanova et al., 2020). And acute cortisol administration in humans increases N-REM sleep, suppresses or substantially reduces REM sleep, and increases the latency of the first REM episode (Friess et al., 2004; Schmid et al., 2008). In rodents, stress induces a reduction in both N-REM and REM sleep, with the amount of reduction varying according to the type of stress experienced and the duration of exposure to it (Papale et al., 2005; Rolls et al., 2010). In humans, stress reduces both N-REM and REM sleep, increases REM sleep latency, and increases REM sleep density (Feng et al., 2023).

In the study by Gonnissen and colleagues (2013), the researchers analyzed the effects of sleep fragmentation. To do it, they recruited a group of healthy male participants (n = 12). Two conditions were compared: (1) a day without sleep fragmentation and (2) a day with sleep fragmentation. In the non-fragmented sleep condition, the average REM sleep time was 83.5 minutes, while in the fragmented sleep condition it was 69.4 minutes: a statistically significant reduction ($p > 0.05$). There was no statistical significance between conditions regarding N-REM sleep latency, wake time, total sleep time, and total time in stage N1. The total sleep time did not change significantly because the reduced REM sleep time was equivalent to the increased time in stage N2. Something particularly relevant is that nighttime cortisol levels were significantly higher in the fragmented sleep condition compared to the non-fragmented condition. Based on my theory, I assert that—given that sleep fragmentation elevates cortisol levels (Gonnissen et al., 2013; Rodenbeck and Hajak, 2001)—stress due to fragmentation reduced REM sleep.

The study by Schmid and colleagues (2008) is interesting because the researchers attempted to replicate—in depressed participants—the widely reported suppression of REM sleep as a result of acute cortisol administration. As the researchers stated, they were unable to do so. The reason, from the perspective of sentinel sleep theory, is simple. Considering that this sample included depressed participants, the presence of this disorder prevented the suppression of REM sleep. *Depression, due to the vulnerability it imposes on the organism, prevents REM sleep from being suppressed, even under acute cortisol administration*.

In summary, the time invested in REM sleep is inversely proportional to muscle strength and body weight, but directly proportional to vulnerability. Increasing vulnerability causes the brain to invest more time in REM period, reduce the latency to the first REM episode, and increase REM sleep intensity; reducing vulnerability causes the brain to invest less time in REM period, increase the latency to the first REM episode, and reduce REM sleep intensity (except when the organism is under the influence of stress hormones).

Whenever the organism is less vulnerable, REM sleep is significantly reduced, allowing the brain to dedicate more time to the essential N-REM sleep. This is why a reduction in total REM sleep time

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may, in some cases, be accompanied by an increase in total N-REM sleep time. However, a marked reduction in REM sleep may also be accompanied by no change in N-REM sleep time. In general, a reduction in REM sleep is accompanied by a reduction in total sleep time. After all, reducing REM sleep time naturally affects total sleep duration.

Analyzing REM sleep in neonates will allow me to further corroborate the arguments I developed for the sentinel function of REM sleep. Therefore, I will dedicate the next few paragraphs to this analysis.

As described by Ribeiro (2020, p. 129), the amount of REM sleep is strongly correlated with the level of physical immaturity at birth. Animals that exhibit high autonomy shortly after birth (e.g., sheep, guinea pigs, and giraffes) have a lower amount of REM sleep: about one hour per day. On the other hand, mammals that are physically immature at birth (e.g., platypuses and humans) show abundant REM sleep at birth, especially in the early stages of life (Ribeiro, 2020, p. 129). A newborn human is incapable of moving, feeding, defending, or cleaning itself. Similarly, a baby platypus is also unable to perform these actions and cannot regulate its own temperature without needing to establish physical contact with its mother (Ribeiro, 2020, pp. 129-130). The high physical immaturity (or fragility, or vulnerability) with which countless organisms begin life represents a clear disadvantage, requiring regular parental care (Ribeiro, 2020, p. 130).

Thus, it is not surprising that high neonatal vulnerability is correlated with a large amount of REM sleep (Chen et al., 2022; Kandel et al., 2013, p. 1150; Ribeiro, 2020, p. 130; Sagan, 1978, p. 156). Newborn humans sleep an average of 16 to 18 hours a day, and about 50% (or more) of this time is spent in REM sleep. In prematurely born babies (who sleep even more), REM sleep time is much more predominant, occurring in about 80% of total sleep time (Grigg-Damberger and Wolfe, 2017; Kandel et al., 2013, p. 1150; Sagan, 1978, p. 156). In addition to humans, scientists also identified a substantially large amount of REM sleep in neonates of numerous species: in chimpanzees (Balzamo et al., 1972), nemestrina monkeys (Reite et al., 1976), rats (Blumberg, 2015; Cui et al., 2019), cats (Jouvet-Mounier et al., 1970), guinea pigs (Jouvet-Mounier et al., 1970), lambs (Ruckebusch et al., 1977; Szeto and Hinman, 1985), and in ferrets (Thurber et al., 2008).

Analyzing ocular activity in fetuses provides another corroboration for my arguments, so I will dedicate this paragraph to that. The density of ocular movements is a way to assess REM sleep activity (Nakahara et al., 2022). Fetal ocular movements consolidate from 23 weeks of gestation, allowing scientists to observe the rapid eye movements typical of REM sleep (Inoue et al., 1986; Okawa et al., 2017). Fetal rapid eye movements potentially denote the existence of REM sleep, although this is inconclusive (Okawa et al., 2017). Despite this limitation, given the possibility that these movements indicate the presence of REM sleep, it is interesting to analyze the results of the study by Okawa and colleagues (2017). In this study, the researchers analyzed, in real-time and over 60 minutes, eye movements in fetuses with a gestational age between 24 and 39 weeks. The results revealed that the period of rapid eye movements was much longer than the period without rapid eye movements. In other studies, scientists showed that between 28 and 30 weeks of gestation, the fetus spends most of its time in REM sleep, with subtle signs of N-REM state (Graven and Browne, 2008; Mizrahi, 2004; Werth et al., 2017). As gestation progresses, REM sleep time is progressively reduced, from 80% (at 30 weeks) to 67% (between 33 and 35 weeks) and then to 58% (between 36 and 38 weeks) (Chen et al., 2022).

At this point, some scientist will attempt to refute my arguments by referencing comparative studies across species that suggest that a potential function of REM sleep is to promote neonatal brain development (e.g., Elgar et al., 1988; Elgar et al., 1990; Zepelin and Rechtschaffen, 1974; Zepelin, 1989; Zepelin et al., 2005). This would explain the vast amount of time spent in REM sleep in neonates. However, as pointed out by Capellini and colleagues (2008), these studies have two major flaws. The first is that the authors did not account for the similarity among the species studied due to their common ancestry, an omission that can lead to erroneous conclusions (Felsenstein, 1985; Harvey and Pagel, 1991; Martins and Garland, 1991). The second flaw is that the comparability of the data has been repeatedly questioned (e.g., Berger, 1990; Campbell and Tobler, 1984; and again by Capellini et al., 2008).

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The research by Capellini and colleagues (2008) is important to my discussion because the authors did not find support for the hypothesis that one function of REM sleep is to promote neonatal brain development. According to the comparative evidence across species in their study, the need for REM sleep was not significantly greater in species with lower neonatal brain mass, even after adjusting for allometry. What makes Capellini and colleagues' study robust is that the scientists considered the similarity among the species studied due to their common ancestry. Moreover, they also relied on high-quality data, taking into account the shortcomings of the aforementioned studies. Capellini and colleagues reported that both N-REM and REM sleep showed significant negative correlations with neonatal body mass and with gestation duration, demonstrating that REM sleep does not promote neonatal brain development. In summary, even after controlling the laboratory conditions and phylogeny, the results of their study *did not* support the hypothesis that REM sleep serves to promote neonatal brain development.

The evidence I presented above reinforces my arguments. Consequently, the correlation between an excessive amount of REM sleep and greater physical immaturity at birth constitutes a vigorous corroboration of the sentinel sleep theory. This is exactly what it predicts. After all, since the time the brain dedicates to REM sleep depends directly on the organism's vulnerability, it is predicted that physically immature newborns have abundant REM sleep compared to more physically mature newborns—being more abundant in premature births and even more so in fetuses the greater their physical immaturity. In the context of greater physical immaturity, especially in premature cases, neural information from proprioceptive mappings seems to be particularly relevant to determine the parameters of REM sleep (e.g., its duration).

It is important to note that the sentinel function of REM sleep can be fully executed only when the organism has reached a mature physical development. When, due to some danger, an organism with sufficient physical maturity is awakened from REM sleep, it is fully capable of defending itself (or the group, or its offspring) with all the vigor that waking up during this period enables. In contrast, many neonates are incapable of such a defensive response. The protective function of REM sleep cannot be accompanied by an appropriate defensive behavioral response at this early (and highly vulnerable) stage of ontogenetic development. *REM sleep is a sentinel mechanism that provides greater protection to the organism during the highly vulnerable N-REM sleep, but this protection can only be effectively achieved if the organism is capable (given the appropriate physical maturity) of fighting or fleeing*.

I did not include, in the results of the studies I analyzed in this section, the standard error and standard deviation information. This also applies to the results of other research presented in other sections. As this information can be easily verified in the articles themselves, I opted to be as concise as possible. I recommend that the reader consult the articles to verify it.

To conclude this section, I will discuss some factors that increase the robustness of an argument whose arguer relies on a correlation to conclude causality. An arguer who aims to establish that *A* causes *B* will increase the robustness of his argument if he demonstrates that the causal direction goes from *A* to *B*, but not from *B* to *A*. After all, a correlation does not indicate a direction of causality (when it exists) (Carnielli and Epstein, 2019, pp. 280-282; Walton, 2008, p. 264; Weston, 2009, p. 49). If the causality from *B* to *A* is as plausible as from *A* to *B*, then it will be impossible to determine a unique causal direction; in this case, it may be that both are causing each other (Walton, 2008, pp. 264-265; Weston, 2009, pp. 49-51). Therefore, clearly demonstrating the implausibility of going from *B* to *A* strengthens the argument for a causal direction from *A* to *B*. This is what I will do for three of the causal arguments I developed.

1. Correlation between greater physical strength and less REM sleep time. What is causing what here? The causal direction is clearly not from REM sleep to greater physical strength. Who would argue that having less REM sleep causes greater muscle strength? Patients medicated with antidepressants experience a total (or near-total) suppression of REM sleep (Kandel et al., 2013, p. 1157; McCarthy et al., 2016; Ribeiro, 2020, p. 171), but they do not develop the muscles typical of high-performance athletes. Clearly, it is not the REM sleep that causes greater muscle strength. It is the greater muscle strength that causes specific changes in REM sleep parameters (e.g., the time invested in it). This

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reinforces my argument that the direction of causality is from greater muscle strength to REM sleep parameters.

2. Correlation between greater body mass and changes in REM sleep parameters. In obese individuals, the changes in REM sleep parameters are analogous to those of high-performance athletes: less REM sleep time and greater latency to the first REM episode. If these changes were responsible for causing obesity, high-performance athletes would constantly be at the mercy of persistent obesity. The alteration of these parameters does not cause obesity. It is obesity that alters these parameters. This reinforces my argument that the direction of causality is from obesity to REM sleep parameters.

3. Correlation between depression and changes in REM sleep parameters. Most humans have REM sleep. Rare are the people who do not (Summer and Singh, 2024). For practical purposes, it is convenient to simplify: virtually all humans have REM sleep. However, not all have depression. In 2023, an estimate presented on the *World Health Organization* (WHO) website pointed to an incidence of depression in about 3.8% of the world's population (WHO, 2023). The causal direction in this case is clearly not from REM sleep to depression. The alteration of REM sleep parameters does not cause depression. If it did, we would all have depression. After all, as I showed, emotional states (e.g., fear and stress) are also correlated with changes in REM sleep parameters. We all experienced these emotions, but not all of us developed depression. It is not the REM sleep that causes depression. It is the depression that causes specific changes in REM sleep parameters. This reinforces my argument that the direction of causality is from depression to REM sleep parameters.

Another important factor is the complexity of causal relationships. Many causes possess a complex chain of causal relations in series. Failing to consider this is an error. It may be that *A* causes *C*, but that this causal relationship occurs due to a third causal factor, *B*, operating between factors *A* and *C*. In this case, it would be more accurate to say that *A* causes *C* indirectly (Walton, 2008, pp. 266-267):

$A \longrightarrow B \longrightarrow C$

When a causal relationship possesses a causality structure in series, it can be described as complex (Walton, 2008, p. 267). And that is precisely what is happening in the causal relationships I addressed in this section. It is not physical exercise (or depression, or stress, etc.) that directly causes changes in REM sleep parameters. Physical exercise increases muscle strength, which in turn reduces the organism's physical vulnerability. And it is this reduction in vulnerability (or its increase in other cases) that causes specific changes in REM sleep parameters. Vulnerability (or the organism's level of protection) is the intermediate causal factor in this complex chain of causal relationships in series.

To reinforce my causal arguments, it remains for me to analyze the possibility of a common cause that could explain the correlations discussed. I will focus on the correlation between greater physical strength and less REM sleep time. Someone might argue that greater physical exertion requires more restorative processes dependent on N-REM sleep, thus costing the time available for REM sleep. Is this plausible? It is a possibility. However, this argument is a double-edged sword: it can be used both to refute and to corroborate my arguments.

According to this line of reasoning, exerting more effort reduces REM sleep due to restorative processes dependent on N-REM sleep and exerting less effort increases REM sleep by requiring less of these processes. It turns out that sedentary behavior in non-obese individuals is correlated with more REM sleep time and shorter REM sleep latency (Seol et al., 2022; Zapalac et al., 2024). And this is something predicted from the sentinel sleep theory. After all, non-obese sedentary individuals lack both the greater muscle strength of more active individuals and the higher body fat of obese individuals. The most reasonable conclusion is that the greater physical vulnerability of non-obese sedentary individuals is the relevant causal factor here. Sedentary behavior reduces muscle strength, and this, in turn, makes the organism more vulnerable than its more active peers, requiring more REM sleep and a shorter latency to the first REM episode.

Moreover, in the study by Kitamura and colleagues (2021), which I discussed at the beginning of this section, the following variables *were not significant* in the comparison between groups: age difference (p = 0.860), time of physical exertion (p = 0.579), and BMI (p = 0.920). One variable that *was significant* between the groups is precisely the difference between muscle mass (p <

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0.001). With this, I aim to demonstrate that since the time of physical exertion (and other variables) did not show a significant difference between the groups, what explains the difference in REM sleep parameters is precisely the difference in muscle mass. Therefore, the double-edged sword proves much more favorable to one interpretation than the other.

The necessary criteria to establish a causal relationship are: (1) it is true that the cause occurred; (2) it is true that the effect occurred; (3) the cause precedes the effect (specific temporal relationship); (4) considering the stipulated conditions, it is practically impossible for the cause to occur and the effect not to occur; (5) the cause plays a crucial role (if the cause does not occur, the effect also does not occur); (6) there is no common cause to explain the cause and effect; (Carnielli and Epstein, 2019, p. 280). The causal arguments I developed based on correlational evidence meet these criteria and, therefore, constitute solid arguments.

An arguer who relies on a correlation to conclude causality (*post hoc* argument) makes his argument fallacious only when the sole evidence he uses to corroborate it is the correlation itself (Walton, 2008, p. 260). This is why correlation alone is incapable of conclusively establishing causality (Carnielli and Epstein, 2019, pp. 277-278; Walton, 2008, p. 260; Weston, 2009, pp. 46-47). The arguments I developed in this section to demonstrate causality are not based solely on correlation itself. I showed that the number of correlations is large enough to indicate coincidence; I provided a robust explanation that connects the causes to the effects; I demonstrated the implausibility of the causal direction occurring in the opposite sense in three of the cases analyzed; I demonstrated that there is no common cause to explain the correlations; and finally, I demonstrated that there is a complex chain of causal connections in a temporal sequence. Therefore, I provided sufficient evidence to conclude causality.

Even if there are some residual flaws in the causal arguments I developed (which does not seem to be the case), they do not compromise them, nor do they turn them into *post hoc* fallacies. Any critical questioning that may arise from my causal arguments will only indicate the need to carry out empirical tests aimed at refuting this causality. This is precisely one of my objectives with this manuscript.

2.3. REM SLEEP IS HIGHLY ADAPTIVE

The scope of the current article does not cover the function of N-REM sleep. However, it is pertinent for me to briefly address its evolutionary origin. As I already stated, the importance of N-REM sleep must be properly understood for the function of REM sleep to be as well. I already addressed (in section 2) the importance of N-REM sleep. However, I can write much more about it. Another way to do it is to address its remote origin and persistence over millions of years of evolution since this behavioral state first emerged.

The term “speculate” is often interpreted in a pejorative sense (Dawkins, 2015b, p. 209), but such a connotation is completely inappropriate in this context. Manifestly, there was no one present to observe the onset of sleep when it first occurred. Moreover, fossils do not include records of organisms' sleep (Nicolau et al., 2000). Therefore, any scientific inquiries into the evolutionary origin of sleep are necessarily speculative. In effect, what interests me is the widely corroborated (and practically indisputable) fact that N-REM sleep is evolutionarily older than REM sleep (Kavanau, 1997; Keene and Duboue, 2018; Nath et al., 2017; Rattenborg and Ungurean, 2023; Ribeiro, 2021, p. 117; Zimmerman et al., 2008), even though we are unable to pinpoint exactly when (and in which lineage) it began.

Sleep debuted in invertebrates because scientists already observed it empirically in zebrafish (*Danio rerio*), fruit flies (*Drosophila melanogaster*), jellyfish (*Cassiopea*), and worms (*C. elegans*) (Nath et al., 2017; Rattenborg and Ungurean, 2023; Zimmerman et al., 2008). Given its predominance in both invertebrates and vertebrates, sleep is certainly a very primeval behavioral state, whose origin may predate the Cambrian Period (Ribeiro, 2021, p. 117), which extends from about 543 to 485.4 million years ago (Paulin and Cahill-Lane, 2021; Robison et al., 2023).

The fact that sleep was observed even in organisms with relatively simple nervous systems (such as C. elegans) implies that sleep constitutes a necessity for any organism that encloses a nervous

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system, no matter how simple or decentralized it may be (Nath et al., 2017; Vyazovskiy and Harris, 2013; Zimmerman et al., 2008). This makes sleep a behavioral state that debuted either concurrently with or shortly after the evolutionary debut of the nervous system. Therefore, the question we must investigate is when the nervous system emerged. Something remarkable about nervous systems and their component units (i.e., the neurons) is that they are highly conserved throughout evolution (Paulin and Cahill-Lane, 2021), which manifests the high adaptive value enclosed by them (Damasio, 2019, pp. 56-60). Despite solid evidence that organisms with nervous systems existed at the beginning of the Cambrian Period, there is still no consensus regarding their evolutionary origin (Paulin and Cahill-Lane, 2021).

Paulin and Cahill-Lane (2021) estimated that the evolutionary origin of neurons and the nervous system occurred during the Ediacaran Period (the geological period immediately preceding the Cambrian Period), which extends from about 635 to 543 million years ago (Rafferty, 2018). Using this estimate for the evolutionary emergence of sleep, it implies that over the more than 543 million years of biological evolution since it arose, sleep—despite the vulnerability to which the organism is subjected during its occurrence—prevailed in organisms with a nervous system. Such an imperative, primeval behavioral state, present in all animals with a nervous system, undoubtedly encloses a crucial biological importance.

Considering the high vulnerability of sleep, what could be done—throughout evolution—to considerably reduce it? There are two ways to deal with it: (1) either non-random elimination removes sleep, or (2) maintains it but finds a way to circumvent the problem of the high vulnerability. I will first address the possibility of sleep being eliminated.

Something notable concerning evolution is that any novelties that provide some adaptive advantage (especially if its impact is substantial) enclose a greater propensity to be conserved and spread over time—hence why they become very old (Dawkins, 2015a, pp. 2-3; Ribeiro, 2021, p. 117). The organisms that survive the sieve of non-random elimination in a given generation are those whose genetic constitution engendered a phenotype that possesses what is necessary to survive and reproduce under the prevailing conditions in the specific niche occupied by their species (Dawkins, 2015a, pp. 2-3, 6; Mayr, 2001, pp. 188-189). Or, more strictly, to survive and reproduce under the conditions that prevailed in the niche when the ancestral generations of the current members of a given species were subjected to the sieve of non-random elimination (Dawkins, 2004, p. 121).

Any evolutionist knows that non-random elimination is prolific when it comes to favoring necessary adaptations (Mayr, 2001, p. 189; Mayr, 2004, p. 214). This means that any necessary adaptation is more prone to spread across various animal lineages, either by emerging in an ancestral species and remaining throughout its various branches through time or by debuting independently (Mayr, 2004, p. 214). Briefly, every adaptive solution that substantially increments the chances of its bearer surviving and reproducing encloses a high biological value.

The current consensus is that all animals exhibit some form of sleep (Libourel and Herrel, 2016). Therefore, given the manifest—and substantial—adaptive advantage of a nervous system associated with the absence of the need to subject the organism to sleep, this phenotypic trait, if it existed, would enclose a high biological value. Consequently, the fact that this phenotypic trait *did not* emerge in any lineage is quite revealing. If there were a way to remove sleep from a lineage of organisms without harming them, the genetic information responsible for this phenotypic effect would be strongly favored by non-random elimination.

If it were possible to overcome the need for sleep under the absence of considerable damage to the organism, this phenotypic trait—presence of a nervous system coupled with the absence of sleep—would spread through the various evolutionary branches from the lineage in which it originally emerged. And not only that. Due to the high adaptive value of a nervous system devoid of the need to subject the organism to sleep, this trait would likely emerge independently in various lineages. Since none of these scenarios occurred, we (evolutionists) can conclude, with considerable confidence, that *sleep constitutes an insurmountable necessity for any organism that encloses a nervous system*.

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In summary, sleep is too important to be removed from organisms with a nervous system. Possessing a nervous system inevitably implies the presence of sleep. And since the solution of removing it is practically impossible, this leads me to the other possibility: finding a way to circumvent the problem of its high vulnerability. What could be done to reduce the vulnerability of sleep? Well, what if the organism's brain, during deep sleep, underwent considerable neural activation (particularly in regions related to attention, detection of dangerous stimuli, and to emotional processing) to make it more alert to the immediate surrounding environment?

The reason for the evolutionary origin of the division of sleep into two periods—N-REM and REM—currently constitutes an enigma to be solved. The question is to elucidate why two sleep states are necessary for the brain (Rattenborg and Ungurean, 2023; Yamazaki et al., 2020). A notable aspect of the sentinel sleep theory is that it unequivocally highlights the answer to this question. N-REM sleep is a non-negotiable biological necessity but encloses a relevant drawback: it makes the organism substantially more vulnerable to predation. Therefore, it is easy to understand that any functionally random evolutionary novelty that led to a significant reduction in the vulnerability of N-REM sleep would inevitably establish itself in the lineage in which it emerged, propagate through various descendant lineages, and would be conserved throughout evolution.

I will present henceforth additional evidence that corroborates my arguments regarding the pressure to develop a way to cope with the heightened vulnerability of N-REM sleep and that REM sleep is a necessary adaptation for those who need to sleep.

Phylogenetic evidence indicates that the central aspects of REM sleep did not evolve independently (Medeiros et al., 2021; Siegel et al., 1998; Yamazaki et al., 2020). As pointed out by Jaggard and colleagues (2021), for more than 50 years, scientists believed that REM sleep was a more recent mechanism, present only in mammals and birds. However, after scientists demonstrated its presence in reptiles, it became believed that REM sleep probably originated in the brainstem of reptiles (the ancestors of birds and mammals) (Siegel et al., 1998). Several subsequent studies reinforced the fact that reptiles also have REM sleep (e.g., Libourel et al., 2018; Shein-Idelson et al., 2016). The alternation between N-REM and REM sleep in birds, mammals, and some reptiles clearly demonstrates a common origin of these wake-sleep cycle mechanisms, as these animals share a common ancestor (Libourel et al., 2018; Medeiros et al., 2021; Siegel et al., 1998).

However, in recent and independent research, scientists showed that, in addition to reptiles, even fish, drosophila, octopuses, and other invertebrate species also have analogs of REM and N-REM sleep (Brown et al., 2006; Frank et al., 2012; Jaggard et al., 2021; Kanaya et al., 2020; Leung et al., 2019; Medeiros et al., 2021; Meisel et al., 2011; Nath et al., 2017; Ramón et al., 2004; Tainton-Heap et al., 2021; van Alphen et al., 2013). The above evidence points to the possibility that a state analogous to REM sleep emerged early in animal evolution, long before the branching of amniotes (around 450 million years ago) (Jaggard et al., 2021; Leung et al., 2019). This initial version would then have become more complex over time until it eventually presented (more recently) the typical characteristics of REM sleep in reptiles, birds, and mammals.

Another possibility is that, instead of a single origin, the REM sleep of reptiles, birds, and mammals and its analog in present in fish, drosophila, octopuses, and other invertebrate species constitute convergent evolution, having debuted independently in evolutionary history (Medeiros et al., 2021). After all, cephalopods (such as octopuses) diverged from vertebrates more than 500 million years ago (Medeiros et al., 2021; Shu et al., 2001; Vitti, 2013). Regardless of the answer—convergent evolution or single origin (homology)—either one strongly corroborates my argument that the sentinel mechanism constitutes a necessary adaptation for any organism that needs to sleep. These phylogenetic and certainly homologous evidence in the case of reptiles, birds, and mammals reinforce my argument regarding the high biological value of REM sleep.

As pointed out by Jaggard and colleagues (2021), basic analogs of both quiet sleep and active sleep (the precursors of N-REM and REM sleep, respectively), as well as N-REM and REM sleep themselves, were discovered from humans to fish, and from drosophila to octopuses. Paradoxical sleep (or active sleep)—similar to the wakeful state—exists from mammals to invertebrates (Jaggard et al., 2021). The evidence of active sleep in drosophila, zebrafish, cuttlefish, and octopuses (Frank et

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al., 2012; Leung et al., 2019; Medeiros et al., 2021; Tainton-Heap et al., 2021; van Alphen et al., 2013) indicates a clear selection pressure, throughout the course of evolution, for the development of mechanisms that enable the transition from quieter sleep to a more active (or protective, as I am arguing) sleep.

This evidence corroborates the arguments I developed regarding the pressure to develop a way to cope with the high vulnerability of N-REM sleep. They also corroborate the arguments that REM sleep is a necessary adaptation for any organisms that need sleep. In fact, the pressure to develop a mechanism to compensate for the vulnerability of quiet sleep is so great that it is possible that many animals developed it independently. The cuttlefish is an animal whose analogue to REM sleep may have independently debuted in this invertebrate species (Frank et al., 2012).

Here is the conclusion of the theme of this section. REM sleep consists of a necessary adaptation for any organism that needs to sleep; it is the solution to the problem of the high vulnerability of N-REM sleep. I demonstrated that the pressure exerted by predation played a significant role in the evolution of sleep. This reinforces the argument that this pressure is much more complex than previously assumed (see Capellini et al., 2008). The problem of vulnerability that REM sleep solves naturally leads me to another much-debated question regarding it: understanding why it is periodically distributed. Or, to put it another way, why sleep is based on cycles that alternate between N-REM and REM sleep throughout the time the organism rests. This is the subject of the section hereinafter.

2.4. REM SLEEP IS CYCLICAL DUE TO ITS PROTECTIVE FUNCTION

The biological function of the alternation between N-REM and REM sleep is currently unknown (Le Bon, 2021; Vyazovskiy and Delogu, 2014). From the perspective provided by the sentinel sleep theory, the answer becomes apparent. Indeed, explaining why REM sleep is cyclical is easier than addressing its evolutionary origin. When the brain is periodically subjected to REM sleep—the state of dormant vigilance—it enables a more consistent defense for the organism. If the brain were subjected to only one REM episode during N-REM sleep (e.g., at the beginning of the night), the protection offered by the state of dormant vigilance would be significantly reduced. After all, during the remaining time of sleep, the organism would be deprived of this defense mechanism that contributes to reducing the vulnerability experienced during N-REM sleep.

Making the brain more alert to the immediate surrounding environment only once during the entire rest period is less efficient as a survival mechanism than doing so based on a periodic distribution. If we compare an organism with only one REM episode to one with multiple episodes, it becomes clear which one has a greater adaptive advantage over the other. This is why non-random elimination favored organisms equipped with the genetic information to develop a central nervous system that—rather than undergoing just one episode of dormant vigilance—was subjected to a greater number of such episodes during N-REM sleep. This brings me to the final topic: the origin of REM sleep (including the origin of its periodic distribution).

2.5. REM SLEEP EVOLVED FROM A BRIEF AWAKENING FROM N-REM SLEEP

Addressing the origin and evolution of REM sleep will allow me to demonstrate that the theory I developed here makes evolutionary sense—a necessity for any arguer aiming to explain the biological function of a trait. This is why this section exists.

For obvious reasons, we (scientists) are incapable of knowing with certainty how the behavioral state we happen to call "REM sleep" first emerged in evolutionary history. Some of its facts will inevitably continue to escape us. Remaining forever as objects of speculation—no matter how well-founded these speculations may be. There are mainly four pieces of information about primeval REM sleep that we can never know factually: (1) how many genes were responsible for engendering this behavioral state the first time it emerged in an organism, (2) the number of REM episodes in that primeval occurrence, (3) its latency, and (4) its intensity (or density).

Up to this point, I described the evolutionary origin of REM sleep as if it were due to the action of only one specific gene. This may certainly have been the case. However, it could also have been

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based on the joint action of two or more genes. Scientists have long known that the formation of a phenotypic trait often involves the influence of more than one gene—what is termed *polygeny* (Mayr, 1982, p. 794; Mukherjee, 2016, p. 197). When a gene has a phenotypic effect, this effect (in the vast majority of cases) is not due to the gene *per se* because phenotypic traits are often engendered through the action of multiple genes (Mayr, 1982, p. 794; Mukherjee, 2016, p. 197). Therefore, the onset of REM sleep could have been based on the action of more than one gene rather than just a single one. The definitive answer to this question, however, we will never know for sure.

I am unable to empirically analyze REM sleep in its primeval occurrence, but I can develop *a priori* arguments about its initial complexity and the number of episodes. This is what I will present hereinafter in the form of a historical narrative.

Because we deal with past events (e.g., the origin of a new trait), we, evolutionary biologists, are unable to empirically test our object of study. Evolutionary phenomena are inaccessible to experimental methods. Thus, to obtain answers to evolutionary questions, we must resort to a non-experimental method called *historical narratives* (Mayr, 2004, pp. 32, 94). This method is based on the formulation of a narrative about past events, primarily supported by their consequences, and whose explanatory value must be tested. To do it, one must rely on any evidence that can refute or corroborate the predictions generated from the historical narrative (Mayr, 2004, pp. 32, 94).

Before presenting the historical narrative I developed to explain the evolutionary debut of REM sleep and its subsequent evolution, I will introduce certain crucial concepts that will serve to ground the proposed narrative. These will be used as a more secure starting point on which to base my speculations. By ensuring a solid foundation, I hope that the proposed narrative is not far from the truth.

As determined by the first law of probability, *the probability of two events occurring together is never greater than the probability of each event occurring separately* (Arkes et al., 2022; Franco, 2009; Lu, 2016; Mlodinow, 2009, p. 32). Putting it another way, the coincidence (i.e., the joint incidence) of two or more events implies multiplied probability (Arkes et al., 2022; Dawkins, 2015b, p. 227). The joint incidence of event A with event B is the product of the isolated probability of event A occurring multiplied by the isolated probability of event B occurring (Arkes et al., 2022; Dawkins, 2015b, p. 206). The probability of a single event occurring is demonstrated by the following formula:

$$P(A) = n(A) / n(\Omega)$$

where $P(A)$ is the probability of event A occurring, $n(A)$ is the number of sample elements referring to event A, and $n(\Omega)$ is the sample space of all possible outcomes. In the case of events that occur together and where the occurrence of one does not affect the probability of the other occurring, the formula is this:

$$P(A \cap B) = p(A) \cdot p(B)$$

where $P(A \cap B)$ is the probability of event A occurring together with event B, $p(A)$ is the isolated probability of event A occurring, and $p(B)$ is the isolated probability of event B occurring. If we added another event—$P(A \cap B \cap C) = p(A) \cdot p(B) \cdot p(C)$—the probability of the events A, B, and C occurring together would be even lower. To illustrate: the chance of someone getting the number 5 on a six-sided dice is 1/6 (or ≅ 16.67%). The chance of someone getting the sequence 5, 3, and 1 is $1/6 \cdot 1/6 \cdot 1/6$ (or ≅ 0.46%). This is why the probability of two (or more) events occurring together cannot be greater than the probability of each event occurring separately.

The first law of probability is closely related to the mathematical concept of complexity, according to which complexity constitutes a statistical concept (Dawkins, 2010a, p. 417; Dawkins, 2010b, p. 361; Dawkins, 2015b, p. 12; Pal and Pal, 1991; Pringle, 1951). Under this sense, complexity is *a priori* associated with high statistical improbability, being inversely proportional to its probability of occurring. The greater the complexity of something, the lower its probability of occurring, and vice versa (Dawkins, 2010b, p. 361; Knight, 2009, p. 558; Pal and Pal, 1991). Complex (or statistically improbable) things do not arise suddenly. To be achieved, complexity—especially in the biological context—requires a countless number of sufficiently simple intermediate steps (Dawkins, 2010a, p. 417; Dawkins, 2015b, pp. 12, 61).

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Biological complexity is distinguished from inorganic complexity (which is comparatively more limited) due to the attribute of *functionality* (e.g., walking, running, flying, swimming, or digging). In general, the functionality that defines the high biological complexity encompasses all mechanisms directly or indirectly responsible for the conservation of life (due to the maintenance of a chemical balance favorable to it) and for reproduction. In addition to these, also included are the mechanisms that allow the organism the ability to find energy and process it, to replace all aging subcomponents that die, and to defend itself from physical injuries and diseases (Damasio, 2003, p. 30; Damasio, 2019, p. 40; Dawkins, 2015a, p. 2; Dawkins, 2015b, pp. 15-16). The high statistical improbability manifested by living beings emerges in the world as a product of a long series of intermediate evolutionary steps that are simple enough (compared to the previous steps) to debut by chance—being, therefore, functionally random (Dawkins, 2010a, p. 417; Dawkins, 2015b, pp. 12, 61).

Everything I discussed in the preceding paragraphs is particularly relevant to the *a priori* arguments about the primeval occurrence of REM sleep that I will develop hereinafter. To better explain the historical narrative, I will first provide a brief overview of the current complexity of REM sleep.

REM sleep is generated by the coordinated action of various neurotransmitter systems in the brainstem, forebrain, and hypothalamus, and by the activation of several brain regions (e.g., amygdala, hippocampus, motor cortex, cingulate cortex [especially the anterior region], brainstem, thalamus, and visual association cortex); it includes intense muscle atonia; and it is based (in humans) on an amount of four to six REM episodes throughout the entire rest period (Akre, 2024; Desseilles et al., 2011; Fraigne et al., 2015; Gazzaniga et al., 2016, pp. 146, 153; Hess et al., 1987; Kandel et al., 2021, p. 1082; Nofzinger et al., 1997). Muscle tone is present during N-REM sleep but is low and does not compare to the intense muscle atonia associated with REM sleep, which practically paralyzes the body. With few exceptions, most of the body remains incapable of movement during REM sleep. The muscles involved in breathing move, but in a milder way. Meanwhile, the muscles that control eye movements, as well as the muscles of the inner ear, move intensely (Akre, 2024; Bear et al., 2016, pp. 659, 915; Kandel et al., 2021, pp. 1082, 1097; Purves et al., 2004, p. 671).

If REM sleep, at the time it first emerged, were already regulated by multiple neural regions, already included, say, five REM episodes alternating with N-REM periods, and intense muscle atonia was already present, we would be dealing with a highly complex neural behavior, based on a series of independent events cooperating for the same purpose: reducing the organism's vulnerability. All this complexity would naturally require efficient coordination between all neural regions involved in this primeval occurrence of REM sleep. However, the probability of all these independent events occurring together is negligible. Therefore, the primeval occurrence of REM sleep was—in all likelihood—not like this.

The scenario of a highly complex primeval REM sleep is equivalent to a huge stroke of luck, a high statistical improbability. After all, the complexity in this case is both structural and behavioral. Therefore, I can assert—with the confidence derived from statistics—that the primeval REM sleep was not based on a highly improbable event. Its onset, for the sake of plausibility, had to be simple. We cannot postulate a primeval occurrence of REM sleep based on multiple independent events occurring simultaneously, as the probability of this occurring is far lower than the probability of just one of these events occurring.

This leads me to the following questions: What is the simplest possible scenario for the primeval occurrence of REM sleep? What scenario requires the least statistical improbability? Considering that the neural mechanisms responsible for regulating deep sleep obviously already existed, the most probable scenario (due to its simplicity) is that REM sleep emerged as an error causing a brief awakening from deep sleep. (Note that the term “error” should be understood in the sense of a failure in the control of the transition from deep sleep to wakefulness, causing the organism to awaken before the usual time.) Consequently, this error provided a limited but not non-existent adaptive advantage for the organism. After all, briefly waking up from deep sleep—a highly vulnerable state—can contribute to survival. This contribution was limited, but the chances of survival were higher for the organism that briefly woke up from deep sleep than for those that remained asleep. The brief

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awakening allowed for more efficient scanning of the surrounding environment for the presence of any potential dangers. From this, it is easy to see that any subsequent modification (due to a functionally random mutation) that enhanced this function would clearly be favored by non-random elimination. What improvement might have occurred next?

The next evolutionary step was probably the brief awakening turning into an *ease* to wake up. Now, instead of REM sleep fully awakening the organism, the brain enters a state that only facilitates awakening. Thus, the advantage of greater neural activity—to ensure vigilance and readiness to fight or flee—is harnessed without affecting the organism's sleep. The selective pressure for this transformation occurred because the awakening caused by the primeval REM sleep inevitably affected the organism's sleep. Notably, at this stage (of the ease of awakening), the intense muscle atonia (due to the high complexity of this mechanism) was probably still absent. Considering that N-REM sleep already had milder muscle atonia, it is possible that REM sleep had it too. However, due to the high intensity of neural activation, this milder muscle atonia (assuming its presence) was probably unable to prevent the organism from moving during REM sleep.

This seems like a problem for my historical narrative. After all, it is obvious that an organism moving while asleep attracts predators or even competitors from its own species (Ribeiro, 2020, p. 129). However, we must ask the following. Who is more vulnerable: an immobile organism while in deep sleep (with low levels of attention, vigilance, and readiness) or a sleepwalking organism during REM sleep (with high levels of attention, vigilance, and readiness)? Which one is better prepared to fight or flee? The answer is self-evident.

The next evolutionary step was probably the occurrence of more than one period of REM sleep. Now, instead of just one, the organism had more than one REM episode (probably two, but there could have been more). The addition of one or more REM episodes provided a more considerable adaptive advantage than the previous version, with just one episode during the entire rest period. After all, with more than one sentinel period during the so vulnerable deep sleep, the organism's brain had more opportunities to effectively scan the surrounding environment. At this stage, the intense muscle atonia was probably also absent.

It is at this point in the narrative that selective pressure for the development of intense atonia of the striated muscles intensified. The presence of more than one REM episode—especially when this number exceeded two—created growing pressure to develop a mechanism capable of significantly *reducing* the movements of striated muscles during REM sleep. The development of this mechanism was probably the next evolutionary step. Subsequently, this mechanism became more complex to the point of effectively *paralyzing* striated muscle movements during REM sleep. (The temporary paralysis of muscle movements certainly came after their reduction. After all, a mechanism to reduce striated muscle movements is less complex [or statistically more probable] than a mechanism to paralyze them. Furthermore, N-REM sleep already had mild atonia, which probably served as the basis for the mechanism of striated muscle paralysis in REM sleep.)

Although we cannot know for sure, the *a priori* arguments I developed here should generally not be far from the truth concerning the primeval REM sleep and its subsequent evolution over countless generations. Obviously, no fossil is (or will be) able to corroborate these claims; fossils do not contain records of sleep (Nicolau et al., 2000; Ribeiro, 2020, pp. 126-127). One fact about the primeval occurrence of REM sleep is the certainty that we will never know for sure how it began. The primeval REM sleep and its subsequent evolution will retain some secrets. The factual details of the evolutionary origin of both N-REM and REM sleep, as well as the origin of this separation, will remain *in perpetuum* as objects of speculation. What we must ensure (for as reliable an understanding as possible) is that these speculations are well-founded. This is what I hope to have achieved with the historical narrative I developed in this section.

3. ATTEMPTS AT REFUTATION

Considering the arguments I developed, it might be—superficially—expected that REM sleep would exhibit a lower arousal threshold than N-REM sleep. However, a search through the scientific literature reveals that REM sleep *does not* have a lower arousal threshold than N-REM sleep, and may

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even be higher (e.g., Ermis et al., 2010; Pilon et al., 2012). Does this constitute a fatal refutation of my arguments? For a careless scientist, the answer would be an emphatic "yes." However, these facts actually support (rather than refute) my arguments.

In section 2.5, I argued that at one point in the evolutionary trajectory there was a growing pressure to develop mechanisms to prevent the organism from waking up during REM sleep. If the arousal threshold in REM sleep were lower than that of N-REM sleep, the organism would wake up much more easily, especially considering the high neural activity of several regions associated with alertness and attention (as I demonstrated in section 2.1). *Therefore, rather than entailing an arousal threshold lower than that of N-REM sleep, it was imperative that REM sleep entail an arousal threshold analogous to or even higher than that of N-REM sleep*.

To provide additional evidence for the assertions of the previous paragraph, I will turn to cholinergic neurons. Cholinergic neurons—responsible for providing the primary source of acetylcholine to the cerebral cortex—are known to help activate the cortex during both wakefulness and REM sleep (Brown et al., 2012; Datta and Siwek, 2002; Deurveilher and Semba, 2011; Watson et al., 2010). One of the effects of acetylcholine is to increase wakefulness (España and Scammell, 2011; Watson et al., 2010). Indeed, the release of acetylcholine during REM sleep in the basal forebrain and pontine reticular formation is significantly greater than during wakefulness (Vazquez and Baghdoyan, 2001; Watson et al., 2010).

What I aim to demonstrate with this evidence is that REM sleep already possesses numerous mechanisms that facilitate awakening, reinforcing my argument that if the arousal threshold during REM sleep were lower, the organism would wake up much more easily, constantly compromising sleep. The sentinel function of REM sleep is to facilitate awakening, but this ease cannot be that high. There must be a limit. Otherwise, almost anything would wake the organism. *The evolution of REM sleep relied on the development of mechanisms that facilitate awakening, but it also involved the co-evolution of mechanisms that prevent this ease of awakening from becoming too easy to the point of disrupting the organism's sleep*.

To better substantiate the preceding assertion, I will discuss serotonin. Electrophysiological, neurochemical, and neuropharmacological evidence indicates that serotonin promotes wakefulness (Brown et al., 2012; Monti and Jantos, 2008). Furthermore, serotonin helps inhibit both REM and N-REM sleep (Boutrel et al., 2002; Horner et al., 1997; Monti and Jantos, 2008). The high and constant activity of serotonergic neurons during wakefulness contributes to preventing the transition from wakefulness to either REM or N-REM sleep (Boutrel et al., 2002; Brown et al., 2012; Monti and Jantos, 2008). Serotonergic neurons fire less during N-REM sleep and (which is particularly important for my arguments) barely fire at all during REM sleep (Brown et al., 2012; Monti and Jantos, 2008).

Additional evidence comes from orexin neurons, which also play a crucial role in promoting wakefulness (De Luca et al., 2022; Feng et al., 2020; Ito et al., 2023; Mogavero et al., 2023; Ono and Yamanaka, 2017). This role is so crucial that during the transition to wakefulness, orexin neurons fire at an intensive rate (de Lecea and Huerta, 2014; Lee et al., 2005; Mileykovskiy et al., 2005). During wakefulness, orexin neurons are highly active. When directed to target regions, orexin elevates alertness, promotes arousal, and helps sustain the wakefulness state (de Lecea and Huerta, 2014; De Luca et al., 2022; Estabrooke et al., 2001; Ito et al., 2023; Mogavero et al., 2023). However, during REM sleep, orexin release decreases or ceases because orexin neurons reduce their activity or become silent (de Lecea and Huerta, 2014; Estabrooke et al., 2001; Mochizuki et al., 2011; Mogavero et al., 2023).

In addition to orexin neurons contributing to promoting or sustaining wakefulness, they also prevent the expression of both REM and N-REM sleep (De Luca et al., 2022; Estabrooke et al., 2001; Kandel et al., 2021, p. 1097; Mochizuki et al., 2011; Sasaki et al., 2011). To promote arousal, orexin neurons indirectly inhibit sleep by acting on the neurons of the ventrolateral preoptic nucleus (VLPO), a crucial region for initiating and maintaining sleep (De Luca et al., 2022). This is known because acute stimulation of the VLPO induces sleep (De Luca et al., 2022), and because local administration of orexin in the VLPO causes animals to awaken from sleep (Mavanji et al., 2015).

Selective loss of orexin causes the intrusion into wakefulness of typical REM sleep elements, such as paralysis episodes called cataplexy (characterized by the sudden loss of muscle tone)

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(Dauvilliers et al., 2007; Mochizuki et al., 2011; Ribeiro, 2020, p. 142). Loss of orexin also causes narcolepsy in rats, humans, and dogs (Dauvilliers et al., 2007; Mileykovskiy et al., 2005; Mochizuki et al., 2011; Sasaki et al., 2011), impairs the maintenance of wakefulness, destabilizes wakefulness and sleep states, and causes fragmented sleep and sudden entry into REM sleep (Dauvilliers et al., 2007; Kandel et al., 2021, p. 1097; Mochizuki et al., 2011; Ribeiro, 2020, p. 142; Sasaki et al., 2011). As we can see, this abundant evidence solidly supports my arguments for the co-evolution of mechanisms that prevent excessively easy awakening during REM sleep.

Another attempt to refute my arguments is to appeal to the locus coeruleus (LC). Since the LC is strongly inhibited during REM sleep (Osorio-Forero et al., 2022; Schwartz and Roth, 2008), this fact might seem like an obvious refutation of the sentinel sleep theory. After all, the LC consists of the primary source of a hormone directly associated with stress and arousal: norepinephrine (NE) (Koshmanova et al., 2023; Poe et al., 2020). The LC-NE system—by increasing the organism's alertness, stress, and arousal—is involved in neurobiological processes that place it as an important component of the fight-or-flight response (España and Scammell, 2011; Osorio-Forero et al., 2022; Ross and Bockstaele, 2021; Yamaguchi et al., 2018).

These facts about the LC-NE system may (superficially, once again) seem like a significant blow to the arguments I am proposing for the protective function of REM sleep. However, a more careful analysis reveals that these facts actually support (rather than refute) the sentinel sleep theory. The reason it is expected that the LC-NE system would be strongly inhibited during REM sleep is that, among other functions, it plays a central role in maintaining wakefulness (Kjaerby et al., 2022; Poe et al., 2020; Watson et al., 2010). Norepinephrine is a hormone known to promote wakefulness (Watson et al., 2010), and it has been widely demonstrated that activating the LC causes the transition from sleep to wakefulness (Carter et al., 2010; Kjaerby et al., 2022; Swift et al., 2018; Yamaguchi et al., 2018). Moreover, the increase in firing frequency in the LC precedes spontaneous awakenings from N-REM sleep (Aston-Jones and Bloom, 1981; Foote et al., 1980; Osorio-Forero et al., 2022; Takahashi et al., 2010). Finally, it has also been shown that mice experience spontaneous awakening from N-REM sleep more frequently when LC neural activity increases (Cardis et al., 2021; Osorio-Forero et al., 2021).

These facts are crucial for understanding why the LC-NE system is almost completely inhibited during REM sleep. They support my argument that there was increasing selective pressure for the development of mechanisms to prevent the organism from waking up too easily during REM sleep. Consequently, any nucleus, region, or brain system directly and crucially involved in the transition from sleep to wakefulness (such as the LC) would have to undergo significant inhibition. *Therefore, even though the LC-NE system is important in the fight-or-flight response—which would make it an ideal candidate to remain highly active during REM sleep—its crucial role in waking the organism ensured its suppression during REM sleep*. If the LC did not play a central role in waking up the organism, this nucleus would certainly be active during REM sleep.

Another attempt to refute my arguments is to point out the fact that N-REM sleep is not a homogeneous sleep state in terms of reduced capacity to awaken (Kjaerby et al., 2022). N-REM sleep encloses a complex microarchitecture that includes periodic episodes of micro-awakenings (Kjaerby et al., 2022). Superficially, this fact might seem to present some level of refutation. However, as with the other examples I provided above, a more careful analysis reveals corroboration rather than refutation. According to the arguments I presented in section 2.5, REM sleep probably debuted in the form of a brief awakening from N-REM sleep. *Therefore, the presence of micro-awakenings during N-REM sleep only reinforces my argument that REM sleep evolved from a brief awakening from N-REM sleep*. If episodes of micro-awakenings already occurred in the distant past, an error in one of them is precisely what could have caused the brief awakening from N-REM sleep—the event I argued was the primeval occurrence of what later became REM sleep as we know it.

I want to end this section with a reflection. What happens when you seek evidence to refute a theory, and they only reinforce that theory? Will your text be filled with corroboration or refutation? Over the more than three years I worked on this paper, I sought several times to refute the sentinel sleep theory. The more I tried, the more evidence supported it. My various attempts to refute it were

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unsuccessful, and—precisely for this reason—they are present throughout this paper as corroborations. I am not claiming that refutations do not exist, nor that the theory is complete. However, considering all the extensive empirical foundation I presented to corroborate the sentinel sleep theory—and especially the numerous attempts that failed to refute it—I believe I clearly demonstrated the primary function of REM sleep. (Let this statement not be interpreted as if I were claiming that secondary or emergent functions do not exist. That is a separate issue and is beyond the scope of an already overlong article.)

4. LIMITATIONS

REM sleep is an exceedingly complex biological mechanism. It would be naïve to think that one could address—in a single article—all the nuances and consequences stemming from its primary biological function. This is impossible. Especially considering that an article does not have the length of a book. And even a book's length would be insufficient. I hope my scientific peers understand that the fact I have not addressed all the nuances and consequences of the sentinel function does not imply that it is not the primary function of REM sleep. I believe I provided sufficient evidence to clearly demonstrate it. However, as I pointed out earlier, this does not mean that the conceptual framework of the sentinel sleep theory is complete. On the contrary, much work remains to be done to expand it. This is a task for future research.

Due to the length of my article, I was unable to address how other hypotheses previously proposed for the function of REM sleep (presented in the introduction) fit into the conceptual framework of the sentinel sleep theory—a significant gap that needs to be addressed in future work. For example, given the well-established relationship between REM sleep and learning (Bear et al., 2016, p. 665; Gazzaniga et al., 2016, pp. 150-151; Moruzzi and Eccles, 1966; Ribeiro, 2003; Ribeiro, 2020, p. 130), the theory of the biological function of REM sleep cannot ignore this within its conceptual framework. It would be incomplete without this explanation. The same applies to the strong relationship between REM sleep and dreams, another well-established fact (Desseilles et al., 2011; Gazzaniga et al., 2016, p. 146; Martin et al., 2020; Ribeiro, 2020, p. 169; Solms, 2000).

In fact, I am already working on two additional articles to address the major gaps in this one. Since these other articles depend on this one being published first, I am obviously unable to present their final versions. Once this article is formally published, my other two articles can also be published. After that, some of the major gaps in this current article will be filled, further reinforcing and expanding the arguments and conceptual framework I developed here. Of course, what I just stated is merely anecdotal and thus lacks scientific value. However, I consider it important to inform my scientific peers that the reason I could not address the aforementioned gaps is due to the length of the article, not due to the nonexistence of those arguments. The conceptual framework I developed for the sentinel sleep theory is too extensive and complex to fit into this single article. I will need to develop it further in subsequent articles.

5. A SIGNIFICANT AND DETRIMENTAL FAVORITISM

Since the current *zeitgeist* is dominated by an excessive favoritism towards the hypothesis that the function of REM sleep is to aid learning (Bear et al., 2016, p. 665; Gazzaniga et al., 2016, pp. 150-151; Kandel et al., 2021, pp. 1091-1092; Moruzzi and Eccles, 1966; Ribeiro, 2020, p. 130), I need to address this issue. After all, this favoritism can hinder the proper understanding of the true biological function of REM sleep.

As pointed out by Capellini and colleagues (2008) and Ribeiro (2020, p. 171), the hypothesis that a function of REM sleep is to aid learning and memory consolidation led many scientists to argue that species with considerable cognitive abilities should require more time invested in REM sleep. However, dolphins—animals whose high intelligence is well-established—do not have REM sleep, while armadillos—less intelligent animals—have it in abundance (Ribeiro, 2020, p. 171). Moreover, if REM sleep played a crucial role in learning, patients medicated for depression (who experience reduced or suppressed REM sleep) should exhibit learning deficits. But why do they not present it? Why is the time spent in the REM period not strongly correlated with learning in humans (Ribeiro,

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2020, p. 171)? I will discuss henceforth a study that, among other objectives, analyzed the REM sleep learning hypothesis by comparing numerous species.

Capellini and colleagues (2008) conducted their research using a database that, as of June 29, 2007, contained records of REM and N-REM sleep from 127 distinct species across 46 families and 17 orders. The researchers also compiled information about laboratory procedures, as different laboratory conditions and measurement methods can affect data analysis (Campbell and Tobler, 1984; Siegel, 2005). As reported by Capellini and colleagues (2008), after controlling the laboratory conditions and phylogeny, the research results *did not support* any of the traditional explanations claiming that REM or N-REM sleep serves to benefit cognition, aid brain development, or to conserve energy.

The above evidence demonstrates that, despite the association with learning, REM sleep does not play a critical role in it. Therefore, my omission of how the sentinel sleep theory explains the association of REM sleep with learning does not compromise the quality of the current work. This is a significant gap but does not impact the arguments I developed to demonstrate the sentinel function of REM sleep. To further reinforce the argument that REM sleep does not play a critical role in learning, I will present henceforth additional evidence from genetics.

Whenever a neuron undergoes the process of encoding a new memory, coding genes capable of modifying synapses are promptly activated. The so-called *Immediate-Early Genes* (IEGs) are the first genes involved in this process, being activated a few minutes after neuronal electrical reverberation begins (Abraham et al., 1991; Bahrami and Drabløs, 2016; Davis et al., 2003; Ribeiro, 2020, pp. 209-210). A specific number of IEGs need to be expressed for other genes essential for consolidating long-term memories to also be expressed (Abraham et al., 1991; Davis et al., 2003; Okuno, 2011; Ribeiro, 2020, p. 210).

Considering the hypothesis that a function of REM sleep is learning—and that IEGs are necessary for long-term synaptic modifications to be caused and for memories to be formed (Okuno, 2011; Ribeiro, 2020, p. 210)—one would expect to find an increase in IEGs expression during sleep, especially during REM sleep. However, in organisms not exposed to new stimuli during wakefulness, sleep (including REM sleep) strongly suppresses the expression of IEGs rather than increasing it (Decker et al., 2010; Pompeiano et al., 1992; Pompeiano et al., 1994; Pompeiano et al., 1995; Pompeiano et al., 1997; Ribeiro et al., 1999; Ribeiro, 2003; Ribeiro, 2020, p. 216). The expression of IEGs increases during REM sleep, but not during N-REM sleep, only when the organism is exposed to new environmental stimuli during recent wakefulness (Ribeiro et al., 1999; Ribeiro, 2003; Ribeiro, 2020, pp. 217, 220).

What may explain this disparity in the expression of IEGs is the difference between the functions of N-REM and REM sleep. Whatever the function (or functions) of N-REM sleep may be, it does not serve as a sentinel mechanism. The sentinel function is carried out by REM sleep, and it is precisely this function that may explain why the expression of IEGs increases during REM sleep but not during N-REM sleep after the organism is exposed to new—and therefore potentially dangerous—environmental stimuli. In other words, the sentinel function of REM sleep seems to explain very well why the expression of IEGs is suppressed during N-REM sleep even after new environmental stimuli are presented during recent wakefulness. For this assertion to make more sense, I need to elaborate it further.

Registering information (i.e., learning about the surrounding environment and its components, including predators) is a substantial adaptive advantage (Damasio, 2012, pp. 67-68; Damasio, 2019, pp. 61-62). As I discussed earlier, when an organism is exposed to new information (or stimuli), IEGs are promptly activated. A noteworthy aspect of the expression of these genes is that it constitutes a protective mechanism, and it is easy to see why. Let us compare an organism whose IEGs expression takes a long time—so long that we could call them very late genes—with one whose expression truly deserves the term "immediate." In the first case, the expression of genes essential for forming and consolidating new memories takes so long that, after interacting with a predator, the prey's brain is unable to quickly modify its synapses to register the new (and biologically relevant) information obtained from that encounter. In the second case, the brain can quickly modify synapses and register

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new memories related to that predator. The second organism has a clear adaptive advantage over the first. But why does this swift expression of genes involved in memory formation and consolidation make it more protected?

Well, memories are biologically valuable especially because they allow the organism to store relevant information for its own survival—derived from both the external environment and the body's internal milieu (Damasio, 2012, pp. 67-68; Damasio, 2019, pp. 61-62, 75-83). Therefore, it is advantageous to respond to sensory novelties with an immediate expression of genes related to the formation and consolidation of new memories because one never knows when such novelty will bring danger with it. Although most of the time the number of neutral stimuli is greater than the number of dangerous ones, it is advantageous to always promptly activate the aforementioned genes precisely because of the times when danger is present. When sensory novelty includes danger, the organism whose memory formation process is faster has a significant advantage over those whose memory formation process is slower—an advantage that can mean the difference between life and death.

This is why an organism that more quickly and effectively stores sensory information obtained from a predator after encountering it is comparatively less vulnerable to it. If you escape from that predator during the first encounter and it (or another of the same species) returns to attack you within a few minutes, the information obtained—and quickly learned by the brain after the first encounter—will make you better protected. After all, the information stored about the predator (e.g., how and where it attacked you) increases your chances of surviving if attacked again by the same predator or any other of the same species. Knowing that you need to avoid its claws or tail makes you better protected compared to a contemporary of yours who did not store this information.

The more information you gather about a predator, and the more quickly your brain registers it, the better you can defend yourself when it attacks you again. Knowing your enemy's attack tactics and typical behaviors increases your chances of staying alive when dealing with it. The more you know your enemy, the better protected you can be from him. This is why *the expression of IEGs constitutes a protective mechanism during wakefulness; it ensures that the organism learns quickly when danger is a possibility*. This brings me to the final part of my argument.

Since IEGs expression constitutes a protective mechanism *during wakefulness*, it is possible to explain—through the sentinel function of REM sleep—its increased expression during REM sleep but not during N-REM sleep after the organism has been exposed to new environmental stimuli. The potential danger inherent in sensory novelty induces the expression of IEGs during wakefulness. Since the function of N-REM sleep is not to serve as a sentinel mechanism (nor is it critically related to learning), IEGs expression is suppressed during this sleep state. However, things change during the REM period. Since the primary function of REM sleep is to serve a protective role, IEGs expression reoccurs.

The reason this occurs is simple: the expression of IEGs during wakefulness serves to reduce the organism's vulnerability when new stimuli are received. And since the function of REM sleep is also to reduce the organism's vulnerability, the expression of IEGs occurs during REM sleep whenever the organism has recently been exposed to new environmental stimuli. When a significant vulnerability is presented during wakefulness, the protective mechanism that deals with it (the expression of IEGs) is reactivated during REM sleep. This only happens due to the protective function of REM sleep. A notable consequence of this is that, if a predator attacks an organism during REM sleep and after this organism has recently been exposed to new stimuli during wakefulness, the information obtained from that attack will be promptly stored by the brain due to the elevated expression of IEGs. Therefore, considering the sentinel function of REM sleep, what would be strange is if the expression of IEGs were suppressed during this sleep period after recent exposure (during wakefulness) to new stimuli.

What many scientists failed to realize is that REM sleep's contribution to memory consolidation may be a *byproduct* of its primary function, not the function itself. Indeed, the evidence that Capellini and colleagues (2008) provided indicates exactly this. Consequently, scientists who believe that learning is the function of REM sleep appear to be following the wrong path rather than the right

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one. If this is indeed the case, the excessive insistence on the learning hypothesis to explain REM sleep's function will prove to be an inappropriate trajectory for solving this mystery, responsible for guiding scientists away from the true answer instead of closer to it. To solve scientific mysteries, we must avoid embarking on paths that could easily lead to error. My warning (for whatever it is worth) is that seeking the answer to the role of REM sleep in learning is the wrong path.

6. CONCLUSIONS

The question "Does REM sleep serve the same purpose across different animal lineages?" remains an open problem (Peever and Fuller, 2017). However, based on my article, the answer to it becomes manifest. The *primary* function of REM sleep is the same for all organisms that possess this behavioral state. For any organism with a nervous system, supplanting sleep is (apparently) impossible. However, this is not the only way to reduce its high vulnerability. REM sleep solves this problem. *REM sleep is a necessary adaptation for every organism with a nervous system that, therefore, needs to sleep*. A mechanism like REM sleep—given its high biological relevance—would certainly become a priority and imperative in the course of biological evolution; it would inevitably spread widely among animals. And that is exactly what happened.

Since the functionally random genetic mutation that engendered the primeval occurrence of what we now describe as "REM sleep," non-random elimination ensured the widespread dissemination and persistence of this mechanism responsible for providing greater defense to the organism during the vulnerable N-REM sleep. REM sleep provided a substantial adaptive advantage to its bearers, as it compensates for the high vulnerability to which organisms are subjected during N-REM sleep. For this reason, the sentinel function of REM sleep has not only been conserved throughout evolution but has also undergone remarkable complexification, achieving a high level of efficiency as a protective mechanism. The biological importance of this mechanism is such that it may even have evolved independently.

REM sleep is regulated directly from information provided by all types of neural mappings: interoceptive (e.g., stress due to the presence of cortisol in the bloodstream), proprioceptive (e.g., muscle strength), and exteroceptive (e.g., exposure to an unknown environment). The information from these three varieties of neural mappings determines the parameters of REM sleep: the time invested in it, its latency, the duration of each episode, and its intensity (or density). In short, REM sleep is a biological mechanism that evolved to depend on any factors directly or indirectly related to protection and vulnerability (e.g., emotions; body weight, muscle strength, and the bilateral occurrence of N-REM sleep). Therefore, for REM sleep to be more precisely studied henceforward, any factors directly or indirectly related to the organism's protection or vulnerability should be isolated because they are confounding factors. Failing to separate the confounding factors that affect REM sleep parameters will lead to disparate results among studies. More precisely guiding future scientific investigations of sleep is one of the central contributions of my article.

The two main reasons for a scientific theory to be accepted as valid and robust are the level of corroboration it has and the number of attempts that failed to refute it. The theory I developed here passes this test. Throughout this article, I presented an extensive factual basis that solidly supports and corroborates the arguments I developed to demonstrate that the primary function of REM sleep is to act as a sentinel mechanism. Through the sentinel sleep theory, it is possible to accurately explain a substantial amount of disparate facts related to REM sleep; facts that come from numerous animals (e.g., zebrafish, cuttlefish, octopuses, drosophila, reptiles, nemestrina monkeys, chimpanzees, humans, rats, mice, birds, sheep, giraffes, cats, guinea pigs, lambs, ferrets, dolphins, belugas, orcas, porpoises, whales, and fur seals).

The arguments I developed to integrate the conceptual framework of the sentinel sleep theory are consistent with biological, embryological, homologous, phylogenetic, genetic, evolutionary, physiological, endocrinological, neurophysiological, neurobiological, neurochemical, neuropharmacological, ontogenetic, allometric, and even mathematical and statistical evidence (when I addressed the concepts of probability and complexity). Additionally, numerous attempts to

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refute it failed. Many pieces of evidence that seemed to offer some degree of refutation ended up revealing corroboration under scrutiny. (A lesson that must be considered in future research.)

In light of all the arguments I developed to compose the conceptual framework of the sentinel sleep theory and the numerous attempts that failed to refute it, it seems appropriate to state that the sentinel sleep theory is the most well-founded explanation ever presented for the biological function of REM sleep. No other can so robustly explain an enormous number of disparate facts pertaining to the domain of REM sleep. (I do not have space to demonstrate the flaws of every hypothesis already proposed to explain the function of REM sleep, but I can remind the reader what they are: hypotheses.)

I demonstrated here that the primary function of REM sleep has finally been unveiled. Through the sentinel sleep theory, I solved the great mystery—once existing—of the primordial biological function of REM sleep. For myself and other scientists, the task remains to improve the sentinel sleep theory in future works; whether by expanding its conceptual framework or testing the hypotheses contained within it. In this way, we can not only further corroborate it but also refine it or remove any flaws that I may have been unable to notice or resolve.

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