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

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The Sentinel Sleep Theory: Unweaving the Biological Function of REM Sleep

Running Title: Unweaving the Function of REM Sleep

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Abstract

The biological function of rapid eye movement (REM) sleep remains one of neuroscience's great mysteries. In this Theoretical Research Paper, I present the evolutionary theory that explains why REM sleep exists. I demonstrate that REM sleep functions to heighten brain alertness to significantly mitigate the high vulnerability inherent in non-REM sleep—especially in deep sleep. Every organism with a nervous system must undergo non-REM sleep, a necessity that accompanies a negative and potentially lethal consequence: higher risk of dying. Because non-REM sleep substantially reduces alertness and increases death risk, REM sleep evolved as an adaptive countermeasure, making it a *necessary adaptation* for any organism that must sleep. My theory—that is grounded in evolutionary biology and in voluminous empirical evidence—provides an eclectic and far-reaching explanatory and predictive capacity. This is because it integrates hundreds of pieces of evidence and generates numerous testable hypotheses that cross multiple scientific fields, such as genetics, phylogenetics, embryology, physiology, endocrinology, and immunology. In this Paper, I discuss **445 references**; most of which serve to support my theory. Based on the available evidence, *all empirically testable predictions that I was able to verify were corroborated*. Furthermore, the theory also resisted numerous other attempts at refutation. Here, I also challenge traditional views (e.g., REM sleep aids learning and memory), arguing that these apparent functions are byproducts rather than primary evolutionary drivers. Thus, this Basic Research Article may contribute to advance theoretical neuroscience and the way REM sleep research is planned and carried out.

Keywords: REM sleep; N-REM sleep; theory; biological function; evolutionary biology; historical narrative; sleep behaviour; vigilance; defence; coevolution

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Significance Statement

The biological function of REM sleep—one of the greatest unresolved problems in neuroscience—remains unsolved. The goal of this Theoretical Article is to provide a solution. I demonstrate that it functions as a safeguard for the organism during the vital yet perilous N-REM sleep; particularly during deep sleep. By integrating empirical evidence from diverse scientific fields, my theory unifiedly explains decades of scattered findings. Solving the function of REM sleep advances both theory and practice not only in neuroscience but also in related domains. From this work, my theory can be refined and will stimulate many new empirical and theoretical investigations.

Highlights

- REM sleep function is to reduce the vulnerability caused by N-REM sleep.
- REM sleep parameters are directly related to the body's protection or vulnerability.
- REM sleep is cyclic due to its protective function.
- REM sleep probably emerged as a brief awakening from N-REM sleep.
- REM density is a measure of the organism's alertness.

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AI Peer Review

My work, in its current form, has already undergone five formal reviews—conducted by humans—across three specialized scientific journals. Based on the reviewers' comments, I was able to substantially improve my work. However, given its present length, no editor is accepting it. For this reason, in order to ensure, as much as possible, that this new version is an extremely robust and scientifically valuable article, I decided to resort to AI-based review. It is the only tool I have left.

I conducted multiple rounds of review (in distinct chats) using *Google Gemini 2.5 Pro*. To minimize the chances of the model being biased in my favor, I required it to review meticulously and to look for any scientific errors or areas I could improve. I also specified specific evaluation criteria. Moreover, because the model allows users to obtain more useful responses by saving their preferences, I set it to be critical, not to always agree with me, and to reason rationally and logically.

Surprisingly, I obtained valuable reviews that allowed me to improve my work. Many of the comments provided by the model raised important issues that no human reviewer had pointed out in any of the five reviews. And, to be frank, I myself had not considered them. I requested a total of 35 reviews from the model, precisely to test different parameters and compare the assessments. I stopped requesting new reviews once the responses indicated that the article was already impeccable. I want to make it clear that I used this AI model exclusively to review my work, and not to write it. I am solely responsible for the writing of this work, which makes all of its claims entirely my responsibility.

1. Introduction

Imagine yourself as a wild animal. The place where you live and sleep—nature—is often dangerous and lethal. Therefore, you must constantly struggle to preserve your life. It turns out that your brain, when sleeping, drastically reduces your attention to the environment and your ability to respond to any dangers. Sleep is necessary, but it greatly hinders your arduous task of staying alive. Whatever the function (or functions) of this deep and restorative sleep may be, its weaknesses—reduced attention, diminished motor responsiveness, and compromised safety—cannot be eliminated. If your only option were to remain at the mercy of this necessary but dangerous sleep, your chances of survival would be drastically shortened. *But what if there were a neural mechanism capable of increasing your safety while you sleep?* Well, in that case, your chances of surviving despite the dangers of sleep would begin to look far more reasonable. Here, I propose that the neural state we call Rapid Eye Movement sleep (or REM sleep) is that mechanism. A simple—and effective—solution to a problem that could cost you your life.

But make no mistake. The simplicity of this mechanism does not translate into ease of unraveling it. Many scientists (especially psychologists, neuroscientists, and physicians) have been trying to discover what REM sleep is for more than 70 years, ever since Eugene Aserinsky and Nathaniel Kleitman (1953), in a landmark and foundational study, contributed to discover and report it. An even earlier work by Maria Denisova and Nicholai Figurin, originally published in Russian, in 1926, is the first study whose authors presented data on what is now recognized as REM sleep. It is available in an English translation (Denisova, 2024) and likewise establishes the authors as important figures in the discovery and description of REM sleep.

Why is it so difficult, at the moment, for scientists to describe what REM sleep is? The primary reason is that they still do not understand the biological function of this sleep state. What is the biological function of the REM sleep remains an unsolved question 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, 2017; Siegel, 2011). And when you do not know the function of a mechanism, you are limited to describing its physical and behavioral aspects. My objective in this Theoretical work is to unweave the evolutionary reason for the existence of REM sleep. I will begin by summarizing some of its fundamental characteristics.

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REM sleep is many things: a brain state, a behavior, a sleep state, a dreaming state, as well as a paradoxical state (Blumberg et al., 2020; Jouvet et al., 1959). 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, 2020; 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 similar 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; Tainton-Heap et al., 2021). This is why REM sleep was originally termed paradoxical sleep (Jouvet et al., 1959).

Especially in mammals and birds, both the REM sleep period and the non-REM (or N-REM) sleep period 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 (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 (or natural selection) 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, but 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); *brain warming function* (Wehr, 1992); *energy regulation* (Siegel, 2005); *sensorimotor integration* (Hong et al., 2009); and *defensive activation of the visual cortex* (Eagleman and Vaughn, 2021).

The Article that Snyder published in 1966 is particularly relevant for my discussion. 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 hypothesis into a *theory* of the function of REM sleep. This highlights the disparity between my work and Snyder's.

As not all scientists are scrupulous with terminology and fail to distinguish between the terms "hypothesis" and "theory," this needs to be considered. After all, 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). For this reason, 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).

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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 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. Furthermore, what makes a theory even better is its ability to solve *significant* conceptual and empirical problems (Laudan, 1977, pp. 13-14, 66, 70-71).

My scientific contribution with this Article is to present a comprehensive conceptual framework—supported by extensive empirical evidence—that will turn the sentinel *hypothesis* into the *theory* of sentinel sleep. This work is a Theoretical Article that aims to contribute to elucidating the function of REM sleep. To demonstrate the validity and robustness of my theory, I need to demonstrate that numerous hypotheses generated from it are true (i.e., factually verified). Therefore, I must present the theory in a way that allows it to be refuted or corroborated by testing the hypotheses derived from it.

2. Methodology

To develop and test the theory I am proposing for the biological function of REM sleep, I adopted the *hypothetico-deductive* (H-D) method—a classic scientific investigation procedure for constructing and justifying scientific theories (Mayr, 2001, p. 49; Nola and Sankey, 2014, p. 170; Popper, 2002, pp. 9-10). This method is based on starting from a theory or hypothesis, formulated conjecturally and still lacking justification, and then deriving logically deduced conclusions. The next step is for the scientist to test these conclusions (or predictions) against empirical evidence (Popper, 2002, pp. 9-10; Sokal and Bricmont, 2016, p. 72). The aim of this method is that, by testing specific predictions, the test determines the validity of the theory. If empirical evidence corroborates the deduced predictions, the theory gains support; if, on the other hand, empirical evidence contradicts those predictions, it also challenges (or falsifies) the theory (Mayr, 2001, p. 49; Popper, 2002, pp. 9-10; Sokal and Bricmont, 2016, p. 72).

To test a theory, we can employ the following procedures (Higgins, 2004; Laudan, 1977, pp. 13-14, 66, 70-71; Popper, 2002, pp. 9-10): (1) logically compare its specific predictions, in order to verify whether the theory is internally consistent; (2) examine the logical structure of the theory, in order to verify whether it possesses an empirical, scientific character, or whether it is a tautology; (3) compare it with other theories or hypotheses, in order to verify whether the newly proposed theory can effectively advance scientific knowledge once it is rigorously tested; (4) derive specific predictions, in order to test them against empirical evidence; (5) verify that it convincingly solves significant empirical and conceptual problems; (6) ensure that it encloses great explanatory power, being able to unify disparate empirical evidence; (7) ensure that it postulates only what is necessary (i.e., it needs to be parsimonious, following Ockham's razor, but without compromising its explanatory power). I followed these seven procedures to test the Sentinel Sleep Theory, ensuring its internal consistency, empirical character, scientific value, easily testable predictions (see Table 2), ability to satisfactorily solve significant empirical and conceptual problems, great explanatory power, and parsimony.

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I chose the H-D method due to the following methodological strengths:

1. **Easy to test empirically.** Allows the scientist to formulate hypotheses that generate specific predictions that can be corroborated or refuted by empirical evidence.
2. **Logical rigor.** The chain "theory ⇒ hypotheses ⇒ specific predictions ⇒ empirical testing" provides methodological rigor and allows us to critically evaluate predictions.
3. **Cumulative corroboration.** In the case of theories that resist successive attempts at refutation, they accumulate a greater number of corroborations, giving rise to new research and hypotheses that expand and refine the theoretical framework.
4. **Reproducibility.** Due to the specific predictions, other researchers can test the hypotheses to verify if they obtain the same results, thus strengthening the reliability of the results obtained.
5. **Experimental orientation.** The H-D method encourages scientists to design controlled and reproducible experiments. This also helps strengthen the reliability of the results obtained.
6. **Interdisciplinary flexibility.** With appropriate caveats (see below), we can apply this method in different scientific fields.

However, it is evident that, like any method, H-D approach entails its own limitations. Below, I list some of its main shortcomings:

1. **It is not immune to the sampling problem.** Although the core of the H-D method is deductive reasoning, it is not entirely devoid of induction (see below). This makes it necessary to address the challenges inherent in the representative sample that the scientist has to select from a population.
2. **It prevents us, in its strictest sense, from confirming a theory.** While excellent for falsification, the H-D method, taken literally, is exclusively negative: it allows us to be certain that certain theories are false, but prevents us from being certain (or as certain as possible) that a theory is true or probable (Marconi and Lakatos, 2021, p. 98; Sokal and Bricmont, 2016, p. 73). In other words, even if we have voluminous concordant results, they are incapable of confirming a hypothesis or theory. All they can do is indicate that we have not yet refuted it.
3. **It depends on auxiliary hypotheses.** False-negative results can result from flaws in experimental design or in data interpretation. Moreover, the H-D method also relies on assumptions concerning mechanisms, measuring instruments, laboratory conditions, *et cetera*.
4. **Without appropriate caveats, it is inadequate for certain sciences.** The H-D method is particularly unsuitable for testing probabilistic theories, a variety that, as Mayr (2001, p. 49) pointed out, includes the majority of biological theories. Any exceptions scientists may find to probabilistic theories do not necessarily indicate that those theories have been falsified. In the case of sciences such as evolutionary biology, for instance, it is difficult, and perhaps even impossible, to conclusively falsify certain theories (Mayr, 2001, p. 49).
5. **In its strict sense, it is inflexible to nuance.** For a strict Popperian, a theory or hypothesis can be refuted by a single reliable observation that refutes it (Laudan, 1977, p. 26; Marconi and Lakatos, 2021, p. 98; Sokal and Bricmont, 2016, p. 72). In other words, the H-D method—combined with radical falsificationism—demands that we categorically abandon a theory or hypothesis if a single well-conducted study yields negative results. This inflexibility may work for the exact sciences; however, as Mayr (2001, p. 50) pointed out, it is inappropriate for sciences such as biology—especially evolutionary biology.
6. **Multiple compatibility problem.** A single set of evidence may be compatible with more than one alternative hypothesis or theory, making it difficult for scientists to determine more easily which explanation is correct. For instance, scientists may use the fact that brain temperature increases during REM sleep (Ungurean et al., 2020; Wehr, 1992) to support the hypothesis that the function of REM sleep is to regulate brain temperature. However, this same evidence also supports the explanation that such warming occurs due to increased blood flow (Parmeggiani, 2007; Pastukhov and Ekimova, 2012). For this reason, the H-D method often requires that we rely on independent and additional evidence and arguments—a strategy I adopted in this Paper to deal with this limitation. (See Section 5, where I discuss the issue of brain warming during REM sleep.)

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Considering its limitations, I decided to complement the H-D method with additional strategies capable of compensating for them, thereby enhancing the rigor of my research. Equally important: I did not adopt the H-D method in its most extreme form, as such an approach is inappropriate in biology—particularly in evolutionary biology (I will return to this point henceforth). This methodological flexibility is necessary when dealing with the real world—a realm full of nuances. We must remember that scientific methods serve to help us elucidate the truth, but not to oppress us. Hereinafter, I detail the strategies I employed to circumvent the limitations of the H-D method. The first of these involves a set of techniques I employed to address the sampling problem, ensuring that it is both appropriate and representative. However, before delving into these techniques, I must explain why the H-D method also encompasses an inductive direction.

First, without inductive reasoning, it is difficult (and perhaps impossible) for the scientists to formulate general assertions from which they can then derive specific predictions. The general and specific hypotheses derived from the theory—as well as the theory itself—rely on reasoning that moves from particular cases (observational data, patterns of evidence, and analogies with known systems) to general conclusions. Second, the H-D method also depends on auxiliary premises, since one must include assumptions regarding mechanisms, measuring instruments, laboratory conditions, scientific procedures, *et cetera*. It turns out that these auxiliary premises also depend on generalizations—another inductive direction.

Moreover, although strict falsificationists (or radical Popperians) tend to avoid the issue, what one hopes to achieve in testing a theory is to arrive at the objective truth about the world—even if our knowledge of that truth is approximate or incomplete (Mayr, 2001, pp. 33-35, 46; Mayr, 2004, p. 11; Sokal and Bricmont, 2016, pp. 63-68). After all, this is the very aim of scientific inquiry (Mayr, 2001, p. 46; Mayr, 2004, p. 11; Sokal and Bricmont, 2016, pp. 63-68). If you disagree, recall that one of the central roles of science is to generate specific predictions upon which practitioners—physicians, engineers, psychologists, pharmacists, neurologists, *et cetera*—can confidently base their work (Sokal and Bricmont, 2016, p. 73).

Therefore, once we have tested a theory developed using the H-D method, we assimilate the positive results, in practice, inductively. This is because we test the specific predictions we deduced from the theory by observing a finite amount of empirical evidence that confirms (or refutes) those predictions. And if the tests corroborate the predictions (a positive outcome), we then extrapolate to assert that the theory possesses validity, corroboration, or truthfulness. That is, in the H-D method, we also generalize the results. In other words, after testing the specific predictions, we generalize from particular cases to assert that the theory has either been corroborated or falsified. (Note that strict Popperians would never agree that a theory has truth or that it has been confirmed [Popper, 2002, p. 10], but I am not one of them.)

Thus, since the H-D approach also entails an inductive direction, this naturally leads us to the sampling problem. That is, my theory requires a body of empirical evidence capable of adequately representing the target population—in this case, the full range of organisms exhibiting REM sleep. Therefore, two critical questions regarding the validity of my theory are: (1) What should the sample size be (i.e., the number of studies collected and critically analyzed) and (2) whether the sample size is sufficiently substantial and unbiased to support the generalizations I am proposing. After all, for us to seriously consider any generalization, the sample must be both voluminous and impartial. In the case of this work, this meant I needed to aggregate a sufficient and unbiased *corpus* of empirical evidence. Only then could my theory hold value, being able to accurately encompass and explain the primary function of REM sleep, including its finer details.

As for the first question—"what should the sample size be"—, it is evident that there is no magic number, no exact and universal answer. What I must do is clearly explain why I selected the references included here and what the inclusion and exclusion criteria were. To satisfy the criterion of sufficient sampling, I selected 445 bibliographic references. Considering that many Theoretical Articles typically contain between 100 and 250 sources, 445 is a large volume. However, volume *per se* is insufficient: large samples are not immune to bias. If properly selected, 445 references constitute

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a sufficient sample for a Theoretical Article, capable of conferring a robust and credible character to the theory. Therefore, I needed to adopt strategies to ensure an appropriate bibliographic *corpus*.

In order to minimize, as much as possible, any biases, I adopted the following techniques when selecting the references:

1. I prioritized Articles that could—directly or indirectly—refute the hypotheses I derived from my theory. Since numerous studies easily corroborated many of the hypotheses, they are present throughout the text as corroboration. However, during my literature search, I also identified numerous pieces of evidence that appeared, at first glance, to refute my hypotheses. However, upon scrutinizing this evidence, I found that they in fact corroborate the hypotheses. I devoted Section 5 to presenting this evidence, as well as my arguments explaining why it corroborates (rather than refutes) the hypotheses. Furthermore, many of the references in Section 5 are there because reviewers pointed them out as refutations of my theory. Again, after scrutinizing them, I was able to demonstrate that they corroborate the theory. (The very fact that I was able to explain—based on empirical evidence—all the apparent refutations proves the value of the theory.)

2. I included studies conducted in a wide range of animal species. This research technique minimizes bias by fulfilling the criterion to adequately represent biological variation. After all, since I aim to explain the biological function of REM sleep, the empirical evidence must sufficiently encompass the breadth of biological diversity. Some of the animals considered here include: 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. Still within the issue of diversity, the evidence must also include the two principal modes by which the brain rests: sleeping with both hemispheres simultaneously (bihemispheric sleep) and sleeping with only one hemisphere at a time (unihemispheric sleep). Finally, it also must include animals whose sleep includes distinct patterns of temporal organization: monophasic sleep (e.g., humans), biphasic sleep (e.g., birds and insects), and polyphasic sleep (e.g., birds, cats, and many wild mammals).

3. I triangulated between multiple sources of evidence, findings, and investigations. That is, I incorporated evidence from diverse scientific disciplines (interdisciplinarity). After all, if a theory is supported by evidence originating from various scientific fields, our confidence in it increases, since it is more likely to be true. For this reason, I included evidence from biology, embryology, homology, phylogenetics, genetics, evolutionary biology, physiology, neurophysiology, endocrinology, immunology, neurobiology, neurochemistry, neuropharmacology, ontogenetics, and other areas. With this interdisciplinary approach, I aim to ensure that the theoretical generalization I am proposing derives from an epistemologically robust bibliographic *corpus*, less susceptible to disciplinary or publication biases.

I conducted the research over a four-year period. I began selecting bibliographic sources on February 6, 2021. I amassed a substantial number of references in that initial year of investigation and, over the subsequent years, continued to curate an even larger number of them to discuss in my work. I stopped including new references on September 19, 2025. Developing my theory over four years allowed me to refine it progressively as I incorporated new empirical evidence to test it against the hypotheses. To locate peer-reviewed scientific publications, I searched in PubMed and Scopus using keywords related to the topics of my research. While I prioritized more recent empirical research, I also included older research. I did not set any limits on the dates of the research I selected for my work. Furthermore, much of the research I discovered and added to my work was due to its presence in Articles I had already selected.

As a selection criterion for sources, I included peer-reviewed publications (Scientific Articles and book chapters) and a few books written by renowned authors in their respective fields (e.g., Antonio Damasio, Ernst Mayr, Larry Laudan, Richard Dawkins, and Sidarta Ribeiro), with special emphasis on technical books. Regarding the scientific research, I included studies of any kind. For instance, Experimental Studies, Theoretical Papers, Comparative Studies, Reviews, Evolutionary Analyses, and Meta-Analyses. I selected the Articles especially from well-reputed scientific journals (e.g.,

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Current Biology, eLife, Journal of Sleep Research, Nature Communications, Nature Reviews Neuroscience, Nature, Neuron, PLoS Biology, PLoS One, PNAS, Scientific Reports, Sleep Medicine Reviews, Sleep Medicine, Sleep, and The Journal of Clinical Endocrinology & Metabolism). However, to avoid biasing my research by including only well-regarded journals, I also included research from lesser-known but equally important journals (what matters most, after all, is the quality of the individual Article). I excluded non-peer-reviewed Scientific Articles. That is, I did not included Preprints (note that I included two Reviewed Preprints, from eLife's new publishing model). Finally, although I prioritized English, especially for scientific Papers, I did not exclude sources based on language.

To address the limitation of reliance on auxiliary hypotheses in the H-D method, I assumed that no single study can falsify the theory or its hypotheses. This is because no study is immune to methodological or interpretive errors. Furthermore, data quality also matters. Therefore, whenever possible, I prioritized citing multiple sources—especially for the most critical arguments. In this way, by drawing on convergent results from replicated research (including via different methods), we can place greater confidence in the evidence. I also gave precedence to studies whose investigators imposed stricter variable controls and included one or more control groups. To minimize (as much as possible) interpretive mistakes on my part, I examined all evidence with the utmost thoroughness of which I am capable. I constructed arguments that align with the current body of evidence and was careful in considering alternative hypotheses.

To address the limitation posed by the H-D method's inappropriateness for certain sciences when used without proper caveats, I assumed that, in biology, exceptions are incapable of refuting certain theories (Mayr, 2001, p. 49). Therefore, even if I found any exceptions, they would be incapable of falsifying the hypotheses or the Sentinel Sleep Theory. Some scientists might consider that I adopt this assumption to immunize my theory against refutation, but they would be mistaken. Given that variation among individuals in a biological population is a cornerstone of evolutionary theory (Darwin, 1859; Dawkins, 2010a; Dawkins, 2015b; Mayr, 1982; Mayr, 2001), it would be naïve to presume that exceptions could overturn the theory I am proposing. Indeed, it is precisely because variation exists among animals that non-random elimination can remove those less well adapted (Darwin, 1859; Dawkins, 2010a; Dawkins, 2015b; Mayr, 2001; Mayr, 2004).

Moreover, we must also acknowledge that REM sleep may be more or less optimized in different species. By this, I mean we should *not* expect REM sleep to be enhanced—or potentiated—in the same manner across the entire animal kingdom. Certain species may exhibit particularly efficient REM sleep, while others may display less so. Let us consider an illustrative example below.

In Section 4, I will argue that REM sleep parameters—in a more optimized form of REM sleep—must adapt to the organism's current level of vulnerability or protection. After all, if an organism is already better protected, it is energetically inefficient to forgo adjusting REM-sleep parameters. Conversely, if the organism is more vulnerable, it becomes necessary to modify those parameters to further enhance the protective function that REM sleep can provide. The key point here is that this variant of REM sleep is what I call an *optimized version*. That does not imply that all animals possess REM sleep in an optimized form; variations in energetic efficiency may occur, and, consequently, such exceptions cannot falsify the hypotheses concerning the efficiency gains achieved by tuning sleep parameters when an animal already possesses a certain quantity of protection.

Another example is the hypothesis that REM sleep is dispensable in species that sleep with only one hemisphere at a time (see Section 4.3, where I discuss this issue in detail). Given that an active cerebral hemisphere during sleep can confer sufficient protection to the organism, REM sleep becomes unnecessary. However, this *does not* imply that all unihemispheric sleepers entirely lack REM sleep; vestiges may still be present. In this case, the optimized strategy would indeed be to eliminate REM sleep. Yet we must consider that non-random elimination may not have had sufficient time to achieve this energy-cost optimization. Therefore, I emphasize again: *we should expect exceptions*. Equally important, these exceptions cannot falsify the hypothesis that it is energetically

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efficient to eliminate REM sleep when the organism already benefits from the protection afforded by a continuously active cerebral hemisphere during sleep.

Note that the same approach I used to address the H-D method's inappropriateness for certain sciences also served to mitigate its lack of sensitivity to nuance. Finally, to manage the limitation of multiple compatibility of evidence, I drew on additional, independent lines of argument and data. By employing all of these strategies to circumvent the H-D method's limitations, I aim to have ensured greater methodological rigor in my work.

3. N-REM Sleep Is Highly Necessary, but Dangerous

Before presenting my theory, 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 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).

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4. The Sentinel Sleep Theory: Hypotheses and Predictions

Based on the voluminous empirical evidence I collected and analyzed, I developed various explanations to build the conceptual framework of the theory. I then derived hypotheses from these explanations and deduced their specific predictions. This review allowed me to analyze a vast number of empirical findings, compare existing hypotheses, examine their inconsistencies and flaws, and, finally, conclude specific assertions that together compose the full scope of my theory. After developing the entire theoretical framework of the Sentinel Sleep Theory, I turned to the scientific literature I collected to verify each of the hypotheses derived from the theory, with their specific predictions. If the predictions aligned with the empirical evidence, the hypotheses would be corroborated and the theory would gain strength. Otherwise, the hypotheses would be refuted, and consequently, the theory would lose strength. As we will see henceforth, the evidence indeed corroborates the predictions of my theory.

In its most general form, my theory is based on eleven facts and six inferences (see Table 1). The central tenet of the theory is that REM sleep serves to reduce the vulnerability of N-REM sleep. That is why it is from the context of the exacerbated vulnerability and the non-negotiable need for N-REM sleep that we can better understand the function of REM sleep. Another tenet of the theory is that REM sleep parameters (duration, latency, and density) should be based on any factors related to protection or vulnerability for the following reasons. (The teleological language I used serves only to explain more easily.)

When one is less vulnerable (or more protected), paying the high energy cost invested in the parameters of REM sleep is an unjustifiable strategy. A better strategy is to invest less energy in these parameters, allowing the organism to pay a lower price. In contrast, when one is more vulnerable (or less protected), paying the additional energy cost necessary to intensify the parameters of REM sleep is a justifiable strategy. After all, survival is at stake in this circumstance. In the first case, the high energy cost is unnecessary; in the second, it is utilitarian. Therefore, for REM sleep to be efficient both as a protective mechanism and in energy consumption, it needs to adapt to any circumstance that reduces or increases the organism's vulnerability.

Regarding the function of REM sleep, there needs to be a balance between investing energy in the protective function (a justified expense) and saving it when the organism is already more protected for some other reason (an unjustified expense). This implies that the parameters of REM sleep must be determined based on information provided by all varieties of neural maps—interoceptive, proprioceptive, and exteroceptive—, because they inform the brain of the current condition of the body and whether any sensory portal detected something capable of affecting the body of the organism, such as a threat (Kandel et al., 2013, p. 475).

Table 1. Structure of the Sentinel Sleep Theory.

	Facts and inferences*	References
Fact No. 1:	Emotions serve to ensure (directly or indirectly) the organism's survival; among other effects, they make the organism less vulnerable to predation, thereby contributing to its survival. (Emphasis on the fight-or-flight response.)	Chand et al., 2021; Chu et al., 2024; Damasio, 2003, pp. 34-35, 53; Damasio, 2015, pp. 51-53; Damasio, 2019, pp. 56-65; Dhabhar, 2018; Gazzaniga et al., 2016, p. 416; Moyne et al., 2022; Wolpert, 2008.
Fact No. 2:	N-REM sleep reduces both environmental alertness and emotional responsiveness, leaving the organism highly vulnerable to predation, thus risking its survival.	Anafi et al., 2019; Bear et al., 2016, p. 659; Capellini et al., 2008; Gazzaniga et al., 2016, p. 148; Moyne et al., 2022; Nath et al., 2017; Rattenborg and Ungurean, 2023.

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Fact No. 3:	N-REM sleep is a non-negotiable necessity for organisms with a nervous system, even if decentralized. In other words, N-REM cannot be eliminated in animals with a nervous system, as it is required for the brain to function properly and for the animal to survive.	Cirelli and Tononi, 2008; Jaggard et al., 2021; Kandel et al., 2021, p. 1097; Libourel and Herrel, 2016; Mignot, 2008; Nath et al., 2017; Urry et al., 2020, p. 1094; Vyazovskiy and Harris, 2013; Zimmerman et al., 2008.
Fact No. 4:	REM sleep currently involves the distinctive neural activation of regions responsible for alertness, attention, and emotional processing (e.g., cingulate cortex, amygdala, hippocampal formation, striatum, and thalamus). In other words, REM sleep is a state of heightened alertness, attention, and emotional responsiveness.	Braun et al., 1997; Caska et al., 2009; Goldstein and Walker, 2014; Peterson et al., 2002; Maquet et al., 1996; Maquet, 2000.
Fact No. 5:	Animals show greater alertness after waking up from REM sleep than after waking up from N-REM sleep. This allows a state of high readiness to defend itself from danger.	Kandel et al., 2013, p. 1157; Lima et al., 2005; Horner et al., 1997b; Reite et al., 1965; Snyder, 1966; Ribeiro, 2021; Tseng et al., 2022.
Fact No. 6:	REM sleep has specific characteristics that allow the animal to awaken quickly after detecting stimuli associated with predators or dangers (e.g., rapid and specific reactivity to predatory stimuli, rapid increase in pupil size, and rapid increase in the ability to move when detecting a predatory stimulus). Which ensures a successful defense against any events capable of threatening the animal's life.	Tseng et al., 2022.
Fact No. 7:	An organism's chances of survival depend on the presence of certain attributes that favor its survival. Thus, not all have the same chances (or probability) of survival.	Dawkins, 2015a, pp. 2-3, 6; Mayr, 2001, pp. 188-189; Mayr, 2009, p. 148.
Inference No. 1:	The attribute of momentarily increasing alertness, attention, and emotional responsiveness during sleep can contribute to the organism's survival.	Inference 1 is a logical consequence of facts 1 to 5.
Fact No. 8:	During REM sleep, brain metabolism increases by approximately 20% due to the heightened intensity of neural activity.	Bear et al., 2016, p. 660; Peever and Fuller, 2017.
Fact No. 9:	Neuronal activity consumes much more energy than neuronal silencing.	Kandel et al., 2013, p. 1157; Lennie, 2003.
Fact No. 10:	Non-random elimination is prolific in removing waste. Nothing so costly lasts for several million years unless it serves an important function.	McFadden, 2022, p. 268; Meredith et al., 2011.
Fact No. 11:	REM sleep has existed for several million years. Numerous lines of evidence indicate the possibility that REM sleep originated early in animal evolution, approximately 450 million years ago, that is, long before the branch of amniotes.	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.
Inference No. 2:	The high energy expenditure of REM sleep and its persistence over millions of years imply that it plays an important role in the survival of organisms that possess it. In other words, REM sleep requires a strong evolutionary justification.	Inference 2 is a logical consequence of facts 8 to 11.

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Inference No. 3:	The primary function of REM sleep is to compensate for the high vulnerability of N-REM sleep. REM sleep is an important biological mechanism that helps increase the organism's chances of survival—a strong evolutionary justification.	Inference 3 is a logical consequence of facts 1 to 11 and inferences 1 and 2.
Inference No. 4:	If (for some reason) the organism is more vulnerable and REM sleep parameters do not adapt to compensate for this vulnerability, the protective function of REM sleep will be less efficient, risking its survival.	Inference 4 is a logical consequence of inference 3.
Inference No. 5:	If (for some reason) the organism is more protected and REM sleep parameters do not adapt to save energy, the protective function of REM sleep will be energetically inefficient. It will spend resources that could be invested in survival, such as collecting food and seeking shelter.	Inference 5 is a logical consequence of inference 3 and facts 8 and 9.
Inference No. 6:	REM sleep parameters (duration, latency, and density) should depend on any factors that affect the organism's protection or vulnerability. They must adapt to conserve energy (when the organism is already protected due to another factor besides REM sleep) or to invest more energy to intensify the protective function (when the organism is vulnerable).	Inference 6 is a logical consequence of inferences 3, 4, and 5 and fact 10.

* "Inference" means a reasoning concluded from the facts listed in the table.

Given the central importance of the concept of *vulnerability* to my theory, I need to provide an operational definition. Before doing so, however, it should be noted that vulnerability and protection are two sides of the same coin. This is because stating that an organism is *more vulnerable* is equivalent to stating that it is *less protected*. Conversely, stating that it is *less vulnerable* is equivalent to stating that it is *more protected*. For this reason, throughout this work, I use the terms "protection" and "vulnerability" to refer to distinct and opposing sides of the same coin. Now, then, the definition.

I define *vulnerability* as a neural mapping of the bodily state (i.e., an internal representation) that informs the brain—either automatically or consciously—about the extent to which internal homeostasis has deviated in a given direction. This means that the content forming this state is always relative to the body of the organism in which it arises. It is content that describes and informs about the conditions of biological regulation within the organism, including the state of all internal operations and the current state of its organs (Damasio, 2019, p. 102). My mechanistic formulation anchors the concept of "vulnerability" not in the external environment (which also includes the environment external to the brain), but rather in a neuro-homeostatic representation relative to threat. In other words, it anchors vulnerability in the neural mapping of the bodily state in response to threat. This implies that, in organisms endowed with consciousness, vulnerability is the *feeling* corresponding to a specific homeostatic state that signals a real, potential, or even imagined threat.

Confronting certain stimuli and events can—actually or potentially—compromise homeostasis. It is due to this danger, even if only potential, that the organism enters an internal state that may appropriately be called *vulnerable* (or *less protected*, if we consider the other side of the coin). One example is an animal moving into a novel environment. Novelty may entail danger, and therefore it is important to respond to it with caution (Kahneman, 2011, p. 67). This is another way of stating that novelty can place the organism in an internal state of perceived vulnerability—even if unconsciously. And how could the organism feel vulnerable without an internal state informing it of exactly that? For this reason, all varieties of neural maps—interoceptive, proprioceptive, and exteroceptive—are central components in the task of mapping and informing the brain about vulnerability (or protection) relative to biological regulation. Such vulnerability may arise both from the internal milieu (the body itself) and from the external milieu (the environment).

Another illustrative example is depression. By leaving the organism with less energy and greater fatigue (Arias et al., 2020; Gazzaniga et al., 2016, p. 620; Stahl, 2002; Targum and Fava, 2011; Wolpert, 2008), such neurochemical disruptions of depression place the organism in a state of increased vulnerability. In other words, depression—negatively—deregulates internal homeostasis,

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contributing to the emergence of a state that I am calling *vulnerable*. And how are these neurochemical disruptions of depression signaled to the brain? This occurs through interoceptive information from the body, which is initially integrated in brainstem nuclei (Damasio, 2019, p. 87).

There are also cases in which the line between vulnerability and protection is more tenuous. For example, the risk of a threat engenders an internal state in which the organism perceives itself as vulnerable. However, vulnerability stems from the threat and its neural mapping, rather than from the neurochemical and physiological state of the body that arises in response to that threat. Therefore, to avoid ambiguities and misunderstandings regarding my operational definition, it is necessary to distinguish between the neural mapping that signals the threat and generates the state of vulnerability, on the one hand, and the neurochemical and physiological responses to that threat, on the other. When an organism perceives an external threat (such as a predator), vulnerability exists as a bodily state that derives directly from the mapping and recognition of that threat and the way in which it disrupts the organism's biological regulation. However, when we analyze the set of neurochemical and physiological responses to that predator, it becomes incorrect to state that this set constitutes a vulnerable state. Quite the opposite, it is precisely these responses that protect the organism's biological regulation and provide it with the chemical means necessary to have any chance of reacting to the threat.

Thus, as the threat escalates, the organism prepares for a fight-or-flight reaction; a response that, in fact, constitutes a mechanism that enhances the individual's protection by providing an internal chemical and physiological means capable of averting the threat and safeguarding life itself (Chand et al., 2021; Chu et al., 2024; Damasio, 2003, p. 53; Damasio, 2015, pp. 52-53; Dhabhar, 2018). Although threats may indeed arise from the external environment (e.g., a predator), it is within the body's internal environment that the signs of such threats are intercepted, mapped, and subsequently signaled to the central nervous system. And then mapped again. As a result, the body can then release a cascade of neurochemical and neurophysiological responses to address the threat and maintain homeostasis (Chand et al., 2021; Damasio, 2003; Damasio, 2019; Dhabhar, 2018). This is why it is necessary to dissociate the neural mapping that signals the threat, on the one hand, from the neurochemical and physiological responses to that threat, on the other.

It is evident that, in relation to a threat that has not yet been mapped by the body and brain, the organism will only register that threat and the homeostatic danger associated with it after interacting with it directly. Therefore, the example I provided above should be understood in the context of an organism that has already previously registered the threat.

Moreover, it is also important to distinguish the homeostatic effects of an adaptive and acute stress response (protective) from those of a pathological and chronic stress state (which generates a perception of vulnerability and exhaustion and is associated with depression). A stress response that persists over time ceases to be protective and becomes, in itself, a problematic, negative homeostatic deviation that induces vulnerability. This means that the quality (protective or vulnerability-generating) of the neuroendocrine stress response depends on both its duration and its context.

It should be noted that this definition of vulnerability explicitly requires distinguishing between an objective risk and a neurally represented risk. As will become clearer henceforth, *what matters for my theory and for the parameters of REM sleep is the neurally represented vulnerability, which does not necessarily correspond to an external and objective risk at all times*. Clarifying this is important to prevent any misunderstanding of what I am proposing. An organism can be objectively vulnerable without perceiving itself as vulnerable. This is because, in order to perceive it, the organism must not only already have a neural mapping of the threat but also intercept—at the present moment—some sensory cue of that threat. In the absence of an already mapped threat (in the form of sensory stimuli or cues), there would be, according to my definition, no internal representation of the homeostatic deviation related to that threat. The organism would therefore be subjectively "not vulnerable" according to the criterion I presented, even though it is in imminent danger.

To better understand what I am proposing, the fundamental questions we must ask are these: In relation to what is an organism more or less vulnerable, more or less protected? How are protection

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or vulnerability translated in neurochemical and neurophysiological terms? What reference does the brain use to determine whether the organism is vulnerable or protected? The answer to these questions is homeostasis. Homeostasis is the reference to which the brain turns in order to "know" whether the organism and its internal operations are safe and functional. More precisely, the answer lies in the homeostatic deviation—whether positive or negative—that an event, action, or stimulus causes in the mechanism of biological regulation. Events that disturb the body's regulated equilibrium range induce a state of vulnerability (if the deviation is negative), whereas events that contribute to or defend regulation generate a state of protection (if the deviation is positive). Put differently, certain events and stimuli may disrupt homeostatic regulation by increasing the threat to the organism's life. Conversely, certain events and stimuli may contribute to homeostatic regulation by enhancing the efficiency of biological regulation or by not interfering with it.

And whatever the homeostatic deviation may be, the body will respond with automatic chemical and neural responses that constitute distinct patterns we conventionally call *emotions* (Damasio, 2003; Damasio, 2019). The outcome of these emotions is to place the organism in a neurobiological condition that is directly or indirectly favorable to its survival and well-being (Chand et al., 2021; Damasio, 2003; Damasio, 2019). In sum, *an organism's vulnerability is intimately related to its biological regulation*.

Considering what I just proposed, it becomes evident why it is necessary to dissociate "vulnerability" from the threat itself (whether external or internal) and to anchor it instead in the mental representation that the brain creates of the body's state in relation to that threat or to any other homeostatic challenge. After all, it is this mental representation—a concrete neurobiological signal—that, once processed by the brain, generates consequences for biological regulation and can affect the circuits that regulate REM sleep. As we shall see in greater detail henceforth, any factor related to protection (or vulnerability) affects the circuits of REM sleep. What I am proposing is that an internal or external condition (e.g., greater muscular strength or exposure to a novel environment) is mapped by the brain (e.g., through proprioceptive or exteroceptive pathways), which then generates a neural representation (not necessarily conscious) of that condition. Finally, this representation modulates the circuits of REM sleep. We can schematize this as follows:

Physical Condition → Neural Mapping → Internal Representation → Sleep Modulation

This formulation provides a neuroanatomical substrate for my theory, postulating that the brain continuously integrates information from three distinct sources to assess vulnerability. These three sources are interoceptive, proprioceptive, and exteroceptive maps. Briefly, **interoception** maps the state of internal organs and the visceral milieu, conveying sensations such as fatigue, pain, hunger, or illness. **Proprioception** maps the state of the musculoskeletal system, providing information about muscle strength, tension, and body position relative to space. Finally, **exteroception** maps the perception of the external environment, particularly regarding its potential impact on the body, such as novelty or the presence of external threats (Damasio, 2012, p. 80). Concerning the locus responsible for integrating these three mapping systems, the neuroscience literature robustly identifies the insular cortex as a critical neural center for this function (Lu et al., 2016; Simone et al., 2025; Wang et al., 2019; Wei and Bao, 2013; Zhang et al., 2024; Zinn et al., 2024).

In sum, my approach to defining vulnerability allows the theory to connect a wide range of factors—from muscle strength to immune status—under a unifying principle: their impact on the organism's biological regulation. The definition I presented situates vulnerability not in the external environment, but in the internal mapping of the body's state in relation to threats. This framework explains why factors as diverse as a novel environment (an exteroceptive challenge), depression (an interoceptive deviation), or muscular weakness (a proprioceptive state) can all predictably modulate REM sleep parameters. All of these factors converge to generate an internal representation of negative homeostatic deviation, which the theory identifies as a state of vulnerability. Having now presented my operational definition of "vulnerability," I can proceed to discuss my theory proper.

For my theory to be validated or invalidated, I need to test the veracity of inferences 3 and 6 of Table 1. To do so, I need to list numerous hypotheses derived from my theory and test each of them.

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If the hypotheses are true, then my theory will also be true. Each of the items below—that summarize the Sentinel Sleep Theory—is a general hypothesis that forms the conceptual body of the theory. I dedicated one Section to each of these general hypotheses (4.1, 4.2, 4.3, 4.4, and 4.5). My goal in each Section is to explain and test the validity of the general hypotheses by verifying the specific hypotheses generated from the general hypotheses (see Table 2). In addition to verifying the veracity of the general and specific hypotheses, I will also demonstrate their factual foundation.

1. ***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 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.
2. ***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.
3. ***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.
4. ***The parameters of REM sleep depend on the organism's vulnerability.*** The time the brain invests in the REM period, the duration of each episode, the REM sleep latency (i.e., the period between the onset of sleep and the occurrence of the first REM sleep episode), and its density (or intensity), depend on the current vulnerability (or level of protection) of the organism's body. Something that is communicated to the brain by all varieties of mental mappings—interoceptive, proprioceptive, and exteroceptive. 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.
5. ***REM sleep probably 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 caused the organism to briefly wake up from N-REM 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.

Table 2. General and specific hypotheses derived from Sentinel Sleep Theory.

General hypotheses	Specific hypotheses	Status•	References
REM sleep is highly adaptive.	Hypothesis 1: organisms that have REM sleep during bihemispheric N-REM sleep have a better chance of surviving than those that do not have it.	Logically confirmed.*	McKinnon et al., 2022; Tseng et al., 2022. (And this Article itself.)
	Hypothesis 2: REM sleep is a necessary adaptation for organisms that sleep with both cerebral hemispheres.	Factually and logically confirmed.	Frank et al., 2012; Jaggard et al., 2021; Leung et al., 2019; Medeiros et al., 2021; Tainton-Heap et al., 2021; van Alphen et al., 2013. (And this Article

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			itself.)
	Hypothesis 3: given the high vulnerability of deep sleep (or quiet sleep), there was a strong evolutionary pressure for animals to develop vigilant sleep (or active sleep).	Factually and logically confirmed.	Frank et al., 2012; Jaggard et al., 2021; Leung et al., 2019; Medeiros et al., 2021; Tainton-Heap et al., 2021; van Alphen et al., 2013. (And this Article itself.)
	Hypothesis 4: predation played a significant role in the evolution of REM sleep.	Factually and logically confirmed.	Capellini et al., 2008; Tseng et al., 2022. (And this Article itself.)
REM sleep is cyclical due to its protective function.	Hypothesis 5: the presence of more than one REM episode offers more efficient protection, increasing the organism's chances of survival.	Logically confirmed.*	This Article itself. (See the arguments I developed in Section 4.2.)
The primary biological function of REM sleep is to reduce the vulnerability caused by N-REM sleep.	Hypothesis 6: REM sleep activates neural regions involved in threat detection.	Factually confirmed.	Bear et al., 2016, p. 670; Corsi-Cabrera et al., 2016; Damasio, 2003, p. 58; Dang-Vu et al., 2010; Davis and Whalen, 2001; Deboer et al., 1998; Eagleman and Vaughn, 2021; Gazzaniga et al., 2016, p. 95; Maquet et al., 1996; Nofzinger et al., 1997; Pignatelli and Beyeler, 2019; Rolls, 2019; Snyder, 1966; Sah et al., 2003; Whalen et al., 2013.
	Hypothesis 7: REM sleep activates neural regions involved in emotional processing.	Factually confirmed.	Bear et al., 2016, p. 670; Corsi-Cabrera et al., 2016; Damasio, 2003, p. 58; Dang-Vu et al., 2010; Gazzaniga et al., 2016, p. 95; Jumah and Dossani, 2022; Maquet et al., 1996; Nofzinger et al., 1997; Pignatelli and Beyeler, 2019; Rolls, 2019; Snyder, 1966; Sah et al., 2003; Whalen et al., 2013.
	Hypothesis 8: REM sleep activates neural regions involved in attention.	Factually confirmed.	Bear et al., 2016, p. 670; Corsi-Cabrera et al., 2016; Damasio, 2003, p. 58; Dang-Vu et al., 2010; Davis and Whalen, 2001; Deboer et al., 1998; Eagleman and Vaughn, 2021; Gazzaniga et al., 2016, p. 95; Jumah and

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			Dossani, 2022; Maquet et al., 1996; Nofzinger et al., 1997; Pignatelli and Beyeler, 2019; Rolls, 2019; Snyder, 1966.
	Hypothesis 9: REM sleep activates neural regions involved in pain processing.	Factually confirmed.	Devinsky et al., 1995; Kandel et al., 2013, p. 545; Maquet et al., 1996; Paus et al., 1997; Schneider et al., 2020; Wu et al., 2017; Xiao and Zhang, 2018; Xiao et al., 2021.
	Hypothesis 10: REM sleep is necessary when N-REM sleep occurs in both hemispheres.	Factually confirmed.	Fuchs et al., 2009; Lyamin et al., 2018; Mascetti, 2016; Rattenborg et al., 1999a; Rattenborg et al., 1999b; Rattenborg, 2006; Rattenborg et al., 2016.
	Hypothesis 11: REM sleep is dispensable when N-REM sleep occurs only in one hemisphere. (Dispensable in the sense that the organism already has sufficient protection provided by an active hemisphere. "Dispensable" does not mean that it cannot appear (with some duration) in some species. Non-random elimination may not have had time to remove REM sleep in organisms in which it makes no sense.)	Factually confirmed.	Fuchs et al., 2009; Lyamin et al., 2008; Lyamin et al., 2018; Mascetti, 2016; Mukhametov, 1995; Mukhametov et al., 1977; Mukhametov et al., 1988; Rattenborg et al., 1999a; Rattenborg et al., 1999b; Rattenborg, 2006; Rattenborg et al., 2016.
	Hypothesis 12: in organisms that *only* have unihemispheric sleep, REM sleep is useless. Either it does not exist or there are some remnants due to the evolutionary past.	Factually confirmed.	Lyamin et al., 2008; Lyamin et al., 2018; Mukhametov, 1995; Mukhametov et al., 1977; Mukhametov et al., 1988; Shurley et al., 1969.
	Hypothesis 13: in organisms that possess both bihemispheric and unihemispheric sleep, suppression of REM sleep during unihemispheric sleep will generally not accompany REM sleep rebound.	Factually confirmed.	Lyamin et al., 2018.
	Hypothesis 14: in organisms that possess both bihemispheric and unihemispheric sleep, suppression of REM sleep during unihemispheric sleep can rarely cause a small rebound of REM sleep.	Factually confirmed.	Lyamin et al., 2018.

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	Hypothesis 15: upon awakening from REM sleep, the body presents full alertness and sensory and motor efficiency.	Factually confirmed.	Kandel et al., 2013, p. 1157; Lima et al., 2005; Horner et al., 1997b; Reite et al., 1965; Snyder, 1966; Ribeiro, 2021; Tseng et al., 2022.
	Hypothesis 16: REM sleep makes waking up easier.	Factually confirmed.	Ermann et al., 1993; Ficca et al., 2004; Klemm, 2011; Ribeiro, 2021; Tseng et al., 2022.
	Hypothesis 17: spontaneous awakenings occur more frequently during, or shortly after, REM sleep.	Factually confirmed.	Ermann et al., 1993; Ficca et al., 2004; Klemm, 2011; Ribeiro, 2021; Tseng et al., 2022.
	Hypothesis 18: REM sleep does not suffer a "negative rebound". Sleeping more one night increases REM sleep time, but does not reduce REM sleep time in the subsequent night.	Factually confirmed.	Le Bon, 2020; Ribeiro, 2021.
	Hypothesis 19: REM sleep suppression does not compromise any neural function other than protective function.	Factually confirmed.	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, 2021.
The parameters of REM sleep depend on the organism's vulnerability.	Hypothesis 20: The parameters of REM sleep—its duration, latency to the first episode, and density—depend on information provided by all varieties of neural maps: interoceptive, proprioceptive, and exteroceptive.	Factually confirmed.	Anderson and Bradley, 2013; Baglioni et al., 2016; Berger and Riemann, 1993; Borniger et al., 2018; Chamorro et al., 2014; Driver et al., 1994; Driver and Taylor, 2000; Elrokhsi et al., 2020; Fang et al., 1995; Gutwein and Fishbein, 1980a; Gutwein and Fishbein, 1980b; Hague et al., 2003; Hrozanova et al., 2020; Imeri and Opp, 2009; Kishi et al., 2023; Kitamura et al., 2021; Kiyono et al., 1981; Krueger and Majde, 1994; Liu et al., 2008; McCarley, 1982; Mirmiran et al., 1982; Myllymäki et al., 2011; Nair et al., 2022; Palagini et al., 2013; Pollmächer et al., 1993; Riemann et al., 2020; Riemann and

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		Berger, 1989; Schmid et al., 2008; Seol et al., 2022; Smith, 1996; Steiger and Pawlowski, 2019; Steiger et al., 2013; Tagney, 1973; Theorell-Haglöw et al., 2010; Toth and Krueger, 1988; van Gool and Mirmiran, 1986; Wichniak et al., 2017; Zapalac et al., 2024.
Hypothesis 21: total REM sleep time is shorter in organisms with higher body fat.	Factually confirmed.	Chamorro et al., 2014; Elrokhsi et al., 2020; Liu et al., 2008; Theorell-Haglöw et al., 2010.
Hypothesis 22: the latency to the first REM episode is greater in organisms with greater body fat.	Factually confirmed.	Chamorro et al., 2014; Liu et al., 2008.
Hypothesis 23: the density (or intensity) of REM sleep is lower in organisms with greater body fat.	Factually confirmed.	Liu et al., 2008.
Hypothesis 24: non-obese sedentary individuals have more REM sleep time compared to more active individuals.	Factually confirmed.	Hague et al., 2003; Seol et al., 2022; Zapalac et al., 2024.
Hypothesis 25: non-obese sedentary individuals have a shorter latency to the first REM episode compared to more active individuals.	Factually confirmed.	Hague et al., 2003; Seol et al., 2022; Zapalac et al., 2024.
Hypothesis 26: non-obese sedentary individuals have greater REM sleep density compared to more active individuals.	Not confirmed nor refuted.❖	
Hypothesis 27: total REM sleep time is shorter in organisms with greater muscle strength or in those who exercised recently.	Factually confirmed.	Brand et al., 2010; Driver et al., 1994; Driver and Taylor, 2000; Hague et al., 2003; Hrozanova et al., 2020; Kubitz et al., 1996; Myllymäki et al., 2011; Seol et al., 2022; Youngstedt et al., 1997; Zapalac et al., 2024.
Hypothesis 28: the latency to the first REM episode is greater in organisms with greater muscular strength or in those who exercised recently.	Factually confirmed.	Driver et al., 1994; Driver and Taylor, 2000; Hague et al., 2003; Seol et al., 2022; Youngstedt et al., 1997; Zapalac et al., 2024.

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Hypothesis 29: REM sleep density is lower in organisms with greater muscular strength or in those who exercised recently.	Not confirmed nor refuted.❖	
Hypothesis 30: recent exposure to a new environment (or new stimuli) increases REM sleep time.	Factually confirmed.	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.
Hypothesis 31: recent exposure to a new environment (or new stimuli) reduces the latency to the first REM episode.	Factually confirmed.	Mirmiran et al., 1982; Nair et al., 2022.
Hypothesis 32: recent exposure to a new environment (or new stimuli) increases REM sleep density.	Not confirmed nor refuted.❖	
Hypothesis 33: depression increases REM sleep time.	Factually confirmed.	Anderson and Bradley, 2013; Baglioni et al., 2016; Berger and Riemann, 1993; Cheeta et al., 1997; Palagini et al., 2013; Steiger and Pawlowski, 2019; Steiger et al., 2013; Vogel et al., 1990; Wichniak et al., 2017.
Hypothesis 34: depression reduces the latency to the first REM episode.	Factually confirmed.	Anderson and Bradley, 2013; Baglioni et al., 2016; Berger and Riemann, 1993; Cheeta et al., 1997; Lam, 2006; McCarley, 1982; Palagini et al., 2013; Riemann and Berger, 1989; Steiger and Pawlowski, 2019; Steiger et al., 2013; Vogel et al., 1990; Wichniak et al., 2017.
Hypothesis 35: depression increases REM sleep density.	Factually confirmed.	Anderson and Bradley, 2013; Baglioni et al., 2016; Berger and Riemann, 1993; Friess et al., 2004; Kishi et al., 2023; Lam, 2006; McCarley, 1982;

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		Palagini et al., 2013; Steiger and Pawlowski, 2019; Steiger et al., 2013; Wichniak et al., 2017.
Hypothesis 36: stress reduces REM sleep time or suppress it.	Factually confirmed.	Feng et al., 2023; Friess et al., 2004; Hrozanova et al., 2020; Papale et al., 2005; Schmid et al., 2008.
Hypothesis 37: stress increases the latency to the first REM episode.	Factually confirmed.	Feng et al., 2023; Friess et al., 2004; Goldberg et al., 2020; Goodenough et al., 1975.
Hypothesis 38: stress increases REM sleep density.	Factually confirmed.	Barbato et al., 1994; Barbato, 2023; Feinberg et al., 1987; Feng et al., 2023; Ficca et al., 2004; Goodenough et al., 1975; Lauer et al., 1987; Rodenbeck and Hajak, 2001.
Hypothesis 39: when other factors remain unchanged, combined vulnerabilities produce more intense effects on REM sleep parameters.	Factually and logically confirmed.	Anderson and Bradley, 2013; Arias et al., 2020; Berger and Riemann, 1993; Kishi et al., 2023; McCarley, 1982; Palagini et al., 2013; Ribeiro, 2021; Riemann et al., 2020; Riemann and Berger, 1989; Schmid et al., 2008; Stahl, 2002; Steiger and Pawlowski, 2019; Steiger et al., 2013; Suchecki et al., 2012; Targum and Fava, 2011; Wichniak et al., 2017; Wolpert, 2008. (And this Article itself.)
Hypothesis 40: when other factors remain unchanged, combined protections produce more intense effects on REM sleep parameters.	Factually and logically confirmed.	Chand et al., 2021; Feinberg et al., 1987; Oken et al., 2006. (And this Article itself.)
Hypothesis 41: REM sleep density is a measure of the organism's level of alertness. Which is directly related to the amount of stress, because stress reduces the organism's vulnerability by increasing vigilance.	Factually and logically confirmed.	Barbato et al., 1994; Barbato, 2023; Chand et al., 2021; Feinberg et al., 1987; Lam, 2006; Oken et al., 2006. (And this Article itself.)
Hypothesis 42: bodily immature neonates have more REM sleep compared to bodily mature neonates.	Factually confirmed.	Balzamo et al., 1972; Blumberg, 2015; Chen et al., 2022; Cui et al., 2019; Grigg-Damberger and

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			Wolfe, 2017; Jouvet-Mounier et al., 1970; Reite et al., 1976; Ruckebusch et al., 1977; Szeto and Hinman, 1985; Thurber et al., 2008.
	Hypothesis 43: bodily immature neonates have a shorter latency to the first REM episode compared to bodily mature neonates.	Not confirmed nor refuted.❖	
	Hypothesis 44: bodily immature neonates have greater REM sleep density compared to bodily mature neonates.	Not confirmed nor refuted.❖	
	Hypothesis 45: in premature births, REM sleep is even more abundant than in newborns.	Factually confirmed.	Chen et al., 2022; Graven and Browne, 2008; Mizrahi, 2004; Okawa et al., 2017; Werth et al., 2017.
	Hypothesis 46: in premature births, the latency to the first REM episode is even shorter than in neonates.	Not confirmed nor refuted.❖	
	Hypothesis 47: in premature births, REM sleep density is even greater than in newborns.	Not confirmed nor refuted.❖	
REM sleep evolved from a brief awakening from N-REM sleep.	Hypothesis 48: REM sleep emerged as an error in the neurobiological mechanisms that control the transition from sleep to wakefulness, causing a brief awakening from N-REM sleep.	Logically plausible.∔	This Article itself. (See the arguments I developed in Section 4.5.)
	Hypothesis 49: primeval REM sleep evolved from a brief awakening to an ease of awakening.	Logically plausible.∔	This Article itself. (See the arguments I developed in Section 4.5.)
	Hypothesis 50: after evolving into an ease of awakening, primeval REM sleep began to include more than one REM episode.	Logically plausible.∔	This Article itself. (See the arguments I developed in Section 4.5.)
	Hypothesis 51: intense muscle atonia appeared after primeval REM sleep began to include more than one REM episode.	Logically plausible.∔	This Article itself. (See the arguments I developed in Section 4.5.)

• "Factually confirmed" means that there is empirical support in scientific literature for the specific hypothesis. "Logically confirmed" means that, due to arguments constructed with the best tools of logical reasoning, we have justification to support the conclusion. * The logical veracity of hypotheses 1 and 5 is a consequence of all empirically confirmed hypotheses, as well as the arguments I presented here for the protective function of REM sleep. ❖ Requires more research. ∔ We will never be able to test these hypotheses empirically.

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Of the 51 specific hypotheses I listed in Table 2, four of them are logically plausible and can never be tested empirically (hypotheses 48, 49, 50, and 51), seven could not be confirmed or refuted due to lack of studies (hypotheses 26, 29, 32, 43, 44, 46, and 47), two were confirmed logically (hypotheses 1 and 5), and 38 were empirically confirmed. That is, based on the available evidence, *all the empirically testable hypotheses that I could analyze in this Article were corroborated; not a single hypothesis was refuted*. The following Sections (4.1, 4.2, 4.3, 4.4, and 4.5) detail the 38 empirically confirmed hypotheses, as well as the hypotheses that I logically tested and those that are logically plausible.

4.1. REM Sleep Is Highly Adaptive

Oftentimes, it is the information concerning the circumstances and the specific moment in evolutionary history that provides the most crucial clues for understanding the adaptive utility of a trait or behavior (Snyder, 1966). It is for this reason that it is so necessary for me to address N-REM sleep. 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 3) the importance of N-REM sleep. 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; 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), 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 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.

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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). 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 properly educated 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*.

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? What if the organism's brain, during sleep, underwent considerable neural activation (particularly in regions related to attention, detection of dangerous stimuli, and 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 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.

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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 at least some reptile species 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. (Remember that our estimate of the origin of sleep is that it is over 543 million years old. In other words, the origin of sleep analogous to N-REM sleep remains earlier than the origin of sleep analogous to REM sleep.)

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 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—due to predation—throughout the course of evolution for the organisms to develop mechanisms that enable the transition from quieter sleep to a more active (or protective, as I am arguing) sleep.

This evidence corroborates *hypothesis 3* (see Section 4.5 for an in-depth discussion regarding the pressure to develop a way to cope with the high vulnerability of N-REM sleep). They also corroborate *hypothesis 2*, 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).

It is crucial to emphasize that the evolution of sleep architecture was not driven exclusively by predation. Other ecological and physiological factors, such as metabolic rate and foraging strategy, also exerted significant selective pressures. However, these factors do not operate in isolation; they

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are deeply interconnected. For instance, small animals with high metabolic rates are often compelled to adopt polyphasic sleep patterns due to the need to forage more frequently. This same strategy, although dictated by energetic demands, fragments sleep periods and may, secondarily, reduce the window of continuous vulnerability to predation.

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), thus confirming hypothesis 4. Regarding hypothesis 1, for ethical reasons, we cannot confirm it in the laboratory. But if I demonstrate throughout this Article that the primary function of REM sleep is to reduce the vulnerability of N-REM sleep, then the truth of hypothesis 1 will be a logical consequence of its function. Despite this difficulty, the findings of Tseng and colleagues (2022) can be considered empirical and logical confirmations of hypothesis 1. (I will discuss this Article in Section 4.3.)

4.2. REM Sleep Is Cyclical Due to Its Protective Function

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.

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 a priori 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. If the function of REM sleep is to reduce the vulnerability of N-REM sleep, the truth of Hypothesis 5 is a logical consequence of this function.

A pertinent issue that I need to address concerns the duration of REM sleep in relation to its protective function. In each N-REM/REM cycle, the N-REM period lasts longer than the REM period (Brinkman et al., 2023; Kandel et al., 2013, p. 1143). Does this undermine my theory? Can REM sleep still be considered adaptive? The reason I devoted time to discussing the importance of N-REM sleep is precisely to demonstrate that it is a non-negotiable necessity. It cannot be dispensed with. As I argued in Sections 3 and 4.1, eliminating N-REM sleep is (apparently) impossible and non-adaptive; if it were relatively easy to eliminate it, such a characteristic would have spread, since it would certainly be adaptive. It logically follows that drastically reducing N-REM sleep time is also not an adaptive possibility—and that is what would need to happen for REM sleep to predominate. In other words, because N-REM sleep is so imperative, it consequently predominates during rest periods. It must predominate in order to fulfill its function. The result is that, with N-REM sleep necessarily occupying most of the sleep period, less time remains for REM sleep. However, this in no way undermines my theory.

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For those who believe that the short duration of REM sleep impedes the sentinel function, it is worth recalling that 50% of an eye like ours is better than 35% of it. More importantly: 1% of that eye is better than having no eye at all (Dawkins, 2015b, pp. 112-113). During sleep, an organism having *some* period in which the brain is distinctly more responsive to potentially harmful stimuli is better than *having no* such period at all. Even if the organism remains vulnerable for most of the rest period (due to N-REM sleep), possessing a state of heightened alertness—even if brief—is still advantageous. *The fact that REM sleep does not predominate over N-REM sleep does not imply that REM sleep is useless or non-adaptive*.

It is an absolutist line of thinking to assume that, in order to be functional, REM sleep must last longer than N-REM sleep. Arguing that REM sleep can only be adaptive if it predominates in duration is analogous to claiming that an eye can only be adaptive if it already possesses, for instance, a well-developed lens, optic nerve, and retina. Therefore, the presence of the sentinel function, even if only for a short period, is more adaptive than its absence. Indeed, this is why, as I argued in this Section, REM sleep occurs repeatedly throughout the rest period.

Now that I explained the *why*, it is worth explaining the *how*. I need to discuss how the requirement for periodic vigilance integrates with the neural mechanisms that govern the ultradian cycle, such as the *flip-flop switch model*, which involves REM-on and REM-off neuronal populations in the brainstem (Lu et al., 2006; Saper et al., 2010). The Sentinel Sleep Theory implies that this switch is a system modulated by a constant assessment of vulnerability (or protection) carried out by the brain. This is a profound implication, as it allows us to evaluate the progression of the sleep cycle in an unprecedented way. At the onset of rest, deep N-REM sleep (the most vulnerable state) predominates; as rest progresses, REM sleep episodes become progressively longer and more frequent (Patel et al., 2024; Ribeiro, 2021; Shrivastava et al., 2014). Why does this happen? At least two answers are possible—and one does not exclude the other.

1. The homeostatic pressure of N-REM sleep progressively reduces. As the homeostatic pressure of N-REM sleep is satisfied (especially at the onset of rest), this allows for a greater "budget" to be allocated to the vigilance mechanism that is REM sleep. This explains why the duration and frequency of REM sleep progressively increase. After all, once the organism has invested the necessary time in deep sleep, it then becomes possible to invest more energy and time in the sentinel function to better safeguard the organism.

2. The activation of neural regions associated with threat detection interferes with REM sleep. Just as the electrical activation of a neural region impacts the memories that reverberate within the mental stream, the reverberating memories can also recruit or modulate the activity of neural regions that would otherwise be less active. That is, it is possible for reverberant activity to affect active, inactive and less active areas. Due to the activation of regions such as the amygdala, the reverberant content may begin to include negatively valenced memories. In other words, the brain may begin to reverberate memories from the mental database (previously stored) that are related to threatening stimuli. The limbic system then reacts to the emotionally negative content of these dreams. When the content of a dream is threatening, the amygdala (which is active) contributes to engendering a state of vigilance that prepares the organism for a potential awakening. That is, the brain may "believe" that the internal "threat"—in the form of electrically active past memories—is in fact an external threat arising from the environment.

In other words, throughout rest, the frequent activations of the amygdala and other regions related to threat detection may trigger a false sense of danger in the organism due to the contents reverberating in its mind. This is particularly the case because, as I argue in Section 6, the memories that reverberate most often are selected based on a neurobiological mechanism that prioritizes those that are more emotionally salient—especially those negatively valenced. Consequently, this virtual danger ends up affecting the parameters of REM sleep: increasing its duration, frequency, and density. In sum, *the internal world of dreams often contains emotionally charged and threatening scenarios. Consequently, the danger that comes from within—from the brain itself—can also affect REM sleep or, more*

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precisely, all of its parameters. And it is evident that, if this internal danger reaches a certain threshold, the organism will awaken.

The perspective I just presented (regarding the flip-flop switch) has direct clinical applications. In disorders such as insomnia, in which hypervigilance is a central feature (Kalmbach et al., 2018), the sleep architecture of insomniac patients may reflect a system pathologically inclined toward the detection of potential threats, with a flip-flop switch excessively sensitive to any signals of vulnerability. The result is fragmented sleep and difficulty in initiating or maintaining deep sleep, both typical characteristics of insomniacs (Kalmbach et al., 2018; Yan et al., 2023). Indeed, it is a highly replicated finding that excessive vigilance and the increase of micro- and macro-arousals in insomniac patients are directly related to REM sleep (Feige et al., 2008; Feige et al., 2023; Morin et al., 2015; Riemann et al., 2012; Riemann et al., 2023). Further evidence for the argument of a flip-flop switch overly sensitive to signals of vulnerability or danger is provided by studies showing that, in insomniacs, sleep exhibits an emotional bias toward negatively valenced stimuli (e.g., Baglioni et al., 2010; Baglioni et al., 2014).

4.3. The Primary Biological Function of REM Sleep Is to Reduce the Vulnerability Caused by N-REM Sleep

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), one of 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, 2021). 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

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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 (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 *hypothesis 6* and, therefore, *hypothesis 20*. 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. Thus, the theory proposed here (the Sentinel Sleep Theory) offers a more empirically grounded explanation for the high activation observed in the primary visual cortex during REM sleep than the defensive activation hypothesis by Eagleman and Vaughn (2021). Furthermore, the defensive activation hypothesis lacks robust empirical support. Notably, Knopper and Hansen (2023) pointed out that recent and important studies do not fully agree with the data that Eagleman and Vaughn (2021) provided to support the defensive activation hypothesis.

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), corroborating *hypotheses 6, 7, and 8*.

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

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processing pain are activated during this sleep period. Thus, the activation of cingulate cortex during REM sleep also corroborates *hypothesis 9*.

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 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. 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; Goleman, 2012, p. 39). 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). The amygdala occupies a privileged position in the brain. When it detects a threat, it quickly dominates the rest of the brain—especially the prefrontal cortex—to project the organism's attention onto whatever the threat is (Goleman, 2012, p. 39).

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. In fact, based on the function of detecting threats and triggering physiological and behavioral responses to danger, We can predict that the amygdala plays an important role in the regulation of REM sleep. Based on this prediction, a strong correlation between REM sleep and the intense activation of the amygdala is expected.

Evidence supports this prediction: the amygdala plays an important role in the regulation of REM sleep (Tang et al., 2005) and 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

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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 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 of this previous evidence regarding the characteristics of the amygdala make it highly suitable for the protective function performed by REM sleep, and supports *hypotheses 6, 7, and 8*. Thus, it is 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 anyone 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.

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

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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 *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.

Another brain structure whose activation makes sense is the thalamus. After all, 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). This structure is so important for attention that damage to higher-order thalamic regions—such as the mediodorsal nucleus and the pulvinar nucleus—can result in severe attention deficits (e.g., Exner et al., 2001; Saalmann and Kastner, 2015). Therefore, considering *hypothesis 8*, the thalamus should be active during REM sleep. Functional neuroimaging demonstrates that during the N-REM period, the thalamus is inactivated. However, during REM sleep, the thalamic nuclei are activated (Jan et al., 2009; Maquet, 2000), confirming *hypothesis 8*.

Additional evidence related to alertness and vigilance comes from the Hypothalamic–Pituitary–Adrenal (HPA) axis; a system that connects the hypothalamus, the pituitary gland, and the adrenal glands, and is responsible for regulating the release of hormones that play a crucial role in the physiological stress response (primarily cortisol and adrenaline) (Goleman, 2012, p. 40). For the protective function of REM sleep to operate, this physiological state must be associated with activation of the HPA axis. After all, one of the central functions of the HPA axis is to keep the organism alert and vigilant (Buckley and Schatzberg, 2005; Chu et al., 2024; Nicolaides et al., 2020; P and Vellapandian, 2024). The evidence shows that REM sleep is indeed associated with HPA axis activity, and that they mutually influence one another (Antonijevic, 2008; Chrousos and Gold, 1992; Mandell et al., 1966; Vgontzas et al., 1997). According to my theory, this influence operates bidirectionally for the following reason: REM sleep, due to its protective function, needs to be influenced by the HPA axis given that this axis maintains a state of alertness and vigilance. In turn, REM sleep can affect the HPA axis because of the content the brain reverberates during the REM period. After all, evoked memories also trigger emotions. And if those evoked memories—due to their emotional content—demand action from the HPA axis, then it will be activated.

To continue with the factual foundation of the Sentinel Sleep Theory, I will now analyze unihemispheric sleep. This will allow me to test, through a "natural experiment", a key prediction of my theory. 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. This evidence provides powerful support for my claims that REM sleep is a necessary adaptation specifically for organisms undergoing bihemispheric sleep, where both hemispheres are simultaneously vulnerable.

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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, 2021). Their brains can sleep using only one cerebral hemisphere at a time (Mascetti, 2016; Ribeiro, 2021). In certain environments and niches, if the organism's brain were subjected to N-REM 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, 2021).

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, 2021). 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 N-REM sleep state predominate in the other (as indicated by low-frequency and high-amplitude waves) (Konadhode et al., 2016; Mascetti, 2016; Ribeiro, 2021). 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, 2021). This characteristic allows cetaceans to maintain constant movement, ensuring periodic surfacing for breathing (Mascetti, 2016; Ribeiro, 2021). Repeated studies on cetaceans failed to find any amount of REM sleep in these animals (Lyamin et al., 2008; Lyamin et al., 2018; Mukhametov, 1995; Mukhametov et al., 1977; Mukhametov et al., 1988). The list of parameters used to analyze sleep in bottlenose dolphins (the most studied cetacean species) includes brain temperature, respiratory rate, electrocardiogram, eye movements, electromyogram, lateral geniculate bodies and hippocampi, and the electroencephalogram of the cortical hemispheres (Lyamin and Siegel, 2019; Mukhametov and Lyamin, 1997). Only a single study in the literature demonstrated the presence of REM sleep in the pilot whale, which lasted only 6 minutes and occurred only once (Shurley et al., 1969). Thus, cetaceans either have a negligible amount or no REM sleep (Lyamin et al., 2008). Therefore, these evidences corroborate *hypotheses 11 and 12*.

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, 2021). 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 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. Furthermore, for REM sleep to exist in animals that sleep with only one hemisphere would be a huge waste of energy.

As we know, cetaceans evolved from terrestrial mammalian ancestors (Coombs et al., 2022; Mancia, 2018). When we combine this fact with *all* the arguments and evidence I presented here to demonstrate the function of REM sleep, it becomes evident that the ancestors of modern cetaceans gradually lost REM sleep because it became dispensable for the reasons I elucidated above. Therefore, it is clear that current cetaceans may exhibit remnants of REM sleep from their ancestors—perhaps too subtle to be detected with the methods currently employed. However, if we could travel back in time and analyze the sleep of cetacean ancestors, we would observe increasing remnants of REM sleep until it became evident, as we approached the branching point from terrestrial mammals,

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manifesting as the distinct REM sleep known in terrestrial mammals. Therefore, for *hypotheses 11 and 12* to be corroborated, it is not necessary for cetaceans to be entirely devoid of REM sleep, as remnants of it are expected due to their evolutionary history.

Unlike cetaceans, other animals have both unihemispheric and bihemispheric sleep. Birds are examples of this (Lesku and Rattenborg, 2022; Rattenborg et al., 2019). A relevant fact for my discussion is that REM sleep occurs in them especially when the brain is subjected to bilateral N-REM sleep; in birds, REM sleep is absent or occurs in very small amounts whenever unihemispheric sleep occurs (Fuchs et al., 2009; Mascetti, 2016; Rattenborg et al., 1999a; Rattenborg et al., 1999b; Rattenborg, 2006; Rattenborg et al., 2016). This corroborates *hypotheses 10 and 11*. 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. Thus, the study by Lyamin and colleagues (2018) corroborates *hypotheses 10, 11, 13, and 14*.

Another fact that corroborates the Sentinel Sleep Theory is the way organisms respond when awakened from REM sleep. When an organism (human or non-human) is awakened from REM sleep,

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it exhibits full alertness (an obvious adaptive advantage) (Kandel et al., 2013, p. 1157; Ribeiro, 2021; Tseng et al., 2022). 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 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; Lima et al., 2005; Horner et al., 1997b; Ribeiro, 2021; Reite et al., 1965; Snyder, 1966; Ribeiro, 2021; Tseng et al., 2022). And in addition to there being empirical support in non-human animals for sentinel sleep (Tseng et al., 2022), it also has empirical support in humans (McKinnon et al., 2022; Samson et al., 2017). All this evidence corroborates *hypothesis 15*. 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.

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, 2021). The aforementioned evidence corroborates *hypotheses 16 and 17*.

Considering the protective function of REM sleep, it is expected that animals wake up during REM sleep if a stimulus associated with a predator is detected. The study by Tseng and colleagues (2022) provides robust evidence supporting this prediction. In their research, the authors tested whether animals react to predator stimuli during REM sleep. Among other findings, they observed that animals exposed to predator stimuli woke up during REM sleep but not during N-REM sleep. Furthermore, the researchers also demonstrated that the level of alertness during REM sleep was higher. Another relevant finding reported is that the same neurons fundamental for defensive responses during wakefulness are activated during REM sleep. All these results indicated, as Tseng and colleagues stated, that REM sleep enables rapid awakening in response to predatory stimuli, ensuring a successful defense against any threats to the animal's life. All these findings corroborate *hypotheses 1, 4, 15, 16, and 17*.

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, 2021). REM sleep rebound is proportional to the duration of its suppression, but (which is particularly relevant to my arguments) the opposite is not true. As affirmed by Ribeiro (2021), increasing N-REM sleep time also increases REM sleep, but it does not cause a subsequent "negative rebound", which corroborates *hypothesis 18*. 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 4.1 and 4.5 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, 2021),

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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 6, where I justify this assertion.) This is why patients medicated with antidepressants can exhibit near-complete or complete REM sleep inhibition for years without showing any notable deficits in learning and the capacity to form new memories, while maintaining normal brain functionality; REM sleep inhibition is an effect caused by practically all antidepressants, with some even interfering with the homeostatic regulation of REM sleep (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, 2021). These facts corroborate *hypothesis 19*.

The greatest harm that REM sleep inhibition causes to an organism is the substantial increase in its vulnerability during sleep. 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, as Lyamin and colleagues (2008) stated, it is common for scientists to also suppress N-REM sleep when studying REM sleep suppression.

4.4. The Parameters of REM Sleep Depend on the Organism's Vulnerability

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. Finally, a lower density of REM sleep is also predicted in less vulnerable organisms.

Before proceeding, I need to justify these statements. In humans, domestic animals, and laboratory animals, muscle atrophy typically limits mobility and physical performance and increases the likelihood of injury as the animal ages. In contrast, in wild animals, muscular performance can mean the difference between life and death. After all, muscle atrophy reduces the physical capacity to avoid predators, directly affecting the animal's chances of survival (Hämäläinen et al., 2015; Hindle et al., 2009). Muscle strength is, therefore, a variable directly related to protection (when muscles are developed) or vulnerability (when muscles are atrophied).

Regarding body mass, it is also related to fitness; reducing it can significantly impact physical fitness in both the short and long term (Amo et al., 2007). Moreover, it is easy to understand that an organism with a higher amount of body fat is physically more protected (or less vulnerable) compared to a leaner one. Concerning the physical blows needed to take down a prey, it is more challenging for a predator to kill a fat prey than a lean one. A predator that sinks its teeth or claws into a lean prey can more easily reach its vital organs. The same cannot be said for a fat prey. Body fat is, therefore, a variable directly related to protection (when there is a lot of body fat) or vulnerability (when there is less body fat).

Among the hypotheses related to how body fat and muscle strength affect REM sleep parameters, except for *hypotheses 26 and 29*, all other hypotheses (21, 22, 23, 24, 25, 27, and 28) are supported by empirical research (Brand et al., 2010; Chamorro et al., 2014; Driver et al., 1994; Driver and Taylor, 2000; Elrokhsi et al., 2020; Hague et al., 2003; Hrozanova et al., 2020; Kitamura et al., 2021; Kubitz et al., 1996; Liu et al., 2008; Myllymäki et al., 2011; Seol et al., 2022; Theorell-Haglöw et al., 2010; Youngstedt et al., 1997; Zapalac et al., 2024). Therefore, all this evidence also corroborates

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hypothesis 20. Next, I will comment on some of the studies I just mentioned. This will allow me to better explain some results from the perspective of Sentinel Sleep Theory.

In the study by Chamorro and colleagues (2014), the authors 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*. 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 Kitamura and colleagues (2021), the difference found between men and women 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 Liu and colleagues (2008), 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 Myllymäki and colleagues (2011), 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, this 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, which supports *hypothesis 20*.

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, a shorter latency to the first REM episode, as well as a greater intensity. 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, its intensity will be greater, 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, a shorter latency to the first REM episode, and a greater intensity of REM sleep*.

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 *hypotheses 30 and 31* and also implicates fear as an emotion capable of affecting REM sleep, corroborating *hypothesis 20*.

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

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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 (proprioceptive information that is mapped by the brain) (Arias et al., 2020; Gazzaniga et al., 2016, p. 620; Stahl, 2002; Targum and Fava, 2011; Wolpert, 2008). Fatigue is so common in depressed patients that it occurs in more than 90% of patients (Arnold, 2008; Demyttenaere et al., 2005; Ghanean et al., 2018). 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 its density (or intensity). 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, 2021), 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*.

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; Baglioni et al., 2016; Berger and Riemann, 1993; Gazzaniga et al., 2016, p. 620; Kishi et al., 2023; Lam, 2006; McCarley, 1982; Palagini et al., 2013; Riemann et al., 2020; Riemann and Berger, 1989; Schmid et al., 2008; Steiger and Pawlowski, 2019; Steiger et al., 2013; Suchecki et al., 2012; Wichniak et al., 2017). These REM sleep abnormalities (especially longer duration, higher frequency, and shorter latency) also manifest in animal models of depression (Cheeta et al., 1997; Vogel et al., 1990). All this evidence corroborates *hypotheses 33, 34, 35, and 39* and, therefore, *hypothesis 20*.

Of course, *vulnerability* is not measured directly. It is inferred here from the neurobiological conditions that depression imposes on the organism. Thus, it is the biochemical dysregulations (e.g., in the serotonergic, noradrenergic, and cholinergic systems) caused by depression that result in a more vulnerable organism. It is these biochemical dysregulations that affect the circuits of the brainstem and other cortical regions (e.g., the amygdala) that regulate REM sleep. 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., 2024; 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., 2024). 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., 2024; Damasio, 2003, p. 53; Dhabhar, 2018). In short, the immediate result of stress is to favor, directly or indirectly, the survival of the organism.

Someone might assume that it is incorrect for me to assert that stress makes the organism better protected (or less vulnerable). This person 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., 2024; Damasio, 2003, p. 53; Damasio, 2015, pp. 52-53; Dhabhar, 2018). Stress automatically

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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. After all, given that N-REM sleep is highly important and that it predominates at the beginning of sleep (Ribeiro, 2021), 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 level of alertness. In other words, REM sleep density is a measure of 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). Therefore, it is not surprising that nocturnal awakenings are frequent in patients with major depressive disorder (MDD) (Benca and Peterson, 2008; Lam, 2006; Leitner et al., 2025; Nutt et al., 2008; Peterson and Benca, 2008; Steiger and Pawlowski, 2019). (Remember that depression is associated with a higher density of REM sleep.) 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, 2021). 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; Lam, 2006, see Table 1 of this Article).

Now that I demonstrated the evidence that corroborates my conclusion that REM sleep density is a measure of 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

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according to the type of stress experienced and the duration of exposure to it (Papale et al., 2005). In humans, stress reduces both N-REM and REM sleep, increases REM sleep latency, and increases REM sleep density (Feng et al., 2023; Goldberg et al., 2020; Goodenough et al., 1975; Lauer et al., 1987).

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 reduces REM sleep.

However, a bit of caution is necessary. Acute administration of cortisol inhibits or suppresses REM sleep, which is consistent with my theory, but the physiological and endocrine reality of the HPA axis and cortisol itself is more nuanced. In the second half of the rest period, the amount of cortisol reaches a peak that directly relates to the greater amount of REM sleep typical of this second half (Born et al., 1988; Fehm et al., 1993; Payne and Nadel, 2004; Van Reeth et al., 2000). In sum, while N-REM sleep is associated with a reduction in cortisol amounts, particularly during the deepest stage of sleep, we observe greater amounts of cortisol in REM sleep relative to N-REM sleep (Steiger, 2002; Steiger, 2007).

At first glance, these facts appear to contradict the predictions of my theory if we think of a linear model in which *protection* increases proportionally to the amount of *cortisol*. If that were the case, increasing cortisol would progressively reduce REM sleep. However, this linear model is incorrect. The appropriate model is a curve, since cortisol rises alongside REM sleep up to a certain point. In other words, REM sleep requires a certain amount of cortisol to operate at optimal efficiency. It is worth recalling that cortisol plays a critical role in safeguarding the organism (Chand et al., 2021; Chu et al., 2024; Damasio, 2003, p. 53; Damasio, 2015, pp. 52-53; Dhabhar, 2018). It is only beyond a certain threshold does cortisol begin to inhibit REM sleep, at which point the curve representing REM sleep starts to decline. If cortisol levels increase further, REM sleep will eventually be effectively suppressed. Therefore, the evidence above does not refute the predictions of my theory, we just need to analyze the issue with the appropriate caution.

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 major depression, the HPA axis becomes overactive (Keller et al., 2017; Mikulska et al., 2021). Therefore, it would be superficially expected that REM sleep duration would decrease and its latency would increase. However, we must remember that the body is more vulnerable during depression (and it seems precisely for this reason that the HPA axis must become hyperactive, given its role in bodily protection via stress). Consequently—given the heightened vulnerability associated with depression—REM sleep must last longer and have a shorter latency. In other words, vulnerability due to depression takes precedence as information for determining REM sleep parameters, since it compromises the organism's safety; a consequence that, in nature, directly endangers its life. But this does not imply that the HPA axis does not affect REM sleep in depressed patients. In these

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individuals, since the HPA axis becomes overactive, it is possible that the interoceptive information from the increased circulation of stress hormones contributes to raising REM sleep density.

It is possible to better understand how the HPA axis affects REM sleep by considering that REM sleep requires a certain amount of stress to operate properly (remember that stress serves a protective role). This implies that, within certain limits, increasing the amount of stress helps make REM sleep more efficient as a protective mechanism. REM sleep becomes less efficient—under the influence of stress—only once stress levels begin to interfere with the organism's sleep, that is, when stress is high enough to awaken the organism. Stress can inhibit REM sleep (in non-depressed individuals) only at higher doses, because the brain interprets that the body is already sufficiently protected, making REM sleep less necessary (resulting in shorter duration and longer latency). Moreover, as we have seen, even higher doses of cortisol can actually suppress REM sleep in non-depressed individuals, as it becomes dispensable given the sufficient bodily protection afforded by extremely high amounts of stress.

All the aforementioned evidence regarding stress corroborates hypotheses 36, 37, 38, 40, and 41. Therefore, they also corroborate hypothesis 20. 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 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.

To further corroborate the arguments I developed for the sentinel function of REM sleep, I will analyze REM sleep in neonates. As described by Ribeiro (2021), 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, 2021). 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, 2021). The high physical immaturity (or fragility, or vulnerability) with which countless organisms begin life represents a clear disadvantage, requiring regular parental care (Ribeiro, 2021).

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, 2021; 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;

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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).

One might 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), the aforementioned 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).

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.

All the evidence I presented above corroborates *hypotheses 42 and 45* and, therefore, *hypothesis 20*. Consequently, the correlation between an excessive amount of REM sleep and greater physical immaturity at birth constitutes a 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).

Moreover, it is possible that the variable "vulnerability" encompasses not only external risk (primarily predation) but also internal risk arising from the developmental process itself. After all, development is highly sensitive and vulnerable (Lamberto et al., 2021; Rice and Barone, 2000). This perspective suggests that REM sleep parameters (especially its quantity) are also finely tuned according to developmental needs, both of the brain and of the body as a whole. This applies particularly to species whose postnatal development is more extensive, which may explain why certain species exhibit much more abundant postnatal REM sleep.

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

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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*.

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. 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). 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).

Moreover, 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 attempt to do to demonstrate that increasing bodily protection causes specific and predictable changes in REM sleep parameters. I will focus on 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, 2021), 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 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.

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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, *et cetera*) 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 ($p < 0.001$). 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

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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, 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.

4.5. 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 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

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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 · 1/6 · 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).

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).

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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 N-REM 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 N-REM sleep. (*Note that the term "error" should be understood in the sense of a failure in the control of the transition from N-REM 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 N-REM 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 N-REM sleep than for those that remained asleep. The brief 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, 2021). However, we must ask the following. Who is more vulnerable: an immobile organism while in N-REM sleep (with low levels of attention, vigilance, and readiness, especially in deep sleep), 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 N-REM sleep, the organism's

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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.)

In this historical narrative I proposed, each step is a small modification that confers a selective advantage over the previous state. Small enough to be likely to arise from a *de novo* mutation (a mutation in an individual's DNA sequence that was not inherited from its parents). 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, 2021). 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.

5. Attempts at Refutation

1. If N-REM sleep already provides a certain quantity of alertness and protection during sleep, why is REM sleep necessary? First, we must recognize that an organism's quantity of protection and alertness varies on a spectrum. This means that an organism—depending on its neural state and its physiological and behavioral responses—can be more or less protected; more or less alert. The spectrum of alertness and protection is distributed as follows: during wakefulness, alertness and protection are very high. During N-REM sleep, they are very low. REM sleep occupies a position between these two, lying closer to wakefulness: it is a sleeping wakefulness.

It is easy to understand why N-REM sleep must retain a minimal amount of alertness. After all, having *some* alertness is certainly better than having *none*. However, it can be improved. *Increase alertness a bit more, and the organism will be even better protected*. This is where REM sleep comes in. N-REM sleep does indeed provide some quantity of alertness (Gazzaniga et al., 2016, p. 146; Moyne et al., 2022), but it cannot match the alertness and the physiological and behavioral responses that REM sleep enables (Tseng et al., 2022). Therefore, the fact that N-REM sleep offers some alertness and protection neither precludes the existence of REM sleep nor diminishes its function. Furthermore, the HPA axis—an important protective component (see Section 4.3)—is quietest when N-REM sleep is at its deepest state, something that happens especially during the first half of the rest period (de Feijter et al., 2022; Vgontzas and Chrousos, 2002; Weitzman et al., 1974; Weitzman et al., 1983). That is, N-REM sleep clearly places the organism at a more pronounced vulnerability than REM sleep.

In Section 4.5, I explained why intense muscle atonia probably evolved over the course of biological evolution. However, some doubts may remain regarding the plausibility of my proposal. After all, pronounced muscle atonia during REM sleep can increase the organism's vulnerability to environmental threats (Lima et al., 2005). Yet we must remember that animals respond more effectively to threats upon waking from REM sleep than from N-REM sleep (Horner et al., 1997b; Tseng et al., 2022; Voss, 2004). Moreover, a recent study indicated that the muscle twitches that occur during REM sleep are capable of causing sensorimotor activity that prepares the organism for

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wakefulness (Brooks and Peever, 2016). Finally, an even more recent study clearly demonstrated that REM sleep possesses specific characteristics enabling an animal to awaken easily upon detecting a dangerous stimulus (Tseng et al., 2022). These characteristics include rapid and specific reactivity to predatory stimuli, rapid increase in pupil size, and rapid increase in the ability to move when detecting a predatory stimulus. All these characteristics ensure that the animal can efficiently defend itself against threats to its life during REM sleep (Tseng et al., 2022).

2. Phasic REM can be less responsive than tonic REM. Another attempt to refute my theory is to resort to the tonic and phasic periods of REM sleep. REM sleep is not a homogeneous state, comprising at least two phases: tonic and phasic (Simor et al., 2020). Evidence indicates that phasic REM is less responsive to certain environmental stimuli than tonic REM (Atienza et al., 2001; Ermis et al., 2010; Price and Kremen, 1980; Sallinen et al., 1996; Takahara et al., 2002). Given this difference, one might conclude that the Sentinel Sleep Theory does not hold. However, for my theory, it matters little whether phasic REM is less responsive to some environmental stimuli than tonic REM (and understanding why falls outside the scope of this work). What matters for my proposal is *responsiveness to potential threats*, not to any environmental stimulus. Note that *all* of the above studies used auditory stimuli, not dangerous auditory stimuli. *This difference matters*. The studies comparing environmental responsiveness between tonic and phasic REM *did not test the specificity for threatening stimuli*. That is what must be tested in order to corroborate or refute my theory. To overturn my theory, it would be necessary to demonstrate that REM sleep *is not* selectively responsive to dangerous stimuli. Yet the evidence shows that REM sleep is, indeed, selectively responsive to dangerous stimuli (Tseng et al., 2022). What remains is to analyze how this specificity varies (or does not vary) between the phasic and tonic periods.

3. Powerful species and REM sleep. Another (incorrect) attempt to refute my theory is to appeal to the fact that powerful species exhibit REM sleep (Allison and Cicchetti, 1976). If REM sleep serves to protect the organism during sleep, is it necessary for powerful species (such as lions and tigers) to have REM sleep? This is indeed an important question, but we must be cautious about oversimplifications. The idea that predators attack and prey defend is an overly simplistic view (Magalhães et al., 2005). We must remember that no predator is born as an adult, physically mature. Young predators are indeed more vulnerable (Magalhães et al., 2005). Therefore, the notion that predators (or powerful species) should not exhibit REM sleep is an error resulting from this simplification. Predators can also be attacked, especially when they are young (Magalhães et al., 2005).

We must always remember that animals can be both predators and prey. Cats hunt mice, but they are also preyed upon by other animals, such as foxes, coyotes, wolves, large birds of prey (e.g., owls and eagles), and even by other wild cats. Lions—apex predators—are likewise threatened by other animals, including hyenas, buffalo, wild dogs, rhinoceroses, elephants, hippopotamuses, crocodiles, leopards, and even other lions (Kingsley, 2024; Williams, 2025).

For the sake of argument, let us suppose that REM sleep is eliminated in predators. What would happen to their vulnerability during sleep? In contrast to REM sleep, awakening from N-REM sleep (especially from deep sleep) is *not* associated with full alertness or optimal sensory and motor efficiency (Kandel et al., 2013, p. 1157; Lima et al., 2005; Horner et al., 1997b; Ribeiro, 2021; Snyder, 1966; Tseng et al., 2022). Consequently, without REM sleep, predators would become more vulnerable during rest. It would be easier for a physically immature predator to be killed during sleep, either by prey or by another predator. Furthermore, it is possible that the absence of REM sleep would pave the way for a prey species (or more than one) to develop the strategy of attacking their predators while they sleep. After all, without REM sleep, a predator attacked during sleep would awaken completely disoriented and would remain momentarily unable to defend itself with the full vigor that only awakening from REM sleep allows. This strategy could also be developed by other predatory species. It is important to remember that there are numerous reports of predators attacking other predators (Bohannon, 2006).

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We must also remember that natural environments are challenging. Predators are strong and generally large (Magalhães et al., 2005), but they are neither invulnerable nor indestructible. Even strong predators can suffer accidents, which increases their vulnerability both during wakefulness and sleep. In addition, predators are not immune to the harmful effects of prolonged hunger, another factor that can place them in a state of greater physical vulnerability. After all, a well-nourished predator has a clearly more advantageous defensive capacity compared to a malnourished predator. (Anyone who watched nature documentaries knows that, on many occasions, predators go without food for long periods.) Therefore, I reiterate: *the idea that strong species should not have REM sleep is incorrect*.

Furthermore, it is possible that *the amount of REM sleep a species exhibits is also a product shaped by that species' overall life-history strategy*. Apex predators, such as lions, tend to follow a life-history strategy characterized by producing fewer offspring compared to prey species, such as rodents (Burak et al., 2018). This implies that, for a predator, losing even a single offspring carries a greater cost for the species' survival. Therefore, investing more in REM sleep—even though it is more energetically costly—makes perfect evolutionary sense. By contrast, for prey species, which tend to produce a larger number of offspring throughout their lifetime, losing a single offspring is less costly from the standpoint of species survival. In this case, survival is better ensured through high reproductive rates. That is, for prey species, the cost-efficient evolutionary strategy is to minimize (as much as possible) the total time spent in the vulnerable state of sleep, even if this means less time in the sentinel state (especially compared to predator species).

In sum, the fact that prey species have shorter gestation periods, faster population dynamics, and overall faster life-history strategies (Abrams, 1992; Abrams, 2000; Becks et al., 2012; Cortez and Ellner, 2010; Post and Palkovacs, 2009) may also help explain why predator species exhibit more REM sleep relative to prey species. In other words, what I am arguing is that *the vulnerability REM sleep seeks to counterbalance may not be strictly immediate. Beyond protecting the individual organism, it may also serve to protect offspring or adult pairs of the same species—thereby ensuring, with greater probability, the species' posterity*. Therefore, if a species produces fewer offspring over its lifetime, has slower population dynamics, and follows a slower life-history strategy, it is evolutionarily advantageous to have more REM sleep.

Below, I will complete the discussion of the amount of REM sleep in predators and prey with an additional argument.

4. Species more vulnerable to predation have less REM sleep. Evidences apparently contrary to the arguments I developed here are provided by the studies of Allison and Cicchetti (1976) and Lesku and colleagues (2006). In these works, the researchers demonstrated that species more vulnerable to predation have less REM sleep time than predator species. However, caution is warranted when interpreting these findings, as the only way for an animal to have significantly more REM sleep time without sacrificing the necessary N-REM sleep is by sleeping longer. And this is something predators can afford to do. This explains why predator species tend to sleep more, whereas prey species tend to sleep less (Allison and Cicchetti, 1976). Furthermore, REM sleep depends on N-REM sleep (Le Bon, 2020), and Le Bon and colleagues (2002) found a strong positive correlation between the proportion of N-REM and REM sleep and the frequency of cycles per night: the more REM/N-REM sleep cycles, the greater the ratio between the durations of REM and N-REM sleep.

Therefore, it is naïve to think that, based on the sentinel function, we should necessarily expect prey species to have more REM sleep than predator species. This conclusion is absurd. If you are a prey sleeping in a more vulnerable environment, you cannot afford the luxury of sleeping extensively; that is the luxury of prey species that sleep in safe locations and—most notably—of predator species. The consequence of this is that, even if the function of REM sleep is to act as a protective mechanism, prey species—being forced to sleep for shorter periods—will tend to have significantly less REM sleep than predators. Hence, the fact that prey species have comparatively less REM sleep than predators does not, in any way, invalidate the sentinel function.

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5. REM sleep and the arousal threshold "paradox". 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 even be higher (e.g., Ermis et al., 2010; Pilon et al., 2012). Does this constitute a fatal refutation of my arguments? I will demonstrate hereinafter that these facts actually support (rather than refute) my arguments.

In Section 4.5, 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 4.3). *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., 1997a; 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

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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) (Dauvilliers et al., 2007; Mochizuki et al., 2011; Ribeiro, 2021). 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, 2021; 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.

6. The locus coeruleus-norepinephrine system and 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 locus coeruleus-norepinephrine (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.

What I hope to have demonstrated is that the arousal threshold of REM sleep and N-REM sleep do not need to differ for my theory to be corroborated. The most important thing, after all, is that REM sleep presents a behavioral response when the organism's brain detects a potentially dangerous stimulus (not just any stimulus; it needs to be a stimulus that is potentially dangerous to that organism). Tseng and colleagues (2022) demonstrated that this behavioral response occurs. Therefore, the theory remains corroborated. *What I am arguing is that REM sleep is biased toward potentially dangerous stimuli—not just* any *external stimuli*. So it is naive to point to a study like Ermis and colleagues (2010) and claim that my theory has been disproved.

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This explanation—about the silencing of key wake-promoting systems during REM sleep—reveals how the brain achieves a remarkable and highly adaptive state: it is internally hyper-alert (driven by cholinergic activity and limbic activation) but simultaneously barricaded from both trivial external stimuli and a full transition to wakefulness (via the inhibition of these key arousal systems). This creates a "sleeping sentinel" that is primed to respond to specific, significant threat-related stimuli, as demonstrated by Tseng and colleagues (2022), but is not constantly disrupted by minor sensory input. The silencing of the LC-NE system, for example, is not a contradiction to a vigilance theory, but a crucial feature that allows the sentinel to remain asleep while on duty.

7. N-REM sleep is heterogeneous. 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 4.5, 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.

Considering that, at present, N-REM micro-awakenings and REM sleep itself appear to be distinct neural phenomena in terms of function and neuronal circuitry (Kjaerby et al., 2022; Luppi et al., 2024), the following question may arise. How could two distinct physiological phenomena share similar evolutionary origins? Well, this is exactly the result of non-random elimination of individuals over countless generations—that which we know as evolution. The fact that two contemporary physiological phenomena differ in their form and function does not imply that they have separate origins. It remains entirely possible that they both arose from a common evolutionary precursor. Indeed, the principle that distinct traits can share a common evolutionary origin is one of the cornerstones of evolutionary biology (Darwin, 1859; Dawkins, 1997; Mayr, 1982). Therefore, the mere present-day difference between REM sleep and the micro-awakenings occurring during N-REM sleep cannot be used to refute my hypothesis that REM sleep evolved from brief awakenings. We must also remember the methodological limitation that sleep leaves no fossil record. Thus, we are unable to access data that would allow us to reconstruct a relatively precise history of sleep's evolution throughout the evolution of organisms.

8. In certain situations, waking up from N-REM sleep may be easier than waking up from REM sleep. Another piece of evidence that seems to refute the Sentinel Sleep Theory is provided by the experiment of Siegel and Langley (1965), whose findings suggest that, on some occasions, it may be easier to awaken from deep sleep than from REM sleep. However, once again, we must interpret this with caution. Based on the sentinel function, we should (obviously) expect a greater ease of awakening during REM sleep. But it is naïve to conclude that this ease applies to *every* type of stimulus. The stimulus that Siegel and Langley used to awaken both cats in the experiment was a series of clicks of varying duration and intensity. More importantly, the cats had learned to associate the end of a series of clicks with a reward: milk. Among other findings, Siegel and Langley (1965) reported that, when exposed to clicks during sleep, the cats awakened more easily during deep sleep than during REM sleep.

However, this result does not refute the Sentinel Sleep Theory. As I am arguing, REM sleep evolved to be *responsive to danger*, not to stimuli associated with an appetitive reward. And, as demonstrated by Tseng and colleagues (2022), REM sleep is indeed particularly responsive to danger. Furthermore, throughout the conditioning and testing phases of Siegel and Langley's experiment, the cats were deprived of food and water. Thus, the results also suggest that N-REM sleep is more responsive to food-related stimuli than REM sleep—especially, perhaps, when the animal is deprived

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of water and food. What would the results have been if the clicks had been associated with shocks or any other negative and harmful experience for the body? I predict that the clicks would have awakened the cats much more easily during REM sleep than during N-REM sleep. Another prediction: if it is not a dangerous stimulus (e.g., associated with a predator), I predict that the intensity of the stimulus will have to be greater to awaken the organism from REM sleep. Which is exactly what Siegel and Langley's results show.

9. Brain temperature and REM sleep. Let us now consider another way to try to challenge my theory. In most mammals studied to date, brain temperature (both cortical and subcortical) increases during REM sleep and decreases during N-REM sleep (Ungurean et al., 2020; Wehr, 1992). This led many scientists to believe that the primary function of REM sleep is to regulate cortical temperature. The first scientist to propose this hypothesis was Thomas Alvin Wehr, in 1992. However, if the function of REM sleep were to warm the brain, then such warming should also occur in other animals (beyond mammals) that exhibit REM sleep or its analog (Ungurean et al., 2020). But this is not the case. In bearded dragons, cerebral temperature does not increase when the brain transitions from N-REM sleep to REM sleep (Ungurean et al., 2020).

Moreover, the evidence indicates that the brain warms during REM sleep merely as a result of increased blood flow, which is necessary for the brain to carry out REM sleep (Bergel et al., 2018; Denoyer et al., 1991; Parmeggiani, 2007; Pastukhov and Ekimova, 2012; Wehr, 1992). (See also the study by Ungurean and colleagues [2020], in which the authors challenge the notion that the primary function of REM sleep is to warm the brain.) This body of evidence demonstrates that the brain warming associated with REM sleep may be merely a functionless byproduct, arising from mechanisms that require increased neuronal activity—such as the sentinel function. Therefore, the fact that brain temperature rises during REM sleep supports (rather than refutes) the Sentinel Sleep Theory. This is because the sentinel function requires increased neuronal activity—a demand that may elevate brain temperature (albeit subtly).

To better understand how REM sleep relates to temperature, we must also consider that low temperatures can reduce blood flow (Bisschops et al., 2010; Croughwell et al., 1992; Ibayashi et al., 2000; Mrozek et al., 2012; Shepherd et al., 1983). Accordingly, since REM sleep requires increased blood flow while the brain executes it, low temperatures—by virtue of their capacity to reduce blood flow—may suppress REM sleep. This can explain why, in mammals exposed to low temperatures, REM sleep is selectively and strongly suppressed (Amici et al., 1998; Amici et al., 2008; Cerri et al., 2005; van Hasselt et al., 2024).

10. Immune system and REM sleep. To end this Section, I will address evidence related to the immune system. This is another line of evidence that, at first glance, appears to refute my arguments. But we just need to analyze more carefully to see that, in fact, they corroborate it. When disease-causing bacteria invade the body, its entire functioning is profoundly altered to respond to the invasion (Krueger et al., 1982). In humans, REM sleep was significantly reduced in response to *Salmonella abortus* endotoxin (Pollmächer et al., 1993). In rabbits inoculated with *Staphylococcus aureus*, from 6 to 10 hours after inoculation, REM sleep was suppressed, remaining at low levels for the rest of the recording period (Toth and Krueger, 1988). Two other notable findings from the Toth and Krueger study are that (1) antibiotics (cephalothin) attenuated the effects of this bacterial infection on sleep and (2) inoculation with dead bacteria also produced analogous changes in sleep and other physiological parameters. In summary, during the progression of most infections, REM sleep will, at some point, be inhibited or experience a reduction in its duration (Fang et al., 1995; Imeri and Opp, 2009; Krueger and Majde, 1994).

Some scientist might conclude, based on the above evidence, that it refutes the Sentinel Sleep Theory by assuming that infection leaves the organism vulnerable and, therefore, this vulnerability would increase (rather than reduce) REM sleep. However, this conclusion is incorrect. This is because the vulnerability arises from what the antigen could do to the body if the organism fails to manage the infection. Furthermore, we must consider that the immune system is a mechanism whose function is *to protect* the organism from bacteria, viruses, parasites, and toxic molecules capable of invading its

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body (Damasio, 2003, p. 31). The immune system is the first line of *defense* that *protects* vertebrates from threats affecting their integrity, whether arising from within their own body or from external sources, due to an infection (Damasio, 2003, p. 31).

Therefore, since antigens (e.g., bacteria) trigger an immune response composed of numerous antibodies capable of neutralizing the invader (Damasio, 2003, p. 58), the body is flooded with proteins that serve to make it *better protected* against the threat. And what happens to REM sleep when the body is more protected? Naturally, it decreases. As the parameters of REM sleep are determined by proprioceptive information, during an infection, proprioceptive signals inform the brain that the organism is inundated with protective proteins—especially at the peak of the immune response. This explains why REM sleep decreases rather than increases. My conclusion is supported by the fact that the administration of cytokines (proteins that play an important role in the immune system) suppresses REM sleep (Opp, 2005). Therefore, the evidence concerning the immune system and its relationship with REM sleep (Fang et al., 1995; Imeri and Opp, 2009; Krueger and Majde, 1994; Pollmächer et al., 1993; Toth and Krueger, 1988) supports the Sentinel Sleep Theory as a whole and, more specifically, *hypothesis 20*.

6. A Significant and Detrimental Consensus

Since the current *zeitgeist* is dominated by a consensus 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, 2021), I need to address this issue. After all, this consensus can hinder the proper understanding of the true biological function of REM sleep.

As pointed out by Capellini and colleagues (2008) and Ribeiro (2021), 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, 2021). 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, 2021)? 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 evidence from Capellini and colleagues (2008) demonstrates that, despite the association with learning, REM sleep does not play a critical role in it. 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, 2021). 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, 2021).

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, 2021)—one would expect to find an increase in IEGs expression during sleep, especially

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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, 2021). 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, 2021).

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 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?

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*

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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 (Tseng et al., 2022), nor is it critically related to learning (Capellini et al., 2008), 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 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.

But how exactly can REM sleep contribute, as a byproduct, to memory consolidation? We known for a long time that memories must be continuously reactivated in order to exist and be preserved over time (Gazzaniga et al., 2016, p. 268; Kandel et al., 2014; Martin et al., 2000; O'Leary et al., 2024; Pignatelli et al., 2019; Ryan and Frankland, 2022; Swaab et al., 2002; Tononi and Cirelli, 2014). We also know that memories are not reactivated based on the order in which they were learned, but rather on their biological value (Damasio, 1996; Damasio, 2012; Damasio, 2019).

Combining these two lines of evidence, we arrive at the following conclusions. *The memories that reverberate most frequently in the mental flow are those with emotionally relevant content*. And this applies especially to sleep. After all, during wakefulness the organism is inundated with stimuli from the senses, and the brain must update the responses to numerous important questions (Kahneman, 2011; Ribeiro, 2021): Did something new occur? Has a threat emerged? Is homeostatic regulation functioning properly? Should attention be redirected to another event or stimulus? Does this task require additional energy? Therefore, neural reverberation based on the importance of memories is particularly relevant during sleep to determine which memories reverberate most often in the mental flow. (I use the term "mental flow" to refer to the electrically active neural patterns in the central nervous system due to memories of current or evoked stimuli, actions, or events.) In turn, understanding which memories reverberate most frequently in the mental flow helps us identify which memories are likely to be best preserved (i.e., learned).

But why does REM sleep appear to contribute especially to learning? The answer depends both on neural activation driven by the biological value of memories and on which brain regions are active during REM sleep. The areas of the brain that become active during sleep—and the intensity of that activation—vary considerably and are directly linked to the two main sleep states: REM sleep and N-

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REM sleep (Han et al., 2024; van der Helm and Walker, 2011). REM sleep, for example, is associated with high neural activity in the occipital cortex, thalamic nuclei, pontine tegmentum, mediobasal prefrontal lobes, as well as in affect-related regions including the amygdala, hippocampus, and anterior cingulate cortex (van der Helm and Walker, 2011). Given the diversity of neural activation across these specific sleep states, REM and N-REM sleep play distinct roles in the memory consolidation process (van der Helm and Walker, 2011).

Although scientists traditionally associated REM sleep with a particular capacity for consolidating emotional memories (Groch et al., 2013; van der Helm and Walker, 2011), a recent study provided evidence that both N-REM sleep and REM sleep contribute to consolidating emotional memories (Yuksel et al., 2025). This evidence reinforces my argument that it is the *frequent activation* of memories within the mental flow—based on their biological value—that drives their consolidation. It is not REM sleep itself that functions to consolidate memories, but rather the electrical activation of those memories during REM sleep—and considering the neurally active regions due to the function of REM sleep—that contributes to their consolidation.

This is why sleep (in general) appears to prioritize the consolidation of emotional memories (Payne et al., 2008; Zhong et al., 2024). *My argument is that this is not a function of sleep—neither N-REM nor REM—but merely a byproduct of the electrical activation based on the biological value*. During sleep, without the overwhelming interference of sensory information from wakefulness, neural electrical activation depends particularly on the importance of the memory. Consequently, it is the emotional memories that reverberate most frequently during sleep and—precisely because of their electrical activation—end up being better consolidated.

At this point, it is worth recalling that one of the neural regions with heightened activity during REM sleep is the hippocampus: a crucial region for memories to be consolidated (Fogwe et al., 2023; Lee and Wilson, 2002; Nolan, 2023; Squire et al., 2015). Moreover, there is evidence that it is hippocampal neural activity during REM sleep (related to theta rhythm) that is involved in memory consolidation (Dragoi et al., 1999; Poe et al., 2000; Stickgold and Walker, 2007; Montgomery et al., 2008; Nishida et al., 2009; Rattenborg et al., 2011). Indeed, Poe and colleagues (2000) provided empirical evidence that it is the *repetition* of neural activity during REM sleep that contributes to memories being consolidated. This is consistent with classical evidence that memory repetition contributes to their retention (e.g., Ebbinghaus, 1913; Hebb, 1961) and also with more recent evidence (e.g., Bridge et al., 2012; Dudai and Eisenberg, 2004; Karpicke and Roediger, 2008). There is also evidence that, during N-REM sleep, the electrical reactivation of hippocampal memories similarly contributes to their consolidation (Lee and Wilson, 2002; Nádasdy et al., 1999).

Additional evidence for my claim that it is electrical activation—not REM sleep—that contributes to consolidation comes from the study by Bass and colleagues (2012). In that work, the authors reported that electrical stimulation of the basolateral amygdala complex resulted in markedly improved memory. The following studies likewise reported enhanced memories due to electrical stimulation (Bass et al., 2014; Bass and Manns, 2015; Geva-Sagiv et al., 2023).

In sum, considering all the evidence above, it is clear that the reason REM sleep contributes to consolidating memories is due to the neural mechanism of electrically activating memories based on their biological value—something that, as I demonstrated, is not confined to REM sleep. N-REM sleep, by that same neural mechanism, also contributes to consolidating emotional memories (Yuksel et al., 2025). Thus, this mechanism explains why sleep—especially REM sleep—plays a critical role in emotional processing (Tempesta et al., 2018) and in psychological health (Kollar et al., 1969). To further clarify what I am proposing, I am not claiming that memory consolidation associated with REM sleep is a byproduct of the sentinel function itself, but rather a byproduct of the neuronal electrical activation that occurs during REM sleep, which, in turn, is associated with the sentinel function.

Neural reverberation being based on biological value and neurally active regions also allows us to reinterpret the proposal of "reverse learning" (Crick and Mitchison, 1983). After all, if spontaneous electrical activation depends on the biological value of memories, then a logical consequence is that

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less important memories will be activated less frequently. The consequence of this, in turn, is that they will tend to be unlearned after sufficient time passes without being electrically active (Tononi and Cirelli, 2014). That is, while sleep contributes to consolidating important memories, it also contributes to deconsolidating those that are infrequently activated. This explains why REM sleep both forms and eliminates synapses (Li et al., 2017). Note that this mechanism is not confined to REM sleep. Because memories are activated according to their biological value, both REM and N-REM sleep passively contribute to the weakening of less important (or effectively unimportant) memories. Although it is obvious that we should expect the effects of this weakening to be distinct in REM sleep and N-REM sleep, precisely because the neural regions active during these states vary (van der Helm and Walker, 2011).

Finally, the aforementioned mechanism also helps to explain why REM sleep is so strongly associated with dreaming—a strong and well-established relationship we known for a long time (Desseilles et al., 2011; Gazzaniga et al., 2016, p. 146; Martin et al., 2020; Ribeiro, 2021; Solms, 2000). After all, with more intense neural activation—especially in the limbic system—it is natural that dreams occurring during this sleep stage are more vivid, more intense, and more emotional.

To conclude this section, I will address a question that may arise within the scientific community: Am I creating a false dichotomy by proposing that memory consolidation associated with REM sleep is a byproduct? Are the two functions mutually exclusive? Or is it possible that they are deeply interconnected? The very process of consolidating memories of emotionally salient events and stimuli—especially threats—is, in itself, a form of long-term vigilance. After all, consolidating what is dangerous is crucial for future survival (Damasio, 2012; Damasio, 2019). What if, instead of being rivals, the Sentinel Sleep Theory and the memory consolidation hypothesis are two sides of the same coin? The neural activity during REM sleep could serve both purposes simultaneously: enhancing the organism's alertness to present dangers (real or dreamed) and providing the neurochemical environment necessary for more effective memory consolidation. Such a synthesis would not weaken my theory. On the contrary, it would integrate what is probably the leading competing hypothesis, elevating it to a fundamental feature of the theory. This is very tempting, but we must proceed with caution.

To better evaluate this integrative possibility I have just outlined, it is necessary to distinguish between the biological function of REM sleep, on the one hand, and the biological functions of memory, on the other. *Memory* here is understood in the general sense applied to living beings: the capacity of a nervous system to acquire, form, consolidate, and retrieve information (Bisaz et al., 2014; Izquierdo, 2018, pp. 1, 4). One of the primary functions of memory is, unquestionably, to regulate life. For this reason, life management and memory storage are intimately connected (Bisaz et al., 2014; Damasio, 2012; Damasio, 2019; Forester et al., 2020; Thornton and Boogert, 2019). This clearly includes the ability to map threats that arise within the range of any sensory modality. Organisms that map threats have higher chances of survival, since encountering the same threat again will trigger a state of vigilance and, if necessary, a state of fight or flight (Chand et al., 2021; Damasio, 2012; Damasio, 2019; Oken et al., 2006). That is, it is true that consolidating information from emotionally competent events and stimuli—especially threats—constitutes a form of long-term surveillance, thereby ensuring biological regulation. However, what is pivotal here is recognizing that this is one of the functions *of memory*, but not necessarily of REM sleep. Thus, the most parsimonious explanation, based on the evidence I discussed, is that memory consolidation associated with REM sleep is indeed a byproduct, rather than one of its functions.

7. How to Assess the Adequacy of a Scientific Theory?

Attempting to answer the question that titles this section with a single criterion would be far too incompatible with best scientific practices—and also incorrect. We, scientists, must assess how adequate a theory is on the basis of multiple criteria rather than only one. And that is precisely how it should be. The quality and adequacy of a scientific theory cannot be evaluated by appealing to a single sufficient criterion. In Section 2, I presented seven criteria that I employed to test my theory.

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In this Section, I will dwell somewhat more on the fifth criterion from that list—a theory's capacity to solve problems. I will do so because many scientists, when considering a theory, tend to value almost exclusively its empirical support. I will argue that, although the extent to which a theory has been empirically corroborated is important, what matters somewhat more are the problems—especially the most significant ones—that it resolves.

When developing scientific theories, one hopes to coherently and adequately solve significant empirical and conceptual problems (Laudan, 1977, p. 70). Furthermore, scientists also develop theories to avoid or resolve the numerous conceptual and empirical problems that predecessor hypotheses or theories have (Laudan, 1977, p. 70). Larry Laudan (1977, pp. 29, 66) proposed that, if two theories compete with one another, we should regard as better and more preferable (or truer) the one that proves more successful both in solving significant problems in a convincing and meaningful way and in resolving a greater number of problems. Mayr (2001, pp. 54, 77) endorses this method of choosing between rival theories, as do I.

I agree with Laudan (1977, pp. 5, 13-14) when he states that a theory's competence to solve *relevant* problems is more important than the extent to which it has been confirmed or refuted. However, I must clarify a few points. Laudan (1977) argues that the best criterion for evaluating a scientific theory is its capacity to solve significant problems. The issue is that Laudan goes so far as to dismiss confirmation or refutation as useful and necessary criteria. In other words, Laudan disregards the truth of a theory as a criterion for evaluating it. On this point, I disagree with him. *The criterion of problem-solving is certainly pragmatic, but it must be coupled with empirical corroboration in order to have scientific value*.

To make my position clear, I am not claiming that a theory's problem-solving capacity outweighs its empirical testing. What I am in fact asserting is that we should place *a little more* value on problem-solving. Thus, both the degree of confirmation and the capacity to solve problems are relevant, but the slightly greater weight should fall on the resolution of *significant* problems. The extent to which a theory has been empirically corroborated is a necessary criterion for any serious scientific theory—and no properly trained scientist would dare to disagree. Nevertheless, I endorse the view that a theory's capacity to solve relevant problems should be regarded as even more significant. And this is by no means incompatible with the pluralism of criteria that we ought to adopt in evaluating scientific theories. After all, advocating a plural criteria does not necessarily commit us to granting equal weight to each of them. *No individual criterion is sufficient, but some may carry more weight than others*.

Many scientists, when questioning a theory's adequacy or robustness, tend merely to count the number of facts (or pieces of empirical evidence) that corroborate or contradict it. However, what we should also consider—and give greater weight to—is the theory's capacity to adequately solve significant empirical problems (Laudan, 1977, pp. 13-14). Moreover, for a theory to be regarded as more adequate than its rivals, it must entail a smaller number of anomalous problems; a property that is closely related to the theory's problem-solving capacity (Laudan, 1977, pp. 18, 30-31, 66).

The objectives of this Section are: (1) to assess whether my theory meets the criterion of adequately solving significant empirical problems in the domain of REM sleep; and (2) to compare my theory with rival hypotheses with regard to the number of anomalous problems.

According to Laudan (1977, p. 29), whenever a theory solves an empirical problem *P*, then *P* henceforth becomes an anomaly for every rival theory that is unable to solve *P*. An *anomaly* here denotes an empirical problem that a given theory cannot resolve but that one or more rivals are able to resolve (Laudan, 1977, p. 17). With respect to REM sleep, my theory lacks rival theories capable of explaining and resolving it—what exists are rival hypotheses. Considering the number of significant empirical problems my theory solves, all of them become anomalies for any rival hypotheses that are unable to resolve them convincingly. In other words, the Sentinel Sleep Theory creates a considerable quantity of anomalous problems—both empirical and conceptual—for all rival hypotheses.

Below I list (not exhaustively) many significant problems that the Sentinel Sleep Theory either solves or contributes to solving:

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(1) What is the survival value of REM sleep;
(2) Why REM sleep is biased toward potentially dangerous stimuli;
(3) Why rapid eye movements occur during REM sleep;
(4) Why REM sleep intensifies activity in the visual cortex;
(5) Why REM sleep is energetically costly;
(6) Why pronounced muscle atonia evolved;
(7) Why animals need to alternate between deeper and more active sleep;
(8) Why REM sleep occurs in cycles throughout sleep;
(9) Why REM sleep activates neural regions associated with threat detection;
(10) Why REM sleep activates neural regions associated with emotional processing;
(11) Why REM sleep activates neural regions associated with attention;
(12) Why REM sleep activates neural regions associated with pain processing;
(13) Why the amygdala is more strongly activated during REM sleep than during wakefulness;
(14) Why REM sleep (with few exceptions) is absent in animals exhibiting unihemispheric sleep;
(15) Why, upon awakening from REM sleep, the organism exhibits full alertness as well as high motor and sensory efficiency;
(16) Why REM sleep facilitates awakening;
(17) Why the amount of body fat affects REM sleep parameters (e.g., why total REM sleep time is lower in individuals with greater body fat);
(18) Why muscle strength affects REM sleep parameters (e.g., why total REM sleep time is lower in individuals with greater muscle strength);
(19) Why depression affects REM sleep parameters;
(20) Why cortisol affects REM sleep parameters;
(21) Why stress (in general) affects REM sleep parameters;
(22) Why exposure to novel stimuli affects REM sleep parameters;
(23) Why body immaturity affects REM sleep parameters;
(24) Why the immune system (particularly during its peak activity) affects REM sleep parameters;
(25) Why REM sleep facilitates learning and memory;
(26) Why the brain inhibits the LC-NE system during REM sleep;
(27) Why inhibition or suppression of REM sleep does not impair cognition;
(28) Why REM sleep must have an arousal threshold analogous to or even higher than that of N-REM sleep;
(29) Why REM sleep also involved the co-evolution of mechanisms that prevent its propensity to facilitate awakening from becoming so pronounced that it would disrupt sleep;
(30) Why REM sleep is necessary when N-REM sleep occurs in both hemispheres, but is dispensable when N-REM occurs in only one hemisphere;
(31) Why, when other factors remain unchanged, combined bodily vulnerabilities produce stronger effects on REM sleep parameters (e.g., increasing REM sleep amount and reducing its latency);
(32) Why, when other factors remain unchanged, combined bodily protections produce stronger effects on REM sleep parameters (e.g., reducing REM sleep amount and increasing its latency);
(33) Why REM sleep density is directly proportional to the amount (or level) of attention;
(34) Why REM sleep is so strongly associated with dreams;
(35) Why brain temperature increases during REM sleep.

All the hypotheses that scientists proposed for REM sleep to date can resolve only a very limited number of empirical problems, failing to solve numerous other significant empirical and conceptual problems. None of them can explain all the significant empirical problems that my theory resolves (to verify this, simply select any rival hypothesis and test it against the non-exhaustive list I presented above). In other words, my theory resolves a much larger number of significant empirical problems.

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Consider the hypothesis of the defensive activation of the visual cortex. It explains why the visual cortex becomes highly active during REM sleep. However, what can it say about all the other empirical and conceptual problems associated with REM sleep? Return to the non-exhaustive list above and you will see that the defensive activation hypothesis answers nothing with respect to numerous other problems. And what of the two hypotheses favored by researchers in the field—the learning hypothesis and the memory hypothesis? They can account for why REM sleep is so strongly associated with learning and the consolidation of memories—especially emotional ones. However, they are unable to resolve numerous other important problems. To cite a few, these hypotheses neither resolve nor help to resolve problems (2), (3), (6), (9), (11), (13), (14), (15), (16), (17), (18), (19), (24), (26), (27), (28), (29), (30), (31), (32), and (33) from my list.

Since my theory resolved a large number of significant empirical problems concerning the domain of REM sleep, it becomes irrational to deny its validity. This is because one would have to explain why some rival hypothesis—which now exhibits a greater number of anomalous problems—is more valid than my theory. Moreover, we must ask—and answer—why any previously proposed *hypothesis* should be given more weight than a *theory*. And even if my theory retains some remaining anomalies, the number of anomalies in the rival hypotheses is much greater. This is important because anomalous problems constitute evidence against a theory (Laudan, 1977, p. 18); or, in this case, against the rival hypotheses. Furthermore, clearly, the empirical problems that a theory resolves count in its favor (Laudan, 1977, p. 18). Consequently, considering all the empirical and conceptual problems that my theory resolves or helps to resolve, together with its smaller number of anomalies relative to any rivals, the rational choice is to accept its adequacy and robustness.

When assessing the merits of a theory, the problems it fails to solve are irrelevant. What truly matters are the problems—empirical and conceptual—that it does solve (Laudan, 1977, pp. 21-22). This does not mean that unsolved problems are unimportant, either for the theory or for science. After all, one reason theories make progress is that they come to solve a greater number of problems (Laudan, 1977, p. 21). What the foregoing assertion really means is that it is irrational to attach exclusive importance to the problems a theory has not solved. For if a theory solves more empirical and conceptual problems—specialy important ones—than any rival, we already have sufficient rational grounds to adopt it despite any remaining anomalies it may possess.

In sum, to deny the validity and robustness of my theory, one would have to disregard all the empirical and conceptual problems it resolves. Yet we cannot do so without offering good reasons, for no rival hypothesis is capable of accounting for all the problems that the Sentinel Sleep Theory resolves. Thus, if we reject my theory, such a rejection will lead us to a considerable loss in scientific efficiency in solving problems. This is because any rival hypothesis (so far proposed for REM sleep) can resolve far fewer empirical and conceptual problems than my theory and, as of this work, possesses a much greater number of anomalous problems.

8. How to Refute My Theory?

In Section 2, I sought to demonstrate that we must consider many nuances before assuming that a biological theory has been refuted. However, understanding these nuances is not the same as immunizing the theory against refutations. Therefore, in order to demonstrate more clearly that I am not attempting to immunize my theory from refutations, it is useful to present hypothetical refuting cases that would pose a serious challenge to it. These cases will add to the long list of hypotheses I already proposed to test my theory (see the Tables in this article, especially Table 2).

1. Discovering an animal lineage with bi-hemispheric sleep, highly vulnerable to predation, but that has completely lost REM sleep. If phylogenetic studies and sleep analyses were to reveal a lineage of mammals highly vulnerable to predation that demonstrably evolved from ancestors possessing REM sleep but eventually lost it, this would undermine the foundations of my proposal. Such a discovery would directly falsify my claim that REM sleep is a necessary adaptation for any organism that must sleep with both hemispheres. It would demonstrate that the "insurmountable vulnerability" of N-REM sleep is, in fact, surmountable, and that REM sleep is an optional

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evolutionary solution rather than an adaptive necessity. Thus, it would erode the pillar of evolutionary necessity upon which my theory rests. Especially if no other compensatory mechanism were evident, such a discovery would constitute a significant and fatal anomaly.

2. Discovering abundant REM sleep in animals that exhibit strictly uni-hemispheric sleep and that are evolutionarily more recent. For example, if we were to discover that cetaceans, in fact, possess a form of REM sleep that has thus far eluded our ability to observe and measure it, and that this REM sleep is abundant, then my theory would face a serious challenge. (This is why part of my mission with this work is also to foster further empirical research on cetaceans based on the conclusions I presented here.)

3. Demonstrating, through robust and independent research, that the primary function of REM sleep is anything other than the sentinel function. This would challenge my claim that the sentinel function is primary. In such a case, the high energetic cost of REM sleep would be justified by this other function, relegating the sentinel function to a secondary role. It should be noted that such a discovery would destabilize a considerable portion of the structure of my theory, since it is entirely grounded in the idea that REM sleep and its various physiological mechanisms evolved primarily to address the vulnerability of N-REM sleep.

4. Demonstrating that vigilance and alertness can be dissociated from REM sleep. If the characteristics and behaviors of alertness, vigilance, and readiness are not intrinsic to REM sleep but rather components that can be dissociated from it, then REM sleep could exist without being tied to a sentinel function. In that case, vigilance and attention to threats could not be its primary and defining biological function, and my entire theory—which links the physiological characteristics of REM sleep to the sentinel function—would be invalidated.

5. Identifying a highly efficient vigilance mechanism associated with N-REM sleep. This would dismantle the fundamental problem that my theory proposes to solve. If N-REM sleep already possessed a highly efficient and energetically cheaper sentinel mechanism, then the evolution of a second, far more costly system (REM sleep) to perform the same function would violate the principle of evolutionary parsimony. REM sleep would become a redundant mechanism and would possibly imply another primary function, thereby refuting my arguments that REM sleep evolved as a countermeasure to the vulnerability of N-REM sleep.

9. Limitations

As I reported before, due to the lack of studies, I was unable to verify the *hypotheses 26, 29, 32, 43, 44, 46, and 47* of Table 2. Those that refer to premature births are particularly difficult to test, as the presence of REM sleep in fetuses (although likely) is inconclusive (Okawa et al., 2017). What causes this difficulty is the fact that scientists cannot use the electroencephalogram (or EEG) on a fetus in the same way as they would on an adult; this is relevant because scientists rely on EEG data to identify sleep states (Okawa et al., 2017). Note that this lack of studies is not a weakness of the theory, but rather a testament to its ability to generate numerous new testable hypotheses. By identifying these specific knowledge gaps, the Sentinel Sleep Theory provides a clear and productive roadmap for future empirical research, which is a key function of a valuable scientific theory.

Due to the length of my Article, I was unable to address how all other hypotheses previously proposed for the function of REM sleep fit into the conceptual framework of the Sentinel Sleep Theory—a significant gap that we need to address in future work. This limitation, however, does not pose an epistemic threat to my theory. This is because, as I argued in Section 7, my theory solves a greater—and more significant—number of empirical and conceptual problems than any rival hypothesis. And it also has fewer anomalous problems relative to rival hypotheses.

A more significant limitation is the fact that I was unable to delve deeper into the circuits underlying the Sentinel Sleep Theory. While I outlined some contributions here, this is a work for future empirical and theoretical research. For example, some of the physiological questions of vulnerability and its association with REM sleep parameters that still require further elucidation may

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be resolved by studying the neural circuits of somatosensory regions and the neurophysiology and neurochemistry of such circuits.

10. Conclusion

The primary function of REM sleep is to reduce the vulnerability caused by N-REM sleep. I explained why and how REM sleep emerged, why and how it became more complex throughout the evolution of species, and why and how REM sleep parameters depend on factors associated with body protection or vulnerability. Considering all the strategies I adopted to select the references and mitigate any biases (see Section 2), it is safe to assert that the assertions I concluded from the data I analyzed are epistemically justified and produced results with methodological integrity.

A critical characteristic of a good scientific theory is its empirical testability. Thus, the greater the amount of empirical information a theory gathers (i.e., its *empirical content*), the greater the number of possibilities for falsification it contains (Popper, 2002, p. 96). The theory I am proposing here contains a vast empirical content, contributing to its robustness, given the numerous ways in which we can test it.

To test the Sentinel Sleep Theory, I drew on a substantial body of evidence and testable hypotheses. After testing the hypotheses and analyzing the evidence, I concluded that they corroborate the Sentinel Sleep Theory. Furthermore, I showed that numerous attempts to refute it failed. Finally, something even more important: I listed 51 specific hypotheses derived from my theory. Among which, 39 are empirically testable, which allows other scientists to exhaustively test the theory I proposed here—especially the hypotheses in Table 2 for which there is little or no research. 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.

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.

It is therefore no surprise that the brainstem—together with other cortical regions such as the amygdala and the hypothalamus—is responsible for generating REM sleep (Fraigne et al., 2015; Jouvet, 1962; Luppi et al., 2024; Maurer et al., 2024; Siegel et al., 1986). After all, this extension of the spinal cord encloses structures that control numerous *survival-related functions*, such as heart rate, respiration, orgasm, swallowing—and, of course, sleep (Angeles Fernández-Gil et al., 2010; Basinger and Hogg, 2023; Calabrò et al., 2019). And, as I am arguing, *REM sleep is a basic mechanism directly associated with survival*. The fact that the amygdala and the hypothalamus are also regions that generate REM sleep (Luppi et al., 2024; Maurer et al., 2024) is consistent with my argument that REM sleep plays a direct role in the survival of the organism. After all, both the amygdala and the

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hypothalamus are crucial neural regions in the physiological fight-or-flight response (Chu et al., 2024).

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 three main reasons for a scientific theory to be accepted as valid and robust are (1) the level of corroboration it has, (2) the number of attempts that failed to refute it, and (3) its ability to generate testable hypotheses. The Sentinel Sleep Theory 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, neurophysiological, endocrinological, immunological, neurobiological, neurochemical, neuropharmacological, ontogenetic, allometric, and even mathematical and statistical evidence (when I addressed probability and complexity). Additionally, numerous attempts to 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.) Given the 445 references I discussed here (most of which I used as empirical support), the evidence strongly suggests that I presented the central biological function of REM sleep. The numerous empirical evidence I gathered to corroborate the Sentinel Sleep Theory and the robust arguments I developed to demonstrate how they are explained are strong enough to ensure that no single Article is capable of disproving it. Especially because the quality of the data matters.

In light of all the arguments I developed to compose the conceptual framework of the Sentinel Sleep Theory, the numerous attempts that failed to refute it, the 38 factually confirmed hypotheses, and the 445 references I discussed here, it seems appropriate to state that the Sentinel Sleep Theory offers the most comprehensive and well-founded explanation to date 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, including its origin and subsequent evolution. 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.

The purpose of theories is to unweave reality, allowing us to understand a given phenomenon more accurately. That is why one of the most important marks of a good theory is its ability to solve problems. Consequently, the best theories are those that untangle the often-chaotic web of empirical facts that enmesh a scientific domain. With my theory, I collected a highly tangled assemblage of numerous disparate facts and explained them in a simple, clear, and precise manner. The arguments and evidence I presented here lead me to the rational belief that the Sentinel Sleep Theory was able to unweave the vast and chaotic web of facts that once entangled the domain of REM sleep.

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