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The Neuroscience of Sleep and Dreams

PATRICK McNAMARA

SECOND EDITION

The Neuroscience of Sleep and Dreams

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Patrick McNamara is Associate Professor of Neurology at Boston University, and Professor of Psychology at National University, USA. His research into sleep and dreams is ground-breaking and recognized as such. He has published numerous papers on sleep and dreams and authored or edited several volumes on the science of sleep and dreams. McNamara won a VA Merit Review Award and two NIH research grant awards for his work.

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The Neuroscience of Sleep and Dreams

Second Edition

Patrick McNamara

National University

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Dedicated to

Ina Livia McNamara

On her fifteenth birthday

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Preface

In the sandstone sculpture "Vishnu in His Cosmic Sleep," pictured in Figure P.1, we see symbolically depicted in Vishnu's posture and surrounding figures both of the major themes of this book: sleep and dreams. Vishnu sleeps and because he sleeps he dreams. He dreams of whole worlds: people, places, and things. The function of Vishnu's sleep, in fact, is to produce dreams. Sleep's relation to dreaming is one of the central questions we will explore in this book. But we will not neglect all of the other key findings in the emerging field of the neuroscience of sleep and dreams. This introduction to the neuroscience of sleep and dreams is part of the Cambridge Fundamentals of Neuroscience in Psychology series. The goal of this series is to introduce readers to the use of neuroscience methods and research to inform psychological questions. A key theme, therefore, of this book will be to introduce the use of neuroscience methods and research in order both to inform readers about the basic science of sleep and dreams and to illuminate psychological questions that arise around sleep and dreams. This book can serve as a supplemental textbook in college/university courses such as Brain and Behavior, Psychopharmacology, Neuropsychology, Behavioral Neuroscience, Psychology of Dreams, and Physiological Psychology; as a main textbook in a college/university course or seminar at an advanced undergraduate level or

Figure P.1 "Vishnu in His Cosmic Sleep" [sandstone sculpture]
Used with PERMISSION from Los Angeles County Museum of Art, Los Angeles, CA, LACMA.org

graduate level (along with supplemental scientific articles); or as a trade book for educated laypeople.

This second edition adds significant amounts of new material to every chapter and virtually every topic covered in the text. Most importantly, this new edition supplements the text with many new figures and tables to both illustrate points made and to supplement the information provided on each topic covered.

The American Academy of Sleep Medicine (AASM; Ramar et al., 2021) recently put out an official position statement on the relation of sleep to all aspects of mental and physical health. It is the position of the AASM that sleep is essential to health. I am in complete agreement with the AASM statement and will be presenting some evidence in this book that broadly supports the AASM position on sleep and health. Although it is now abundantly clear that sleep is essential to health for both adults and children, the AASM position statement notes that data from surveys conducted by the Centers for Disease Control and Prevention (CDC) and the Maternal and Child Health Bureau (MCHB) show that 34.1 percent of children, 74.6 percent of high school students, and 32.5 percent of adults fail to get a sufficient duration of sleep on a regular basis. In addition, the people who should alert the public to the need for better sleep are not getting the instruction on sleep they need to help the patients they serve. For example, according to the AASM position statement, a multination survey of medical schools found that the average amount of time spent on sleep education is just under 2.5 hours, with 27 percent responding that their medical school provides no sleep education at all. Similarly, a survey of pediatric residency program directors in ten countries found that the average amount of time spent on sleep education is 4.4 hours, with 23 percent responding that their program provides no sleep education at all. In addition, there is no formal training or certification in sleep medicine specifically for nurses and physician assistants (i.e., frontline health personnel). Only 6 percent of clinical psychology programs offer formal didactic courses in sleep medicine, with only 31 percent of programs offering training in the treatment of sleep disorders. Furthermore, actively practicing clinical psychologists in the United States and Canada reported a median of 10.0 hours of didactic sleep training across their training or career, and 95 percent of respondents reported no clinical sleep training at all.

In short, there is a clear and significant need for greater dissemination of the latest findings on sleep and its relation to health at all levels of society, but particularly in higher education and in clinical practice. I hope that this second edition of my introduction to the neuroscience of sleep and dreams will contribute to that overall effort to provide greater resources toward promoting sleep education. One shortcoming of the AASM position statement is that they

do not mention the role of dreams in health. We will see that dreams, in fact, are absolutely crucial for mental health and may also play a significant role in physical health. I will therefore present in this book some of the emerging research on the role of dreams and dreaming in the maintenance and promotion of mental and physical health.

Some of the questions I will be addressing include: What is sleep, and why are there two basic forms of sleep (REM and NREM, at least among terrestrial mammals)? Why is the amygdala activated and the dorsal-prefrontal cortex downregulated during REM? What is the evidence for immune system repair during slow-wave sleep? What is sleep debt and how is it related to brain function? What are the psychological consequences of chronic sleep debt? What do the major parasomnias teach us about conscious states? The many intriguing and bizarre clinical symptoms of various sleep disorders (sleepwalking, REM behavior disorder, narcolepsy, parasomnias, etc.) will be discussed, as well as the latest findings on the role of sleep and dreams in memory, learning, and mental health. With respect to dreams, some of the questions to be addressed are: Why do some people recall very few dreams, while others are flooded with dream memories on a daily basis? Why are social interactions so ubiquitous in dreams? Can certain dream experiences signal illness or even death? Why are some dreams extraordinarily moving and others quite banal and forgetful? Why do some people find it easy to realize they are dreaming when they are in fact dreaming ("lucid dreams") while others never achieve "lucidity"? Do we need to dream in order to remember things? Do we need dreams in order to be creative? How is the new rage for using smartphones and apps to track sleep patterns and dreams altering our understanding of sleep and dreams? What about nightmares? Why do they occur, and is there anything we can do about them? These are only a few of the fascinating puzzles concerning sleep and dreams that will be addressed in this book.

Unlike other introductory texts on sleep and dreams, I adopt a consistently evolutionary and social neuroscience approach to understanding the neuropsychology of sleep and dreams. I adopt this orientation as functional aspects of physiological systems are more easily understood within the framework of Darwinian evolutionary biology. To study sleep within an evolutionary context inevitably leads us to consider sleep as a social behavior, given that for most animals, fitness trade-offs occur within social interactions. I will therefore argue that sleep can be profitably studied and understood, at least in part, as a social phenomenon. For example, fetal and infant sleep cannot be understood in the absence of its social context, that is, the infant's interactions with its mother. Similarly, sleep states from toddlerhood up to adulthood also occur within social contexts (attachment relationships with parents in childhood and then attachment relationships with sexual partners in adulthood, etc.) that

shape all aspects of sleep expression. Sleep expression differs in the solitary sleeper as compared to co-sleepers. Co-sleeping is very likely the evolutionary default for human beings. Our ancestors were all co-sleepers, and that fact can help explain some of sleep's peculiar biologic features. While these elementary facts concerning sleep and social context have been assumed and occasionally acknowledged by sleep scientists, they have never received the sustained or explicit attention they deserve, it seems to me. Placing sleep within its social context will assist readers of this introductory text on the neuropsychology of sleep and dreams by illuminating the everyday functional aspects of sleep and its disorders.

One final note for this preface. This second edition of the text was written during the COVID pandemic of 2020–2022. After reviewing existing studies on the effects of lockdowns on sleep and dreams, it became clear that the argument we made in the first edition of this book – the social nature of sleep (and dreams) – was abundantly confirmed. The isolation and loneliness of many individuals during the COVID-related lockdowns significantly predicted altered sleep architecture, risk for nightmares, and unpleasant dream content. These sleep and dream outcomes, in turn, could either exacerbate COVID's effects on the individual or mitigate those effects, depending on individual differences and circumstances. Kahawage et al. (2022) content-analyzed responses of 997 people from all over the world to a survey about COVID and the subjective experience of social disruption due to social distancing/lockdown policies. In describing their subjective experiences around daily and nightly routines during social distancing procedures, people tended to report four major themes: (1) loss of daily timed activities; (2) the role of social interaction; (3) altered time perception; and (4) disruption to motivation and associated psychological effects. The overall sense most people reported feeling was being psychologically "adrift" in a time where sleep, dreams, and waking activities tended to blend in together. Participants described feeling disoriented in both time and place, where days blended into one another, and many reported that there was a sense of meaninglessness. Paradoxically, lockdown and social distancing mandates made people socially isolated AND sometimes trapped people into daily contact with housemates who they were not normally in contact with daily. The intensity of social contact in such cases tended to be a challenge, especially when the sleep–wake schedules of the other members were irregular and unpredictable. Interrelations among the four major themes were organized into a provisional heuristic map (Figure P.2). Once social distancing procedures were in place, psychological drift tended to emerge and then people had to choose how to handle that sense of drift. Successful responses to that drift often involved setting up regular exercise and sleep–wake routines and schedules. Failure to do so resulted in poorer outcomes and chronic distress.

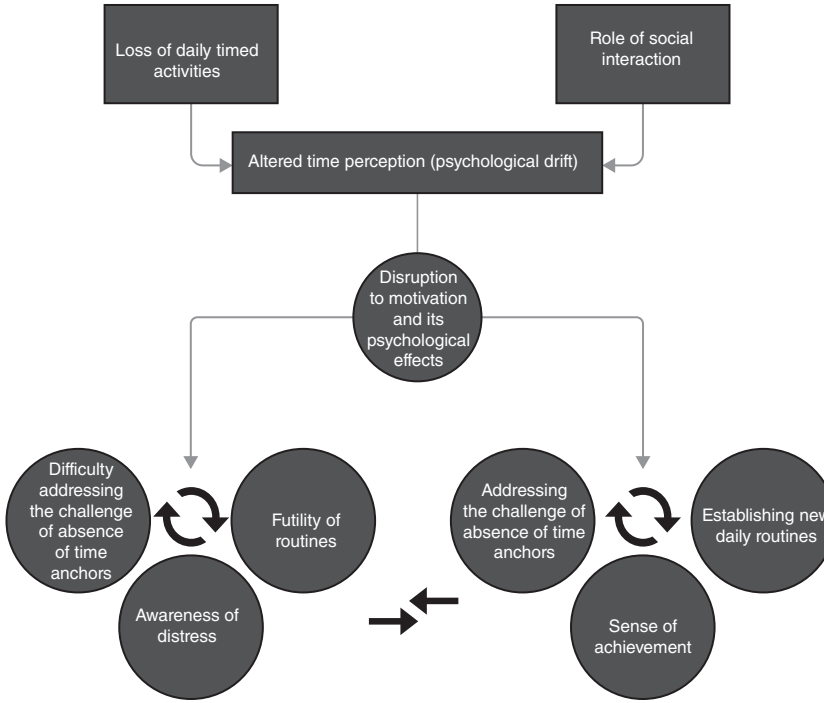

Figure P.2 Heuristic map of relationships between four themes involving COVID sleep effects
Used with permission from Kahawage et al. (2022)

Studies on the ways in which the dreaming brain responded to the pandemic, including the threat of illness and the associated social distancing policies, in many ways revealed the power of dreams. To take just one example, Šćepanović et al. (2022) utilized a deep-learning algorithm that extracted mentions of medical conditions from text and applied it to two datasets collected during the pandemic: 2,888 dream reports (dreaming life experiences) and 57 million tweets (waking life experiences) mentioning the pandemic. Analyses of these datasets revealed that health expressions common to both sets were phrases concerned with or depicting typical COVID-19 symptoms (e.g., cough, fever, and anxiety). Expressions in waking life, however, described symptoms in realistic or common-sensical ways (e.g., nasal pain, SARS, H1N1), while those from dreams appeared to be more metaphorical and uncommon (e.g., maggots, deformities, snake bites) or conditions of surreal nature (e.g., teeth falling out, body crumbling into sand). In the figure depicting these findings, note that the dreams (blue nodes) produce accounts of the COVID experience that appear to be exploring the outer edges or

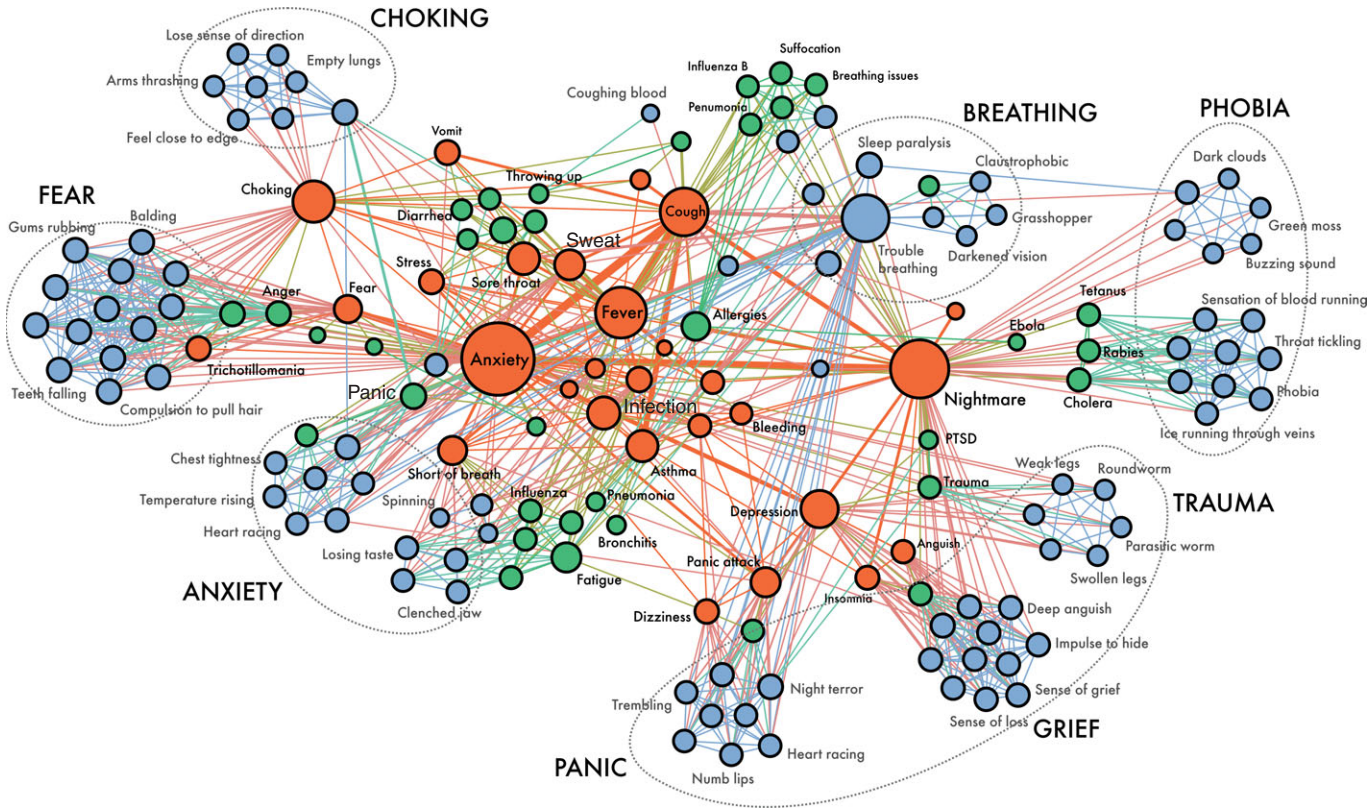

Figure P.3 The co-occurrence of medical conditions within the network of dream reports
Orange: disorders equally prevalent in waking and dream reports; green: typical of waking reports; blue: typical of dream reports). Select disorders in light grey are the conditions more prevalent in dream reports).
Used with permission from Šćepanović et al. (2022)

difficult-to-define margins of the experience. This kind of information is invaluable to people infected with the virus (to help prepare for what may come), to those helping ill patients, and to the rest of us who are just trying to cope as best we can with the daily challenges of a pandemic. In ordinary people, therefore, dreams both predicted and captured/described a larger part of the COVID experience than discursive waking reflections on the experience. No account of sleep, then, will be adequate without an account of dreams. There is no neuroscience of sleep. There is only a neuroscience of sleep and dreams.

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CHAPTER ONE

What Is Sleep? The Evolutionary Background

Learning Objectives

- Identify the homeostatic nature of sleep
- Evaluate the key elements in the scientific definition of sleep
- Evaluate the evidence for the social nature of sleep
- Distinguish biologic characteristics of reptilian, avian, mammalian, nonhuman primate sleep and human sleep

1.1 Introduction

Sleep is a need, just like food, water, or oxygen. If we don't get enough of it, we die. But right off the bat, we need to qualify this claim a bit. There are at least two forms of sleep documented in human beings: NREM (non–rapid eye movement) and REM sleep. While it is clear that we will, in fact, die without NREM, the case of REM is more complicated. Since this book is also an introduction to the science of dreams, it is worth digging into REM biology right at the start as well, as it is the form of sleep wherein we most often experience vivid dreams. Without REM, it seems, we will likely crumble mentally, but we may not die.

There are some indications, in fact, that REM is not only *not* necessary for life but may actually be harmful for life! Take, for example, the fact that REM involves general paralysis of the muscles (except eye muscles) so the individual is utterly unable to move (i.e., is paralyzed from the neck down). This REM-related paralysis, of course, makes us vulnerable to predators or other dangers in a way that the unconsciousness associated with NREM does not. It is obviously more problematic to emerge (e.g., to ward off a predator) from a sleep + paralysis state than it is from a sleep-alone state. To make matters worse, while the individual is paralyzed, the body in REM begins to undergo what are known as autonomic nervous system (ANS) storms involving intense and periodic electrical discharges in multiple ANS ganglia. These ANS storms lead to intense cardiac and systems instabilities, which in turn contribute to the fact that a disproportionate number of heart attacks occur during peak REM periods in the very early-morning hours (Verrier, Muller, & Hobson, 1996).

In addition to the paralysis and the ANS storms, oxygen desaturation levels are maximal during REM. The natural response to lowered O_2 levels is to increase inspiratory breathing, but this response (the hypoxic ventilatory response) is decreased by more than 50 percent of normal capacity during REM. Thus, REM-related hypoxemia and abnormal breathing patterns may cause life-threatening complications in vulnerable persons, including infants with immature lung capacity (perhaps contributing to sudden infant death syndrome) and in adults with various respiratory ailments and disorders. In addition, REM appears to involve a reversion to a poikilothermic state (Parmeggiani, 2000) – meaning that normal temperature regulation is lost during REM. Although brain temperature rises during REM, thermoregulatory responses such as sweating and panting do not effectively occur in REM. The body, of course, absolutely needs to effectively regulate its internal temperature and respond to any heat changes in the environment, but it cannot do so effectively during REM.

While the individual's physiology is erupting on all sides into multiple instabilities, REM adds yet another twist to the picture: Every REM period is associated with penile erections in males (even infant males), clitoral engorgement, uterine contractions, and sometimes pelvic thrusting in females. So while the individual cannot move, and while his or her physiology is in an uproar, the sexual systems are activated. If all these changes are not peculiar enough, consider now what happens to the brain during REM. First the eyes flit back and forth under the closed eyelids (yielding the name REM sleep), and then access to external sensory information is reduced and in some cases (e.g., visual information) effectively blocked. Nevertheless, REM-related brain waves beginning in the hypothalamus and medial septal and hippocampal regions called theta waves facilitate the activation of PGO (Pontine Geniculo Occipital) electrical spikes, which then propagate through the visual centers and then to other brain regions, subsequently recruiting and intensely activating select limbic and neocortical regions of the brain. In rats, REM-induced theta and gamma bursts in the hippocampus are followed by hyperemic bursts, or vascular surges, and then increased blood flow through all activated regions of the brain. These blood flow events in REM far surpass waking blood flow patterns in both rats and humans. Once REM theta activity in the hippocampus triggers PGO waves and its hemodynamic surges and its related limbic and neocortical activations, theta waves tend to become coupled with gamma waves – especially over ventromedial prefrontal cortical sites. At this point, the paralyzed, sexually activated, thermodynamically mute, and poorly ventilated but hypercognitively alert individual is then forced to watch these things we call **dreams**.

While disconnected from the external environment and entirely vulnerable to predators (due to the paralysis and closed eyes), and while our physiologies

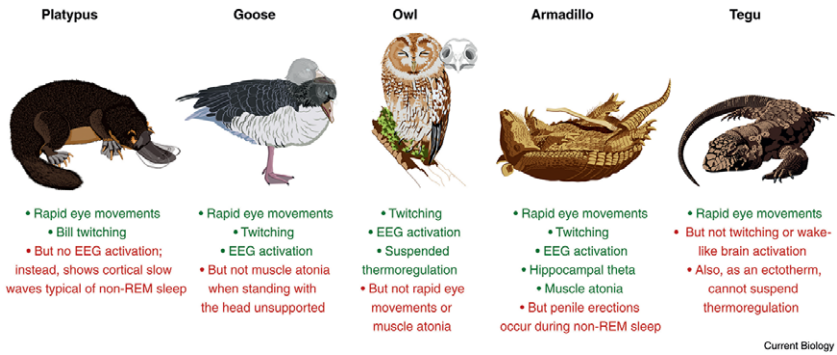

Figure 1.1 Animals that challenge traditional definitions of REM sleep
Used with permission from Blumberg et al. (2020)

undergo intense instabilities, including a bizarre sexual activation, we are transfixed by these hallucinatory simulations we call dreams. As the night progresses, these REM periods and intense narrative scenarios (dreams) regularly repeat at intervals of about ninety minutes in humans and become more and more violent, eliciting more intense ANS storms, serious fluctuations in heart rate and respiratory functions, and more elaborate and hallucinatory dream narratives . . . until we awaken dazed and confused from the night's adventures to face another day.

Blumberg et al. (2020) took note of these apparently risky, injurious, costly, and paradoxical properties of REM and then asked some fundamental questions concerning REM. Since all terrestrial mammals and birds have REM along with its associated temporary suspension of heat regulatory reflexes, should we conclude, for example, that REM functions to ensure endothermic requirements of the animal via the suspension of heat regulatory reflexes? As we will see shortly, some key electrophysiologic characteristics of REM actually occur in some animals, for example, the Australian bearded dragon (*Pogona vitticeps*), a reptile. Reptiles are incapable of producing heat internally or suspending that capacity during sleep, so suspension of heat regulatory reflexes during REM is not necessarily a universal or defining characteristic of REM. Blumberg et al. argue (see Figure 1.1) that given that evolution occurs through modifications of developmental processes, we should advocate a developmental-comparative approach.

From left to right, the platypus, a monotreme, exhibits a single mixed sleep state consisting of bill twitching and REMs (typical of REM sleep), but unlike REM, the cortical EEG exhibits slow waves. Unlike REM in other animals, geese and other birds demonstrate only partial reductions in neck muscle tone,

but they do not show general muscle paralysis during REM. Owls engage in REM sleep typical of most birds but lack REMs because their eyes are largely immobile within the skull (inset). Armadillos show classical aspects of REM except, unlike others, they (the males) do not exhibit penile erections. Instead, their sleep-related penile erections occur during NREM sleep. In contrast to the wake-like brain activity that traditionally defines REM sleep, sleeping tegus exhibit a novel 15-Hz brain oscillation, associated with REMs, that is not present during wakefulness. Also, as an ectotherm, they cannot suspend thermoregulatory effectors (e.g., sweating, shivering) like endothermic mammals and birds do during REM sleep.

In their examination of the diverse expressions of REM, Blumberg et al. (2020) argue that if we want to escape human- and mammal-centric understandings of sleep and if we truly want to understand the potential functions of sleep and dreams, we must adopt evolutionary and comparative perspectives. It is to that task we turn next.

1.2 Major Characteristics of Sleep

To move beyond human- and mammal-centric understanding of sleep, we nevertheless must begin with those models of sleep, as that is where we have some clear facts. But even though we must necessarily emphasize human sleep, we will consistently attempt to bring in comparative and evolutionary perspectives. First let's propose a provisional definition of human sleep. Human sleep is something more than a basic need. Human sleep is a restorative process that is brain state–regulated, reversible, homeostatic, embedded in a circadian and social-physiologic organization, and involving ritualistic signaling behaviors at sleep onset, a species-specific quiescent posture, some amount of perceptual disengagement and elevated arousal thresholds during sleep**.** That's a mouthful, I know, but we will go over each element of this definition in some detail and note some exceptions along the way (e.g., REM sleep is not homeostatically regulated in all nonhuman species, while NREM is). Most organisms that sleep, however, exhibit at least several elements of this definition derived from human sleep.

Note that this definition is composed of behavioral, functional, physiologic, and electrophysiological traits (see Table 1.1). For most nonhuman animals, sleep can only be identified via measurement of its behavioral and functional sleep traits, as their nervous systems do not support what has become known as full polygraphic sleep, or sleep that can be measured with an electroencephalograph machine that records brain waves through the skull. Full polygraphic

Table 1.1 Human sleep traits

1 Behavioral
- –Typical usually quiescent body posture
- –Specific sleeping site
- –Behavioral rituals before sleep (e.g., circling, yawning)
- –Physical quiescence
- –Elevated threshold for arousal and reactivity
- –Rapid state reversibility
- –Circadian organization of rest–activity cycles – entrained to and sensitive to social cues and to activities of conspecifics
- –Different from hibernation/torpor

2 Electrophysiological
- –EEG
- –NREM: high-voltage slow waves, delta power (quiet sleep)
- –Spindles in some animals
- –K-complexes in some primates
- –REM: low-voltage fast waves (REM, paradoxical sleep, or AS [active sleep])
- –Hippocampal theta; PGO waves
- –Electro-oculogram (EOG)
- –NREM: absence of eye movements or presence of slow-rolling eye movements
- –REM: rapid eye movements
- –EMG
- –Progressive loss of muscle tone from wake → NREM → REM

3 Physiological
- –REM: instabilities in heart rate, breathing, body temperature, etc. Other: penile tumescence
- –NREM: reduction in physiologic/metabolic processes; reduction of about 2°C in core body temperature

4 Functional
- –Compensation of sleep deficit (homeostatic regulation)
- –Enhancement of sleep time after sleep deprivation

sleep refers to electrophysiologic measures of both REM and NREM sleep stages: N1, N2, and N3, identified via the electroencephalogram (EEG). It has become common, however, to use the term "full polygraphic sleep" to refer to an animal exhibiting most or all of the other three major components of sleep in addition to the electrophysiologic measures. When an animal exhibits all

four major components of sleep, including the behavioral, electrophysiologic, physiologic, and functional components, then it is said that the animal exhibits full polygraphic sleep. Full polygraphic sleep, in this sense, has so far only been documented in primates (including humans).

Behavioral traits of sleep include a species-specific body posture that typically involves a recumbent nonmoving animal (quiescence), though some animals can engage in some limited sleep while standing (e.g., cows). There is also typically a species-specific sleeping site that is constructed to conserve warmth and protect the sleeping animal from predators. Before relaxing into the sleep site an animal usually engages in behavioral rituals such as circling the nest and yawning before laying down to sleep. It is unclear why these behavioral rituals are needed before sleep. Other behavioral indicators of sleep include reduced muscle tone, reduction in neck/nuchal muscle tone, paralysis of the antigravity muscles in some species, increased arousal threshold, and rapid reversibility to wakefulness. Physiologic indices of sleep include significant reductions in metabolic activity during NREM and significant lability in the autonomic nervous system activity (ANS), as well as cardiovascular and respiratory measures during REM. Electrophysiologic measures of REM include low voltage fast waves, theta rhythms in the hippocampus, and pontine-geniculo-occipital or PGO waves. Electrophysiologic measures of NREM include high voltage slow waves, spindles, and K-complexes. Functional indices of sleep include increased amounts of sleep after sleep deprivation and increased sleep intensity after sleep deprivation – though there may be exceptions to this functional rule. For example, frigate birds when flying over the ocean for ten-day periods sleep less than one hour per day and do not exhibit any sleep rebound after their migratory flight. Fur seals exhibit about eighty minutes of REM sleep per day when they are on dry land, but when swimming in water they average about three minutes of REM a day. They exhibit no REM "rebound" when they return to land.

1.2.1 Sleep Is a Homeostatically Regulated Process

Despite rare exceptions (e.g., frigate birds or fur seals) to the rule of rebound sleep after periods of sleep deprivation, most animals do exhibit sleep rebound. Sleep, by and large, is homeostatically regulated. By homeostatically regulated we mean that the amount and intensity of sleep you experience is controlled by a kind of internal thermostat. If you get too little sleep you cumulate a sleep debt that needs to eventually be paid back or made up. To make up for lost sleep time you sleep a bit longer and a bit more intensely on subsequent nights. In short, you sleep in proportion to wake time – at least with respect to NREM sleep. The longer the wake time (or the greater the amount of sleep

deprivation), the greater the subsequent NREM sleep time and intensity. Lots of people sleep little during the work week and then sleep in (or sleep longer) during the weekend. In other words, we make up for lost sleep time during the work week by sleeping more on the weekends.

1.2.2 Sleep Is Restorative

If we use an electroencephalograph or EEG (to be discussed more fully later) to record brain waves during catch-up sleep, we see that the brain exhibits a lot of so-called delta power. The longer the wake time before sleep, the stronger or higher the delta power during NREM sleep. Once you go to sleep, however, delta power begins to decline across the night, indicating that delta signals "need for sleep" or the intensity with which you sleep during catch-up sleep. The greater the delta power, the more intense the catch-up sleep. When delta power is high at the beginning of the night and declines across the night, people report high-quality sleep. So it is not so much the amount of sleep but the intensity of sleep that counts in catch-up sleep. The more intense the sleep as measured by delta power, the more refreshing the sleep. Thus, we can pay back a sleep deficit by sleeping more intensely and sleeping longer.

The phenomenon of catch-up sleep suggests that something, some chemical process within the body or the brain perhaps, builds up during wake and is discharged during sleep. Delta activity (during slow wave sleep; N3) indexes the efficiency with which this wake-related chemical process, call it Process S, is discharged. Delta power is doing something that reverses whatever Process S induces during wake. For example, if Process S is some sort of fuel for body and brain that gets depleted during waking, then delta power would presumably index some sort of manufacturing process that produces some chemical that would refuel the body and brain. If we could identify the physical factor responsible for Process S, we could possibly gain insight about the actual function of NREM sleep. The relationships between sleep deprivation and the nature of catch-up or recovery sleep are important because they give us clues as to what it is that sleep actually does for us, that is, the function of sleep.

Is that function some sort of energy conservation Process S that refuels the body? Maybe, but the picture is complex. Consider, for example, hibernation. Hibernation is an adaptation that allows some warm-blooded animals to engage in a period of prolonged inactivity with dramatically reduced needs for food and warmth. Bears are the great hibernators. Hibernation in the bear is triggered by a gradual waning of their circadian rhythms of sleep and wake. Bears actually regulate their body temperatures by shivering and increasing their metabolic rates. They show mostly NREM sleep and REM sleep with brief episodes of wakefulness throughout the hibernation season. The animal

finds a well-protected site like a cave or constructs a burrow or rest site and then hunkers down for the winter. It looks like the animal is sleeping, but it is not. This shows that sleep is not mere quiescence or inactivity. The hibernating animal reduces its core body temperature and metabolic activity and enters a period of immobility, but it still needs to sleep! Given that the hibernating animal is in a very reduced metabolic state, it is not at all clear that its body is working or creating a metabolic need for refueling. Yet, the bear periodically arouses and enters into slow wave sleep as if it is catching up on sleep. Hibernation must be construed as a state such as waking in that it apparently involves some kind of work and deprives the organism of sleep. Hibernation allows the animal to survive periods such as long winters when food is scarce and there is no benefit to expending calories looking for it. Torpor does the same for animals such as squirrels, but these animals only need to be in torpor for short periods of time, not the entire winter. While the animal in torpor can drastically reduce its need for food and water and warmth, it cannot reduce its need for sleep, so it must periodically arouse and engage in sleep. When an animal arouses out of torpor it immediately goes into slow wave activity (SWA) as if to make up for lost sleep. But the amount of slow wave sleep the animal engages in appears to be tied to its body and brain temperature, as the lower the temperature of the brain, the greater the SWA immediately following the arousal. Given these facts concerning hibernating animals, it seems clear that sleep's function cannot be solely that of metabolic refueling.

1.2.3 The Role of Local Slow Waves in Restorative Sleep

It is important to note that there are two broad ways in which the body and brain recover metabolically after doing work. One is a top-down process that puts the whole organism asleep all at once. The other occurs from the bottom-up and involves local groups of neurons generating synchronized activity that manifests as slow wave activity in these local small networks of neurons. Therefore, in addition to the exponential decline of delta power across a single night of sleep, local transient increases in various regions of the brain in delta power can also occur, in relation to the amount of use of that area of the brain. These local increases in sleep or delta power indicate use-dependent functions of recovery sleep. That is, if you use a part of the brain intensely during some waking activity, that area of the brain will evidence local increases in delta wave activity, thus indicating some sort of recuperative process for that particular brain region. The regular, more global changes in delta wave activity that occur each night appear to be more strongly related to use or engagement

of particular regions of the frontal lobes and its interconnected regions than to other areas of the brain (Halasz et al., 2014). Sleep deprivation, in general, has been shown to enhance the frontal predominance of delta-indexed SWA during recovery sleep (Horne, 1993; Cajochen et al. 1999; Finelli et al., 2001), especially in the left hemisphere (Achermann et al., 2001).

1.2.4 The Unusual Regulation of REM in Relation to Restorative Sleep

While it is clear that NREM is homeostatically regulated (given that the deprivation of NREM sleep during waking hours leads to a dramatic increase of EEG delta slow waves that are then dissipated across the night resulting in recovery sleep), the case is more complicated for REM. While there is some evidence of the sleep rebound phenomena associated with deprivation of REM sleep, it is unclear whether an intensity dimension (as measured by delta waves in NREM) exists for REM sleep. With REM rebound we see an increase in the density of rapid eye movements, but it is unclear if that indexes increased sleep intensity. Nor does REM rebound occur in all cases or across all species. While the pressure or need for NREM increases in proportion to the amount of work done during waking hours, the need (or pressure) for REM sleep is not clearly proportional to work done during waking alone. It may be that REM pressure increases in proportion to wake time as well as NREM sleep duration, or both (Benington & Heller, 1994). To complicate matters further, REM sleep quotas and intensity appear to also be influenced by circadian cues (Dijk & Czeisler, 1995) and genetic factors (Franken et al., 1998).

When we look at the neural machinery that regulates REM expression, the interactions with NREM circuitry becomes more clear as the two states appear to mutually inhibit one another. Pathological brain states occur when they (REM and NREM) intermix. If on the other hand, REM and the waking state intermix, the individual subjectively experiences a dreamy – even hallucinatory – state. Thus, we need circuitry that keeps the three states from intermixing. REM and NREM alternate with one another, avoiding the wake state over the course of the night and are regulated by neural circuits that turn REM on or off in tandem with NREM. An early proposal by Hobson and McCarley (1975) noted that REM expression was regulated by mutually inhibiting cholinergic and monoaminergic nuclei. When monoamines (serotonin and noradraline) are low and cholinergic activity is high in brainstem sites, REM is expressed. More recently, other neuromodulators have entered the picture, again playing mutually inhibitory roles in regulating expression of REM. For example, a role for mutually inhibiting GABAergic neurons and orexinergic neurons contained

within REM-on and REM-off brainstem regions has been documented (Mignot et al. 2002; Saperet al., 2005; Lu et al., 2006). The onset of REM sleep may be regulated by hypothalamic GABA neurons through the recruitment of the extended ventrolateral preoptic area (Lu et al., 2002). In addition, a subset of melanin-concentrating hormone (MCH) neurons in the lateral hypothalamus are active at REM onset and fire exclusively during REM sleep, whereas MCH-negative GABA neurons in the lateral hypothalamus increase firing as NREM progresses to REM sleep. To transition out of REM sleep, the hypothalamic orexin/hypocretin neurons activate until the wake state emerges.

1.2.5 Sleep Is a Reversible State

By reversible state we mean that when you go to sleep, you are not stuck there as may be the case with an irreversible coma. With appropriate forms of stimulation, such as a loud noise and a vigorous shaking, you can return to wakefulness. Typically the brain spontaneously shifts itself out of sleep and back into waking after about five to eight hours in adult human beings.

1.2.6 Sleep Is Brain State Regulated by Both Top-Down and Bottom-Up Processes

At the organismal level, the brain initiates sleep, maintains sleep, promotes transitions between one form of sleep and another, and triggers wakefulness – it is therefore a brain-dependent and regulated process through and through. But as mentioned, SWA emerges spontaneously after some amount of work is done with even a small number of neurons. Spontaneous SWA is a local bottom-up process that serves to facilitate recovery in these local neuronal networks. As more and more local regions engage in limited SWA, synchronization of SWA across these small networks starts to coalesce and spread to larger regions of the brain until large sweeping slow waves propagate throughout many regions of the brain, resulting in NREM.

This ever-spreading SWA synchronizes widespread regions of the brain, resulting in globally diminished brain activity. Both PET (Maquet, 1995; Hofle et al., 1997; Dang-Vu et al., 2005) and fMRI (Czisch et al., 2004; Kaufmann et al., 2005) studies have shown that global cerebral activity, including that of frontal areas, progressively diminishes with deepening NREM sleep. The transition from waking to NREM involves a prominent thalamic deactivation in addition to widespread cortical and subcortical deactivations (Hofle et al., 1997; Dang-Vu, 2005). Progressive deactivation within NREM sleep appears to be centered on regions in the social brain network including the medial prefrontal cortex, anterior medial areas in BA 9 and 10, orbitofrontal cortices

(BA 11), caudal orbital basal forebrain, anterior cingulate (BA24), bilateral anterior insula, basal forebrain/anterior hypothalamus, bilateral putamen, and left precuneus (Dang Vu et al., 2005).

1.2.7 Sleep Is Embedded within Circadian and Social-Physiologic Organization

Sleep occurs within a circadian or twenty-four-hour cycle and within a social context. In humans and other primates, sleep is usually monophasic or consolidated into one large sleep period at night, although some studies of sleep among hunter gatherers suggest that human sleep may be naturally biphasic with one long bout at night and then another short bout in the late afternoon. In most mammals, however, sleep is polyphasic, with bouts of sleep occurring during the day and night. In species such as the cat and the guinea pig, sleep occurs in short bouts at virtually any time of the day or night. The factors responsible for the different patterns of sleep cycle phasing remain unclear, but keeping open the ability to pursue social interactions is likely one such factor. Prolonged quiescence makes an animal vulnerable to predation and it also increases the chances that it will miss out on opportunities for social alliances or reproductive opportunities. Therefore, if an animal can obtain the benefits of sleep in a few short bouts rather than in one long prolonged bout, that option will be common.

In nonhuman primates and humans sleep is exquisitely sensitive to social cues. Although biological rhythms are entrained to light–dark phases, social cues can dramatically influence expression of sleep. We cannot fall asleep when we cross time zones not just because the light cycle is off but also because the people around us are not sleeping. In nonhuman primates and possibly also in humans, contagious yawning signals to conspecifics that a sleep phase is approaching and sleep partnerships need to be initiated. Once sleep commences, individual physiologic systems appear to assume that individuals are not sleeping alone. For example, in REM, thermoregulatory reflexes are relaxed probably because warmth can be conserved by sleeping next to another warm body. Sexual activation also occurs during REM for most mammalian species. This activation can be seen as nothing more than a byproduct of brainstem activation during REM or as an opportunistic function given the assumption that the individual does not sleep alone. When sexual partners co-sleep their brains and bodies and their biological rhythms become intertwined and resonant as do co-sleeping babies and mothers. In the infant, several forms of nighttime signaling such as crying, sucking, nursing, smiling, grasping, twitching, cooing, babbling, and other vocalizations influence the mother's sleep/wake patterns as well as daytime attachment processes. All of

these behaviors are more likely to either occur in, or emerge from, REM than NREM sleep. Co-sleeping with the parent influences number and duration of night-waking episodes in the infant (McKenna et al., 1990, 1993, 1994) as well. In general, bed-sharing infants are more likely to nurse and, in turn, are more likely to awaken more frequently during the night to feed.

In ancestral communities children very likely grew up co-sleeping as well. When children reached maturity they graduated into sleeping with sexual partners and/or with extended family members. It is only in the last hundred years or so that human beings began to sleep alone in a minority of rich countries in Europe and North America. Throughout human history sleep has been a social behavior.

Box 1.1 Social isolation and sleep

There was a gigantic global experiment on the effects of social isolation on sleep patterns due to COVID pandemic lockdowns. Piclher et al. reviewed the extant literature in 2022. They (Pilcher et al., 2022) found that studies surveying populations across more than a dozen countries tended to find that about a third of adults reported less sleep and poorer quality sleep. Adolescents tended to report greater amounts of sleep, though not more high-quality sleep. A common pattern was that at the onset of quarantines there were delays in sleep timing including later bedtimes, mid-sleep times, and wake-up times, but then after adjusting to the new routines, people tended to settle into patterns of less sleep and poorer quality sleep, particularly for those most isolated (i.e., those living alone).

Gemignani et al. (2014) studied the effects of social isolation on sleep in a simulated spaceflight environment in six healthy volunteers who lived in the spaceship simulator Mars (MARS500) for 105 days. Volunteers were sealed in the spaceship simulator for 105 days and studied at five specific time-points of the simulation period. The researchers found that although cortisol levels were within normal limits, higher cortisol levels were associated with fragmented sleep in the form of shorter sleep durations, increased numbers of arousals, reduced REM latencies, reduction of delta power, and enhancement of sigma and beta in NREM N3. Social isolation, even with cortisol fluctuations in the normal range, significantly alters sleep structure and sleep EEG spectral content.

The effects of social isolation on sleep are perhaps most strongly felt by people living alone. Many older adults find themselves in that situation. Using the largest national database to date McLay et al. (2021) found that loneliness and social isolation is associated with sleep problems among older community–dwelling women and men with complex needs. In an analysis of 140,423 sleep and social assessments from 95,045 (women: 61.0 percent) community-living

Box 1.1 (cont.)

older adults aged sixty-five years and older, having standardized home care visits, overall, 23.6 percent of women and 18.9 percent of men reported feeling lonely, while 53.8 percent of women and 33.8 percent of men were living alone. In adjusted longitudinal analyses, those who were lonely and socially isolated experienced significantly poorer quality sleep and shorter sleep times.

1.2.8 NREM Is a Quiescent State While REM Is Not

Quiescent state simply means reduced physical activity relative to the resting waking state. So imagine your most relaxed, resting state during waking life and then reduce physical activity even more and eventually you will arrive at the NREM quiescent state. Quiescence does not require complete cessation of physical activity. You can observe some minor movement during the different phases of sleep. During NREM most cerebral brain networks are largely synchronized, which is consistent with the overall quiescence of the organism. In REM, however, the brain is not quiescent. Instead, selective neural networks are actually more active than they are during the waking state. We will discuss which neural networks are more active in REM versus waking consciousness in a later chapter.

1.2.9 Sleep Is Also Characterized by Perceptual Disengagement

One of the most striking characteristics of a sleeping animal or person is that they do not respond normally to environmental stimuli. If you open the eyelids of a sleeping mammal the eyes will not see normally – they are functionally blind. Some visual information apparently gets in, but it is not normally processed as it is truncated or attenuated; same with the other sensing systems. Stimuli are registered but not processed normally and they fail to rouse the individual. Perceptual disengagement presumably serves the function of protecting sleep, so some authors do not count it as part of the definition of sleep itself. But insofar as sleep would be impossible without it, it seems essential to its definition. Nevertheless, many animals (including humans) use the intermediate state of drowsiness to derive some benefits of sleep without total perceptual disengagement. In the drowsiness state the eyelids are half closed and eyes continue to process visual stimuli normally. Microsleeps, where the animal dips fleetingly into deep sleep and then quickly arouses again into drowsiness, happen continuously under drowsiness.

1.2.10 Sleep Is Associated with a Species-Specific Posture and Eye Closure

In most terrestrial mammals sleep occurs at a specially constructed sleep site with the animal in a recumbent position and with eyes closed. Animals construct sleep nests in order to protect against the cold and predators, and to co-sleep with a sleeping partner or set of partners, but it is unclear why most animals sleep with their eyes closed. Is it because closing the eyes protects sleep? If your eyes are closed you are less likely to see things that will wake you up. But many animals sleep with their eyes only half closed (ruminants) or with one eye open (some aquatic mammals and some birds). Some people actually can sleep with their eyes open, so the purpose of eye closure during sleep may not be due solely to the need to protect sleep.

In birds and in some aquatic mammals (such as dolphins and whales) sleep occurs in one brain hemisphere at a time. The open eye in these species is usually contralateral to the hemisphere that is asleep and thus it is reasonable to assume that the open eye is transmitting information primarily to the awake hemisphere rather than the asleep hemisphere. It is possible that some information from the pathway from the open eye to the awake hemisphere leaks over to the pathway to the asleep hemisphere. In any case, unilateral eye closure (or keeping one eye and hemisphere awake) functions to allow the animal to "sleep on the wing"; that is, aquatic mammals can continue to swim while one hemisphere sleeps, and birds can continue to fly while one hemisphere sleeps. Some deep cave–dwelling fish who have lost their eyes due to lack of sunlight sleep very little or possibly not at all.

Most terrestrial animals sleep in a protected site in a recumbent position. In other words, they lay down in their sleep nest and then fall asleep. Laying down presumably conserves energy, but conservation of energy cannot be the whole story as the brain is very highly activated during REM sleep, thus precluding energy conservation from being a major causal factor in sleep recumbency. It may be that animals lie down to sleep simply because any other posture is incompatible with the muscle atonia and paralysis associated with REM. Ruminants such as cows can sleep while standing. Not surprisingly they exhibit very little REM. Sea otters, on the other hand, prefer to sleep floating on the oceans' surface. Bats sleep while hanging upside down from a cave wall. Lying down may also help to conserve heat, especially if you are co-sleeping with another. Many juvenile mammals sleep next to siblings or mothers, thus deriving heat, comfort, and protection from these relatives. Sleep is not a passive process in mammalian juveniles as they can grasp, suck, and snuggle while asleep. Sleep in the juvenile rat, for example, "expects" a

social environment and appears to be adapted to sleeping in groups near a mother who provides heat, protection, and nutrients. Adult rodents sleep curled up in groups within a hidden niche or burrow.

Sleep sites vary systematically with social organization in primates (Anderson, 1998). Social relationships among individuals in a group influence arrangements of sleeping clusters in primates. Kin relations, reproductive status, and dominance relations influence spatial and huddling relations during sleep. Fruth and McGrew (1998; see also Fruth & Hohmann, 1993) have noted that among the great apes, a number of filiative and cooperative interactions such as play, grooming, sexual encounters, and infant nursing take place in the nests at sleep sites. These sleep-onset filiative social interactions suggest that sleep processes themselves are intimately shaped by social needs among the great apes.

1.2.11 Sleep Is Associated with Elevated Arousal Thresholds

One of the defining features of sleep is that it is difficult to arouse the sleeping animal with sensory input that does not exceed a threshold of touch, loudness, or light, etc. The sensory input has to go beyond that threshold to wake someone up. The brain employs protective mechanisms to keep you asleep once you are asleep. If a noise occurs within the room you are sleeping in, the brain will take that information and suppress it so that it does not wake you up. The brain uses neuronal inhibitory mechanisms to prevent the sound information from reaching arousal centers of the brain. Those inhibitory mechanisms are sometimes indexed by so-called K-complexes and sleep spindles, which I will discuss more fully shortly.

1.2.12 Sleep Onset Is Associated with Ritualized Social Signaling Behaviors

When animals are about to go to sleep they engage in ritualistic signaling behaviors such as self-grooming, nest circling, sleep-site tending, vocalizations, and yawning. In primates, yawning may be contagious such that if I see or hear you yawning I will experience an irresistible urge to do so myself. Yawning may be contagious because it can function as a signal to conspecifics that can help synchronize sleep times among these conspecifics. For example, once one monkey yawns within a troop, other monkeys begin to do so and then a suite of behaviors kick in: searching for a suitable sleep site, construction of a sleep site, ritual circling of the sleep site, and choosing partners to sleep with

and then bedding down, etc. At an individual level yawning is associated with attempts to change the brain state either from quiescence into more alert states or from alert states into quiescence. Yawning appears to occur in all mammals, in some birds, and may even occur in reptiles. Yawns often involve involuntary openings of the mouth, inspiration of a breath, closing of the eyes, and stretching of torso and limbs. Like REM sleep, yawning is associated with cholinergic excitation and dopaminergic inhibition. Oxytocin and testosterone infusions can induce yawns as well. Interestingly, when oxytocin is injected into the paraventricular nucleus or the hippocampus it induces both yawning and penile erections (Argiolas & Gessa, 1991). REM sleep, too, is associated with erections. Yawning even occurs in the fetus. The yawn's wide taxonomic distribution in the animal kingdom suggests an ancient lineage as well as important functional relationships with sleep states.

1.2.13 Interim Summary

We have defined sleep and examined some of its key behavioral, functional, physiologic, and electrophysiological traits, so we are now in a position to examine its expression across the animal kingdom. While examining sleep characteristics in various animal species we will keep our eyes focused on what comparative sleep data can tell us about functional aspects of sleep.

1.3 Comparative Sleep

Our survey of sleep across the animal kingdom will begin with simple organisms and then proceed to species ancestrally related to us. That will enable us to consider what is special and unusual about human sleep.

1.3.1 Invertebrates

Invertebrates are animals without a backbone and tend to be simple organisms such as Drosophila or fruit flies. NREM- and REM-like activity has been documented in these insects. Their NREM-like phases of sleep exhibit brain-wide slow oscillations of neuronal activity within the ellipsoid body. Fruit flies also exhibit some evidence of sleep rebound after sleep deprivation. Worms such as Caenorhabditis elegans sleep during lethargus, a two to three hour period at the transition between larval stages. Their sleep appears to be promoted by two second-order interneurons called ALA and RIS, both of which manufacture a cocktail of neuropeptides that promote quiescence.

1.3.2 Vertebrates

Fish

There are more than 33,000 species of fishes inhabiting vastly different aquatic niches ranging from tiny and transient rainwater pools to the world's vast oceans. Using fluorescence-based polysomnography (fPSG) in zebrafish, Leung et al. (2019) monitored muscle tone, heartbeat, and eye movement as well as brain activity across rest-activity cycles in these animals. The authors documented a quiet sleep phase called slow bursting sleep (SBS) and a more active sleep phase called propagating wave sleep (PWS). SBS rebounds after sleep deprivation. Like mammalian REM, PWS is characterized by muscle atonia and twitches, brainstem activation, and a posterior–anterior wave of neural activity propagating from the pons. Similarly, cuttlefish are mollusks that display two phases of sleep, characterized by no color changes or no chromophore activity, and intense chromophore activity and eye movements. Octopuses display a complex pattern with 50 percent of the day spent in quiet sleep and then sudden bouts of active sleep but making up no more than eight minutes per day. Fish species that live in deep caves without sunlight lose pigmentation, functional eye systems, and to some extent circadian regulatory systems. The loss of light-entrained circadian rhythms in some of these animals along with some preserved sleep patterns and the reduction in sleep despite preserved circadian rhythms, suggests that the two systems can operate independently. But most of the time sleep is dramatically reduced in cave fish.

Particularly important for an analysis of sleep's evolutionary history and functions is to identify changes in sleep patterns as a function of divergences between species in evolutionary pathways. Modern mammalian and avian lineages, for example, are thought to have diverged from their reptilian ancestors about 250 million years ago. Modern extant forms of reptiles may retain some of the sleep characteristics of their ancestors who flourished before the rise of mammals and we mammals, in turn, may inherit some of the features of reptilian sleep. Thus, studies of modern reptiles may reveal the form of sleep from which mammalian and avian sleep evolved.

Reptiles

Although birds are more closely related to reptiles than they are to mammals, the sleep processes of birds are more similar to mammals than they are to reptiles. Yet clear unequivocal electrophysiologic signs of both REM and NREM sleep states have been identified in birds and mammals but not as clearly or unequivocally in reptiles until recently. High-voltage slow waves (HVSW) or high-amplitude spikes and sharp waves appearing in tandem with clear behavioral

signs of sleep (e.g., eye movement patterns or arousal thresholds) in reptiles have been proposed as reptilian precursors of SWA found in the sleep of mammals. The equation of reptilian HVSW with mammalian SWA is supported by findings of compensatory rebound of sleep-related processes including EEG spikes after sleep deprivation (SD) in some reptiles. Karamanova (1982) argued that some reptiles evidenced these electrophysiologic precursors to REM and NREM sleep. Shein-Idelson and colleagues (2016) identified in the Australian dragon lizard, Pogona vitticeps, electrophysiologic signs of REM and NREM sleep states that are similar to those seen in mammals and birds. What was most interesting in this report was that the lizard's REM and NREM sleep phases alternated with one another just as they do in mammals. A phase characterized by low-frequency/high-amplitude sharp waves (homologous to mammalian slow wave sleep) alternated with a phase characterized by awake-like brain activity and rapid eye movements (homologous to mammalian REM). In Pogona, SWS and REM alternate regularly throughout the night with a short period (about 80 seconds), generating up to 350 SWS-REMS cycles (compared with four to five ninety-minute cycles in humans). Shien-Idelson et al. also recorded coordinated activity of the lizard's cortex with the dorsal ventricular ridge during slow wave sleep. Similar coordinated neural activity occurs in mammals between the cortex and the hippocampus and may underlie memory consolidation processes in mammals.

1.3.3 Avian Sleep

Birds show the same EEG characteristics of NREM and REM sleep as do mammals, but REM sleep periods typically last only a few seconds in birds (though there are many of them in any given sleep period). The percent of total sleep time occupied by REM in birds is less than half that of mammals. Birds may be able to sleep "on the wing" during periods of migration. When the migratory white crowned sparrows are studied under laboratory conditions they show "migratory restlessness." When they begin to show migratory restlessness they dramatically reduce the time they spend in sleep (only 13 percent of day activities spent in sleep), suggesting that they also do so when on the wing. As in aquatic mammals, unilateral eye closure and unihemispheric slow wave sleep or USWS also occur in birds (reviewed in Rattenborg et al., 2000, 2009). In USWS only one hemisphere sleeps at a time and there is some evidence that birds in migratory formations sleep this way while flying. SWS in birds does not appear to be homeostatically regulated. SWS in NREM sleep in pigeons does not decline in the course of the dark period, suggesting that SWS in these animals is not building up some chemical that was depleted during waking. Unlike mammals, sleep spindles are absent during NREM in

birds. In addition to conventional SWS, birds also display sleep states that simultaneously combine features of both wakefulness and SWS. Some birds such as ostriches and budgerigars spend more than 25 percent of their sleeping time in REM, similar to the amount of REM found in humans

Monotremes

Composed of three extant species (two species of echnida and the duck-billed platypus), these mammals are thought to have diverged from the main mammalian line prior to the divergence of marsupials and placental mammals. While initial studies of the short-beaked echnida (*Tachyglossus aculeatus*) suggested unequivocal SWS but no EEG signs of REM, follow-up work revealed that REM could be characterized by concurrent cortical activation, reduced tonic EMG activity, and rapid eye movements in short-beaked echidnas under low, thermo-neutral, and high-ambient temperatures. Apparently irregular reticular discharge patterns during SWS in the short-beaked echidna constitutes a kind of mixture of REM and NREM. Rapid eye movements were also later recorded in the duck-billed platypus despite no overt EEG signs of REM. Thus, the monotremes appear to exhibit a mixed, indeterminate form of sleep containing elements of both REM and NREM mammalian sleep states. It is possible that mammalian sleep states emerged out of this primordial hybrid state of indeterminate sleep, with SWS and REM segregating into independent brain states dependent on CNS organization of the animal.

1.3.4 Aquatic Mammals

Sleep in marine mammals, such as the bottlenose dolphin, whale, manatee, walrus, and seal, is remarkably different from sleep in terrestrial animals (reviewed in Lyamin et al., 2008). Like many avian species and unlike terrestrial mammals, marine mammals tend to exhibit unihemispheric sleep wherein one hemisphere of the brain sleeps at a time. That sleeping hemisphere engages in NREM but not REM sleep. As in birds, unihemispheric sleep in aquatic mammals is associated with keeping one eye open during sleep – typically the eye contralateral to the hemisphere that is asleep. When in the water, fur seals always use unihemispheric sleep, but when on land, they, like other terrestrial mammals, show bilateral hemispheric sleep. Although EEG signs of REM are absent, cetaceans show other behavioral signs of REM including rapid eye movements, penile erections, and muscle twitching. The two main families of Pinnipeds, Otariidae (sea lions and fur seals) and Phocidae (true seals), show both unihemispheric and bihemispheric forms of sleep. Phocids sleep under water (obviously holding their breath) while both

hemispheres exhibit either REM or SWS. Amazonian manatees (*Trichechus inunguis*) also sleep while under water, exhibiting three sleep states: bihemispheric REM, bihemispheric SWS (BSWS), and unihemispheric SWS. Both hemispheres awaken to surface and breathe. Whales (*Delphinapterus leucas*) and dolphins (*Tursiops runcates*) show only USWS. Northern fur seals and sea lions (family Otariidae) are aquatic and terrestrial. While in water these animals have USWS, like cetaceans, but on land they have both USWS and BSWS. It is unclear whether cetaceans have REM sleep, whereas Otariidae have REM sleep on land, and it is always bilateral.

The fact that the Otariidae exhibit the typical mammalian sleep pattern when on land but unihemispheric sleep when in water strongly suggests that sleep architecture or the patterning of sleep functioning in general across species is at least partially plastic or facultative – that is, sleep can accomplish its vital functions apparently via the expression of two states (bilateral REM and NREM); one state (unihemipsheric SWS) or some hybrid combination of both REM and NREM. Slow wave sleep appears to be the more essential state rather than REM. No animal studied to date functions without some form of SWS. REM on the other hand may be entirely absent in some species. Given also that when the Otariidae return to land they show little or no "rebound" of REM sleep, the evidence is mounting that some aspect of REM appears to be nonobligatory for normal functioning of an animal. The sleep rebound phenomenon is interesting from another point of view as well. Rebound effects may be restorative for only selected sections of the brain. What happens if we attempt to prevent USWS? That hemisphere and that hemisphere alone incurs a sleep debt and will evidence rebound (increased amount and intensity of USWS) once the hemisphere is no longer prevented from entering USWS. This fact, that sleep rebound occurs in only one hemisphere in these species, implies that the homeostatic need for sleep accumulates independently in each hemisphere. If homeostatic need is hemisphere-specific then the thing that gets depleted with wake has to be in that hemisphere.

1.3.5 Terrestrial Mammals

Moving from the oceans onto the land, now we come to the sleep of terrestrial animals. The sleep of terrestrial mammals varies tremendously. Average total daily sleep duration ranges between three hours in the donkey (*Equus asinus*) to twenty hours in armadillos (*Chaetophractus villosus*; Affani, Cervino, & Marcos, 2001), while average sleep cycles vary from six minutes in the chinchilla (*Chinchilla lanigera*) to ninety minutes in humans and chimpanzees (*Pan troglodytes*). Comparative studies of sleep quotas/values in terrestrial

mammals suggest that NREM and REM sleep quotas increase in tandem with one another. That is, whenever there is an evolutionary increase in NREM duration, REM too will increase its duration (Capellini, Barton, et al., 2008). Both REM and NREM sleep durations are lower when animals sleep in more exposed and vulnerable sites and have a more herbivorous diet, suggesting that total sleep time is constrained in species that experience higher predation risk.

1.3.6 Primates

Sleep in primates is reviewed in Nunn et al. (2010). While living primates are divided into two groups, the Strepsirhini (lemurs and lorises) and the Haplorhini (monkeys, apes, and the tarsier), we are primarily interested in the Haplorhines – the line that gave rise to humans. Haplorhines include two groups, the Platyrrhini and the Catarrhini. Platyrrhines are monkeys that are native to the New World. Catarrhines include both Old World monkeys and the apes.

Nonhuman primates exhibit two major sleep phases: REM and NREM. In some apes NREM exhibits two subphases as well: a phase of light sleep and deep sleep characterized by SWA. Owl monkeys, cotton top tamarins (*Saguinus oedipus*), and mouse lemurs (*Microcebus murinus*) exhibit average total sleep time per day from thirteen to seventeen hours. The short sleepers (averaging between eight and eleven hours total sleep) include humans, the chimpanzee, a handful of cercopithecine monkeys, a lemur, and some New World primates. Time devoted to REM sleep among primates varies from a little more than thirty minutes per day in the vervet monkey (*Cercopithecus aethiops*) to two hours per day in the chimpanzee and human. Relative to other primates, humans have exceptionally shorter sleep times but a significantly higher proportion of REM (Samson & Nunn, 2015).

In general, primate sleep is characterized by (1) consolidation of sleep into a single long bout, or two relatively long bouts, possibly to achieve greater sleep intensities; (2) reductions in total sleep times among diurnal primate species including humans, which could reflect a number of different advantages or constraints associated with diurnality (or being active in daylight); (3) increased sleep intensity, possibly associated with the differentiation of NREM sleep stages into lighter and deeper stages of sleep; and (4) maintenance of social contact during sleep, which likely has advantages in terms of infant care, predation risk, and thermoregulation.

1.3.7 Ancestral Human Sleep

Cumulative cultural evolution took off in the upper paleolithic in the Eurasian ecologic context. Anatomically modern humans (AMH) dispersing out of

Africa between 75,000 and 50,000 years ago and meeting other human populations such as the Neanderthals had to adapt to these new populations, new diseases, and colder climates. Recent analyses of Neanderthal DNA (Danemann & Kelso, 2018) have demonstrated that migrating AMH peoples mated with some Neanderthals. Among the Neanderthal genes preserved in AMH populations are genes (e.g., ASB1, EXOC6) that enhanced REM sleep processes in several ways and resulted in a higher risk for narcolepsy – a disorder wherein REM neurobiology is dramatically disinhibited, resulting in REM processes seeping into daily waking consciousness. REM neurobiology therefore became available in a new way for AMH peoples of the upper paleolithic

Nunn and Samson (2018) analyzed a large data set composed of a host of sleep, ecological, physiologic, and life history characteristics of some thirty differing primate species including humans. Using phylogenetic methods including two different Bayesian methods (phylogenetic prediction based on phylogenetic generalized least squares and a multistate Onrstein-Uhlenbeck (OU) evolutionary model of random drift and stabilizing selection), the authors were able to identify what kind of sleep characteristics a primate like us should evidence given the physiologic, ecologic, and life history characteristics we possess. It turns out that humans sleep less than predicted for a primate of our body mass, predation risk, brain size, foraging needs, sexual selection, and diet. Humans were predicted to spend 13.8 percent of their TST in REM. The observed value was 22.3 percent. Humans pack an unexpectedly higher proportion of REM sleep into a shorter overall sleep duration compared to other primates. We, in short, have invested in REM sleep to an unusual degree, relative to other primates.

There is an ongoing debate among some scholars about the normal human sleep pattern, with some claiming that humans sleep for a few hours during the night and then take a long nap in the late afternoon. This is called the "bimodal sleep pattern." Other scientists claim that that bimodal pattern occurs during the dark period, which is split up into two bouts of sleep with a period of wakefulness during the night. Yet, other scholars claim that humans sleep in one long bout during the dark period; that is, that there is no bimodal sleep pattern at all. Historians and anthropologists have presented extensive evidence that a bimodal pattern was common in preindustrial societies. Ekirch (2005) notes that traditional peoples often refer to "first" and "second" sleep. He provides the example of the Asante and Fante on the West African coast, for whom the phrase "*woadá ayi d. Fā*" in their native Tshi language signifies that "they lie in the first sleep," whereas "*wayi (or wada) d. Biakō*" reads as "he has slept the first part of the night." The bimodal pattern allows traditional peoples to engage in numerous social interactions during the dark period (i.e.,

from tending to children to forming social alliances and keeping watch against nighttime predators) (Yetish et al., 2015).

1.3.8 Summary

Sleep in the form of regularly occurring periods of quiescence and some amount of sleep rebound can be found in even the most simplest of organisms, from earthworms and fruit flies to nonhuman primates and human beings. Birds and aquatic mammals also evidence distinct sleep states including the phenomenon of unihemispheric sleep, which allows these animals to sleep while flying or swimming. REM may only occur bihemispherically. The presence of high-voltage slow waves as well as REM–like brain activation patterns in reptiles, birds, and mammals suggests that the biphasic, REM, and NREM sleep phases we find in humans is a very ancient adaptation indeed, and that its benefits outweigh the risks associated with quiescence and reduced responsiveness to the environment. Thus, it is looking more and more like two major sleep processes that in humans are expressed as REM and NREM have a very ancient lineage going all the way back to the fruit fly and worm. Many environmental factors, including food availability, social interactions, and temperature, potently impact sleep in diverse phyla (McNamara et al., 2009; Capellini, 2010). For example, sleep is reduced in flies reared in isolation, and exposure of male flies to females suppresses sleep, revealing robust modulation of sleep by social stimuli (Machado et al., 2017). The fact that sleep is inherently social in complex organisms is underlined by the fact that most mammalian animals sleep socially. Nevertheless, sleep function remains a mystery, particularly active phases of sleep and REM sleep in humans.

Nothing is more practical in understanding sleep than a theoretical model. For the purposes of this book we adopt standard Darwinian evolutionary theory to understand REM and NREM sleep. To help use evolutionary theory to understand human sleep we will adopt mid-level theories such as life history theory, parent-offspring conflict theory, sexual conflict theory, and most importantly the social-ecological theory (Figure 1.2**)**. The social-ecological framework assumes that sleep in human beings is inherently suffused with social functions and factors. It is shaped by the surrounding ecology that again is mostly social forces. The social-ecological theory also allows us to build models of the factors that might cause poor sleep or sleep dysfunction, so it will be the key background framework we will adopt in this book.

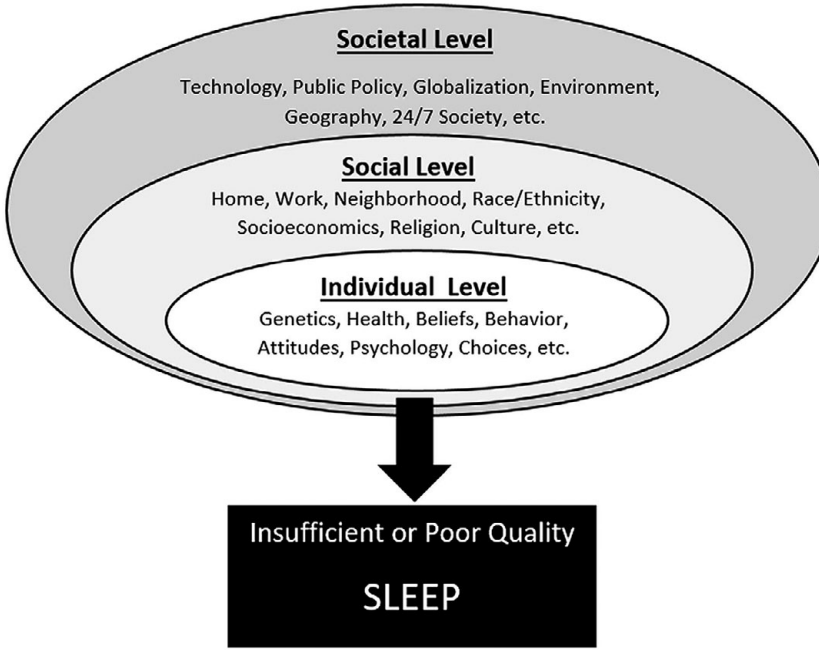

Figure 1.2 Social ecological model of sleep
Used with permission from Grandner, M. A. (2017)

1.4 Review Questions

- Delta brain waves indicate the intensity with which we sleep. Why do you think that these waves are strongest over the frontal lobes during sleep?
- Slow wave sleep can occur in only one brain hemisphere at a time. REM sleep, on the other hand, as far as we know, never occurs unihemishperically. Why do you think REM sleep requires both hemispheres to manifest?
- Why do you suppose some birds and some aquatic mammals, such as dolphins, sleep with only one brain hemisphere awake and the other asleep?
- What strengths and weaknesses do you see with the scientific claim that sleep is social?

Further Reading

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PART I

Sleep

CHAPTER TWO

From Biological Rhythms to the Sleep Cycle

Learning Objectives

- Describe how the sleep cycle fits into the larger twenty-four-hour circadian cycle
- Identify the regulatory functions of the master biological clock: the suprachiasmatic nucleus (SCN)
- Identify the causes and consequences of major disorders of biological rhythmicity
- Identify the major electrophysiologically defined sleep stage components of the sleep cycle

2.1 Introduction

All terrestrial animals live their lives embedded in the twenty-four-hour light-dark cycle. How does the sleep cycle fit into the larger twenty-four-hour or circadian cycle? The current belief is that a brain-based circadian pacemaker or master clock is synched-up with, or entrained to the twenty-four-hour light-dark cycle such that it sends chemical messages to the rest of the brain that signal changes in the daily light–dark cycle. As light turns to dark and dark turns to light the master clock sends the appropriate chemical messenger into the appropriate brain regions that turn sleep on and off. A homeostatic process linked to the pacemaker region regulates (with the help of pacemaker genes) or influences the amount and timing of sleep, possibly via accumulation of adenosine or some other neuroendocrine or neurochemical substance that signals sleep need and sleep debt. Adenosine accumulates as the individual goes about his waking day and with it the urge to sleep increases until sleep occurs and adenosine levels reset. The circadian pacemaker regulates the release of adenosine and related chemical messengers via its control of the neuroendocrine hypothalamic region that contains the master clock.

The master clock is believed to be housed in the hypothalamus and is usually identified with the suprachiasmatic nucleus (SCN). For example, the SCN of the hypothalamus receives information from the visual system that tells it whether the individual is entering a light or dark phase, and then based

on that information the SCN will release melatonin (that is synthesized in the pineal gland), which will then trigger termination or initiation of sleep. We will discuss this process in more detail in the next section, as its dysfunction results in several types of sleep and circadian rhythm disorders.

2.2 Circadian Rhythms and Sleep

2.2.1 Introduction

The brain region that appears to act as the master circadian clock in humans is the SCN. The SCN is located above the optic chiasm and at the bottom of the hypothalamus. It receives direct input from the retina via the retinohypothlamic tract. The retinohypothalamic tract transmits light information from the optic nerve in the eye to the geniculate nucleus of the thalamus, and thence to the visual cortex and the SCN. The core SCN cells receive light information from ganglion cells in the retina, which contain the unique photosensitive pigment called melanopsin. When light information arrives at the SCN cells within the core of the SCN they entrain their firing patterns with the light and dark cycle. Another group of cells surround the core cells of the SCN. These are called the shell cells and they are involved in regulation of melatonin release.

Melatonin is a hormone produced in the pineal gland and is linked to the onset of seasonal estrus in seasonally reproducing animals. It is released primarily during the night in both diurnal and nocturnal animals and thus it can be used by seasonal breeders as a measure of day length. As day length increases in the spring, the longer light period inhibits melatonin production, leading to shorter durations for nighttime peak values. These changes in nocturnal melatonin levels then disinhibit release of gonadotropins, bringing the animal into reproductive mode.

In humans, melatonin release begins to increase at around 10 P.M., with central nervous system (CNS) levels peaking around 3 A.M. (when REM predominates) and then declining to very low levels by 8 A.M. Daytime levels are almost undetectable. Circulating levels of melatonin decrease dramatically in prepubertal children and may contribute to disinhibition of gonadotropins and development of nocturnal entrainment of release of these gonadotropins. When melatonin is administered to humans its primary effect is to increase sleepiness.

Melatonin is synthesized and released from the pineal gland, which sits between the cerebral hemispheres and is connected to the brain by a small stalk. However, the neural connection to the pineal does not go through that stalk. Cells from the shell of the SCN project to a nearby region in the hypothalamus called the paraventricular nucleus. Then connections from this nucleus to the

Table 2.1 Disorders of biological rhythms

Disorder	Symptoms	Pathophysiology and Treatment
Shift work and jet lag	Fatigue, sleepiness, confusion, listless, low energy, GI ailments	Individual's biorhythms are out of phase or sync with social group's rhythms and activities (e.g., due to shift work or skipping time zones due to air travel). Treatment: It takes a week to adjust to local time schedules; melatonin administration can help reset internal clock.
Seasonal affective disorder or SAD	Mood dysfunction and depression during dark months	Reduced day length or duration and intensity of sunlight during fall-winter alters SCN output and disrupts circadian organization. Treatment involves phototherapy (exposure to varying light intensities to reset SCN) and antidepressants medications.
Bipolar 1 disorder	Cycling between periods of extreme manic episodes lasting about one week and depressive episodes also lasting about one week. During manic phases there are feelings of euphoria and elation. There is disinhibition, agitation, sleeplessness, and occasionally delusional beliefs. During the depressive phase there is major depression (sadness, despair, anhedonia, etc.).	Causes are unknown but clock genes expressed in the SCN have been implicated. Treatment: antipsychotics, mood stabilizers, antidepressants
Delayed sleep phase wake disorder	The individual's sleep-wake times are two or more hours ahead of the social reference point.	Cause is unknown but one possibility is that the delayed sleepiness represents an exaggerated reaction to the normal shift in the internal clocks that is seen in adolescents after puberty. Treatment: phototherapy with bright light exposure in the morning, melatonin (taken six hours before desired sleep time shifts clock backward).
Advanced sleep phase wake disorder	The individual goes to sleep earlier and awakens earlier than the social reference point.	Treatment: bright light exposure in the evening and melatonin in the morning

SCN help to regulate melatonin release. Once melatonin is released from the pineal it comes under the control of SCN, thereby helping the clock to entrain physiologic processes with the twenty-four-hour light–dark cycle.

2.2.2 The REM–NREM Cycle

Sleepiness is triggered by the SCN-related melatonin activity. Once an individual mammal is asleep another cyclic process is activated within the overall twenty-four-hour circadian cycle called the ultradian cycle. This ultradian cycle involves the alternation between REM and NREM sleep states. In other words, about every ninety minutes one undergoes a period of REM and a period of NREM sleep. In the adult human the ultradian cycle involves sleep onset through NREM sleep stages and sleep offset through REM sleep stages, with NREM sleep states predominating in the first third of the night and REM sleep periods predominating in the last third of the night. Most of us awaken from a REM rather than an NREM episode. This ninety-minute ultraradian sleep cycle takes years to develop in the human.

Now let us look a little more closely at how this sleep cycle works during a single night of sleep in an average adult man or woman. We enter sleep through NREM or phases N1, N2, and N3 or slow wave sleep (SWS). After about an hour of NREM sleep, we leave deep SWS as sleep lightens and we briefly pass back up through stages 2 and 1; EEG amplitude declines and the wave form gets faster and faster, almost resembling waking activity. The slow, rolling eye movements, characteristic of N3, begin to show periodic spikes with the eyes darting back and forth under the closed eyelids. Muscle tone is lost and we are unable to move. Heart rate increases and the sexual system becomes activated with penile erection in males and clitoral engorgement in females. All of these dramatic changes signal the onset of the first episode of REM sleep of the night. Once we enter this first REM episode we will have undergone one full NREM to REM cycle. While this first REM period will last about only ten to twenty minutes, by the time morning comes these REM episodes will last about thirty to forty minutes. After the first full NREM to REM cycle we begin to cycle back down again into slow wave sleep. We "descend" from REM through brief phases of N1 and N2 into N3, with slow wave activity on EEG, increased muscle tone, slow rolling eye movements, no sexual activation, and regular breathing and heart patterns. After about an hour, the EEG again returns rapidly through stages 2 and 1 to another episode of REM sleep, and so on throughout the night.

When we use a graphic representation, called the hypnogram, to portray the changes in EEG and arousal states over the NREM–REM cycle, we can see the changes in the distribution of REM and NREM sleep times across a single

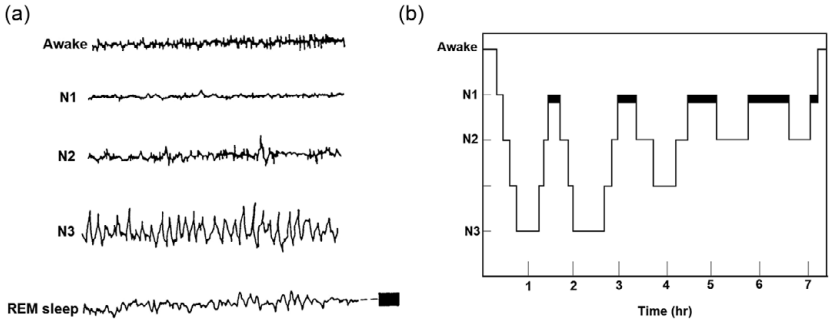

Figure 2.1 Human sleep architecture (hypnogram)
(public domain)

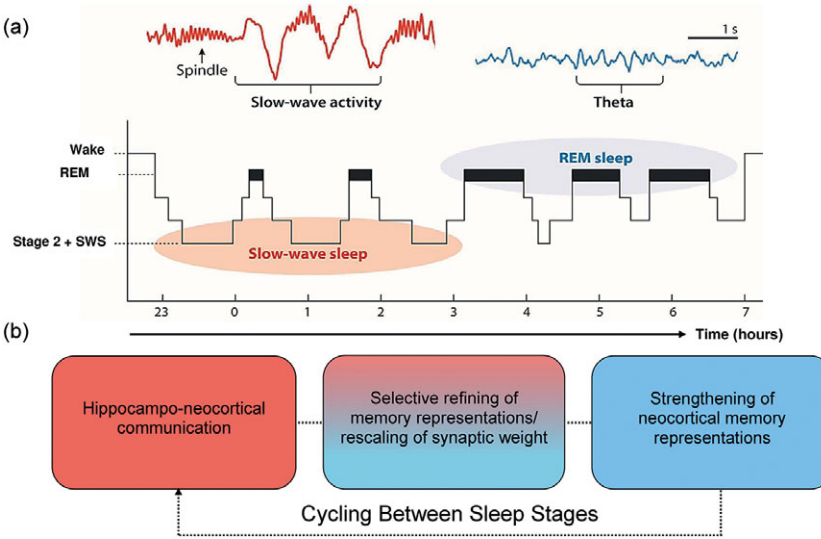

Figure 2.2 Schematic of sleep architecture in humans and associated oscillatory activity and stages of memory consolidation
Used with permission from Cross et al. (2018)

night of sleep. Time is on the horizontal axis, and sleep state is on the vertical axis. We call the resulting picture of sleep over a single night the sleep architecture. In Figure 2.1 the new nomenclature for sleep states will assimilate stage 3 and 4 into one stage, N3.

In Figure 2.2 we see how sleep cycles are likely related to facilitation of cognitive functions such as memory. SWS is most prominent during the first

half of the sleep period, and is dominated by slow wave activity throughout the neocortex but originating with the thalamus. Thalamic spindles appear most often in stage N2. By contrast, REM sleep is most prominent during the second half of the sleep period and is characterized by ponto-geniculo-occipital waves, increased acetylcholine (Ach), and theta oscillations originating in the medial septal region and hippocampus. The cyclic occurrence of SWS and REM differentially facilitate memory consolidation. The hierarchical nesting of sharp-wave ripples and spindles during SWS facilitate the transfer of information from the hippocampal complex to the neocortex. These neocortically distributed memory representations are strengthened by REM theta oscillations and increases in Ach.

2.2.3 What Triggers the Ultradian Sleep Cycle?

In the mid-1990s, Saper and colleagues (reviewed in Saper et al. 2005) argued that there was a sleep switch in the anterior hypothalamus. Neurons within the anterior hypothalamus increase their activity just before the transition into sleep. Gamma-aminobutyric acid, or GABA neurons, project to all the brain stem wake–promoting neurons and turn them off. Interestingly, the GABA sleep–promoting neurons in the anterior hypothalamus and related neurons within the hypothalamus such as histaminergic neurons, hypocretin neurons, and the sleep-on neurons, receive inputs directly or indirectly from the nearby suprachiasmatic nucleus (SCN), which as we noted previously, transduces light signals from the retina into hormonal and neuronal messages concerning the dark-light circadian cycle. In short, what triggers the NREM–REM sleep cycle is messages coming from the retina to the SCN in the hypothalamus that signal that the dark period has commenced. Then messages from the SCN to the anterior thalamus activate gabaergic sleep-on neurons in the anterior hypothalamus, which then send inhibitory signals to neurons in the brain stem that maintain wakefulness, basically instructing them to turn off, and thus sleep onset commences. When sleep has finished and the light phase of the daily cycle commences, this information is sent from the SCN down to hypocretin neurons in the hypothalamus and these neurons then send excitatory signals down to the wake-promoting nuclei in the brainstem, which then send inhibitory signals back to the hypothalamic sleep-on cells at the same time they are activating the cortex.

With the advent of new technologies, such as optogenetics and CRISPR-Cas, there have been major advances in the understanding of this neuronal circuitry (Shiromani & Peever, 2017). The processes of wake and sleep promotion are now known to be mutually inhibitory. When wake arrives it inhibits sleep and vice versa. The two states do not mix. If they do mix, the

result is pathology or mental dysfunction. This mutual inhibition dynamic very likely extends to REM and NREM within sleep. When REM arrives it inhibits NREM and vice versa. Hybrid REM and NREM states tend to cause mental dysfunction. This mutual inhibition process operates as follows: During waking the need for sleep gradually increases (a homeostatic process) as a function of the accumulation of a neurochemical in the brain (likely the cholinergic-rich basal forebrain) of the awake person. When the neurochemical accumulation process (Process S) reaches a critical point, sleep promotion outweighs wake promotion and a rapid switch occurs (cell groups A stop firing and cell groups B start firing in the hypothalamus), thereby triggering sleep. But there is flexibility around that window of time when sleep promotion outweighs wake promotion. Into this window of flexibility, around the timing of the switch into sleep or wake, enters a host of factors such as social interactions, special needs, metabolic issues, and the circadian regulatory process itself. Call this circadian process, Process C. Any of these factors can delay sleep onset for a while – but not indefinitely. Eventually the need for sleep, which is promoted by the accumulation of a neurochemical substance such as adenosine, becomes so overwhelming that it cannot be delayed any longer and the switch occurs. But Process C interacts with Process S in such a way as to regulate sleep timing and intensity.

2.2.4 The Interaction of Circadian Rhythms with Sleep Rhythms: The Two-Process Sleep Regulation Model

The most dramatic effect of sleep deprivation in every mammalian species studied thus far has been the phenomenon of "compensatory rebound," or the increase over baseline of sleep times and intensity where intensity is measured by higher arousal thresholds, enhanced slow wave activity, enhanced rapid eye movement frequencies per unit time, and "deeper" and longer sleep cycles (Borbeley, 1980; Borbeley et al., 1984). After sleep deprivation, mammalian animals attempt to make up for lost sleep by enhancing the intensity and duration of subsequent sleep. During the wakefulness or deprivation period a neurochemical, possibly adenosine (Blanco-Centurion et al., 2006; Strecter et al., 2006), accumulates in proportion to the length of the wake period. As its levels increase in a sleep center (possibly the basal forebrain) they exert an inhibitory effect on neurons in the brainstem and forebrain) that promotes arousal, thus increasing sleepiness. During sleep, adenosine stops accumulating and existing stores begin to dissipate at a rate dependent on sleep intensity until they return to baseline. When the individual awakens he will once again need to cumulate adenosine throughout the day in order to successfully initiate sleep.

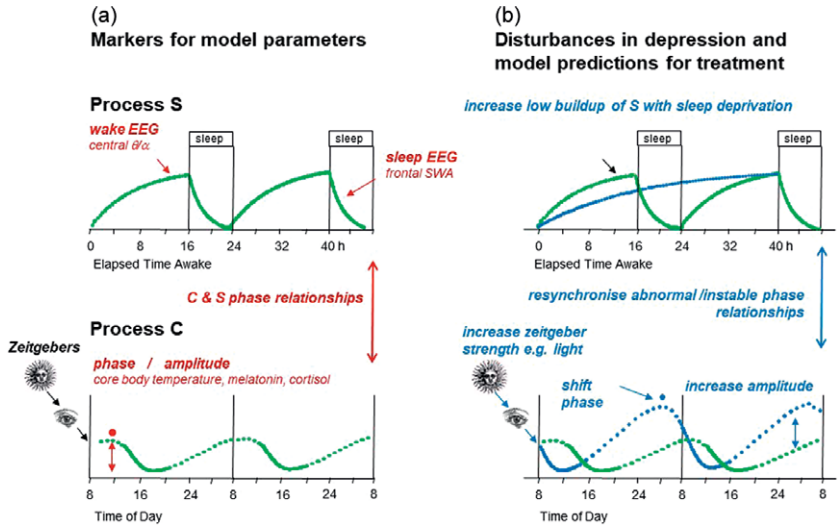

Figure 2.3 Schematic of the two-process model and its application to depression. Normal situation with markers for model parameters (a) and putative pathological changes and their remedies (b).

Used with permission from Borbely et al. (2016)

Borbely (1982) first formalized the insight that mammalian sleep involved a balance between sleep amount and sleep intensity, and that sleep was therefore under homeostatic control. In his "two-process" model of sleep regulation (Figure 2.3) a sleep need factor called Process S (presumably associated with adenosine levels) increases during waking (or sleep deprivation) and decreases during sleep. This part of the model indexes restorative aspects of sleep and explicitly predicts that sleep is required for some restorative process of the brain or the body or both. Process S is proposed to interact with input from the light-regulated circadian system (Process C) that is independent of sleep and wakefulness rhythms. SWA is taken as an indicator of the time course of Process S because SWA is known to correlate with arousal thresholds and markedly increase during the previous waking period and during the rebound period after sleep deprivation in all mammals studied. Once a threshold value of Process S is reached (i.e., once the appropriate amount and intensity of SWS is reached), Process C will be activated. Simulations using the model's assumptions show that the homeostatic component of sleep falls in a sigmoidal manner during waking and rises in a saturating exponential manner during sleep. Process S at sleep onset is responsible for the increase in sleep tendency, and is, therefore, heavily influenced by prior wakefulness. Process C is

dictated by the recurrent variations in sleep tendency (SP) (i.e., the ability to fall asleep) and thus presumably is controlled by a circadian pacemaker. Because wake and SWS delta activity are mutually inhibitory when Process S (SWS delta activity) is high, sleep need is high and Process C is low; when it declines/dissipates during sleep, Process C slowly rises (see left-hand panel of Figure 2.3). This is a normal case. Process C is suppose to rise at the end of the sleep cycle, but if it rises earlier, it will wake the individual prematurely. This then sleep deprives the individual, and mood dysfunction or even depression will result. You can treat this kind of mood dysfunction by resynchronizing Process S and C (see right-hand panel of Figure 2.3).

As just noted, this simple two-process model has allowed investigators to model real-life sleep disturbances by simply modulating one of the basic components or parameters of the model. For example, insomnia or sleep deprivation can be modeled by diminishing or deleting the S process when it interacts with C. Thus, if process S does not result in enough adenosine to effectively inhibit arousal centers, then the individual will not sleep or sleep well. The two-process model was subsequently developed to show that the reaction to sleep deprivation in both long and short sleepers could be predicted by using the same S time constants (Aeschbach et al., 2017). More S is found in short sleepers than long sleepers. It has been found that, when compared to the S of long sleepers, higher levels occur in short sleepers. To account for cognitive effects of sleep deprivation investigators have used nonlinear bi-directional interactions between the circadian C and the homeostatic S processes instead of the unidirectional effect of C on S processes, as captured by the original model. Attempts have also been made to incorporate the ultraradian NREM-REM cycle within the overarching two–process C versus S model.

Although the elegant simplicity of the two-process model captures normal functioning of the sleep-wake cycle, multi-oscillator models have been developed to capture more complex interactions of sleep cycles with other behavioral variables. In a multi-oscillator model, two or more mutually interacting oscillators create the cycles of each of the behavioral variables being modeled. For example, one oscillator might be the suprachiasmatic nucleus (SCN) – the master clock. The SCN, in turn, drives temperature rhythms and melatonin rhythms. A second oscillator might be the controller for sleep-wake rhythms, perhaps centered on the ventrolateral preoptic nucleus (VLPO). This drives REM–NREM cycles. Discovery of the so-called flip-flop switch in the VLPO, mentioned previously, has prompted development of flip-flop models of sleep-wake state regulation. A flip-flop switch refers to the mechanism wherein whenever the switch is in one position (sleep) the other position (wake) is prevented and vice versa. In one recent flip-flop model it is assumed that sleep-active neurons within the VLPO (S-R) oppose wake active neurons

within the hypothalamus and brainstem that fire during wakefulness and reduce firing during REM (W-A neurons); a third group of neurons that fire during REM within the extended VLPO region and a final fourth group of neurons within the basal forebrain that fire during both wake and REM (W-R group of neurons) interact with the first two group of neurons. The four interacting groups of neurons promote a flip or switch of brain state when sleep-promoting substances (e.g., adenosine) accumulate during wake, thereby activating S-R neurons and then slowly dissipating during sleep. A second kind of sleep-promoting substance (possibly a hormone-like somatostatin or growth hormone, etc.) accumulates during wake and during NREM. It then would activate W-R neurons of the extended VLPO and dissipate during REM.

2.2.5 Social Modulation of Biological Rhythms

We have just seen how the two-process model of the interaction of circadian rhythms with sleep cycles can account for mood dysfunction when those circadian cycles are out of phase with sleep cycles. In short, unless circadian rhythms are entrained with sleep rhythms, physiologic and mental dysfunction inevitably follows. To entrain our biological cycles with circadian rhythmicity we need timekeepers or cues that the mind/body can use as reliable timekeepers. If, for example, everyday at around 7 A.M. social activity starts to pick up and requires my attention and responses, then that social activity can act as a timekeeper for my physiologic preparedness. We call that timekeeping function a social *zeitgeber* (German for timekeeper). Ehlers, Frank, and Kupfer (1988) and Grandin et al. (2006) proposed that mood and other mental disorders can arise as a consequence of life events disturbing social zeitgebers, which, in turn, derail social and biological rhythms. According to this theory, disruptions in these rhythms influence internal biological rhythms as well as sleep-wake schedules such that, mood dysfunction can develop in vulnerable individuals (see Figure 2.4**)**. Indeed, depressed individuals evidence out-of-phase circadian and other biological rhythms. They experience early morning awakenings and abnormal sleep-wake cycles, temperature, melatonin, and cortisol rhythms. You can sometimes effectively treat mood dysfunction by realigning biological rhythms (Crowe et al., 2020). The social zeitgeber theory of mood dysfunction implies that anything that disrupts social entrainment of biological rhythms has the potential to create mood dysfunction in vulnerable individuals. These are individuals who for a variety of reasons (genetic, medical, historical, etc.) possess physiologies that are easily perturbable. Call these vulnerabilities (such as genetic anomalies in pacemaker genes) “internal triggers” to rhythm disruption. External triggers to rhythm disruption involve social and environmental zeitgebers.

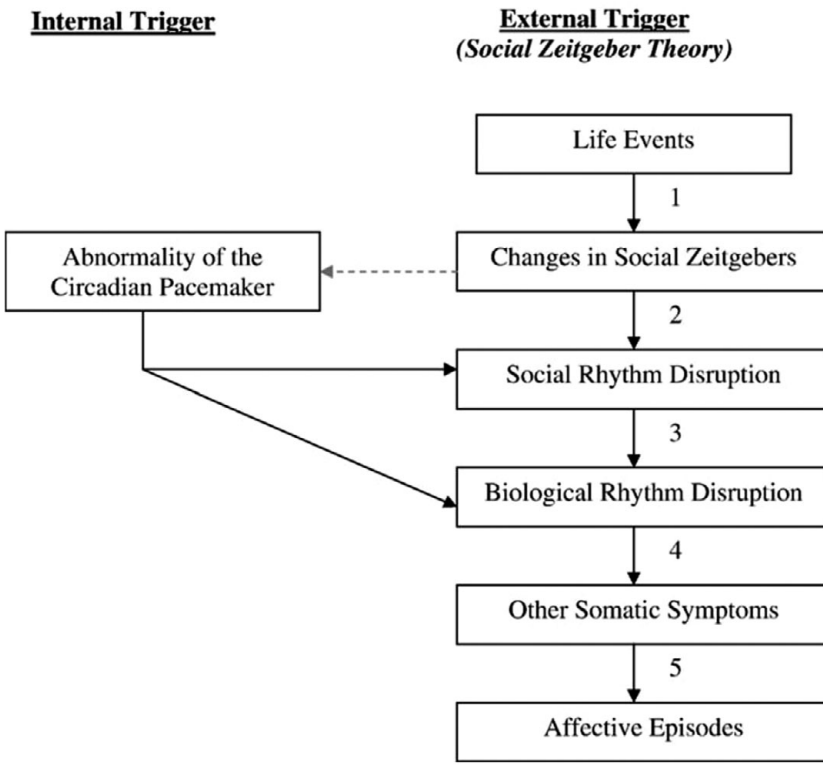

Figure 2.4 Social *zeitgeber* theory of mood dysfunction
Used with permission from Grandin et al. (2006)

The COVID pandemic and governments' lockdown or social isolation strategies created huge disruptions in normal social zeitgeber effects. If social zeitgebers do indeed act to entrain our biological rhythms, and this entrainment protects against disorders of biological rhythms, then we should expect a dramatic uptick in these disorders, including mood dysfunction disorders. Kawage et al. (2022) surveyed a total of N = 997 participants who were subjected to lockdowns, looking for subtle effects, rather than outright disorders of biological rhythmicity. In-depth qualitative interviews were conducted with all of these individuals. An overall and dramatic effect that the authors called "psychic drift" was observed. People simply lost track of time, became unfocused, daydreamed quite a bit, and generally felt sluggish, lost, as if they were going nowhere, and mildly dysphoric. Four ancillary themes were identified: (1) loss of daily timed activities; (2) role of social interaction; (3)

altered time perception; and (4) disruption to motivation and associated psychological effects. Participants described a sense of feeling lost in both time and place, that they were living the same day over and over again, with days "blurring into one another." In short, disruption of social zeitgebers and circadian rhythms brought on by the social isolation associated with COVD lockdowns has very definite deleterious psychic effects. Indeed, the COVID lockdowns appear to have promoted a general drift in people's sleep-wake schedules with delay in sleep onset and offset times, leading to a shift toward eveningness chronotype, even in previously morningness chronotypes, as well as an increase in overall time spent asleep. Despite greater sleep amounts, sleep quality drastically declined for most people (Kantermann, 2020; Marelli et al., 2020).

Both the ultradian and the circadian rhythms are clearly exquisitely sensitive to social cues in human beings. The amount and quality of social interactions exert a profound influence on the body's rhythms. Social rhythms and light cues are known to act as powerful zeitgebers, or timekeepers or signals, which entrain bodily rhythms to regularly occurring social events. These social zeitgebers do so by preparing individuals to take advantage of regularly occurring opportunities (for social meals, formation of social alliances, social negotiations, reproductive opportunities, etc.) or threats in the environment. It may be that "morningness" and "eveningness" (are you a morning person or a night person?) developed in response to the extent to which individuals are sensitive to differing social cues or zeitgebers. Morning people usually prefer to rise between 5 A.M. and 7 A.M. and retire between 9 P.M. and 11 P.M. Evening people tend to prefer both a later wake up (9 A.M. to 11 A.M.) and a later bedtime (11 P.M. to 3 A.M.). Circadian and sleep cycles tend to be less variable in morning people.

We call the preference for morning or evening activities a chronotype. Most people congregate in the middle of the continuum between preference for morning versus evening, but there are fascinating differences between people who lie at the extremes. For example, the morningness chronotypes tend to be more emotionally balanced, more satisfied with life, and less vulnerable to psychiatric disorders. Conversely, eveningness is associated with more variability in sleep-wake schedules, frequent mood swings, mood disorders, and vulnerability to psychiatric illness.

Another regularly occurring sleep-related phenomenon called the "post-lunch dip" or the daytime "siesta" period is also exquisitely sensitive to social cues. The siesta period is marked by increased sleepiness and sluggishness, and decreased alertness that strikes most people between 1 P.M. and 4 P.M. in the afternoon. In offices and factories all over the world people start to fall asleep at their desks or they begin to make small errors when their attention

lapses and they become sleepy. During the siesta period body temperature declines significantly as if the body were preparing for sleep. This siesta period is consistent with the idea that the ancestral human sleep pattern is bimodal, with a long bout of sleep at night and a short bout in the late afternoon, which in the modern era has become a siesta period instead of a napping period. The fact that what was once a daytime sleep period has become a mere siesta period in most cultures (and no rest at all in many cultures) demonstrates the extent to which sleep is influenced by social norms, and that at least some amount of sleep is non-obligate.

Box 2.1 Morningness–Eveningness questionnaire

1. What time would you get up if you were entirely free to plan your day?
2. What time would you go to bed if you were entirely free to plan your evening?
3. If there is a specific time at which you have to get up in the morning, to what extent do you depend on being woken up by an alarm clock?
4. How easy do you find it to get up in the morning (when you are not woken up unexpectedly)?
5. How alert do you feel during the first half hour after you wake up in the morning?
6. How hungry do you feel during the first half hour after you wake up in the morning?
7. During the first half hour after you wake up in the morning, how tired do you feel?
8. If you have no commitments the next day, what time would you go to bed compared to your usual bedtime?
9. You have decided to engage in some physical exercise. A friend suggests that you do this for one hour, twice a week, and the best time for him is between 7:00 and 8:00 A.M. Bearing in mind nothing but your own internal "clock," how do you think you would perform?
10. At what time of day do you feel you become tired as a result of need for sleep?
11. You want to be at your peak performance for a test that you know is going to be mentally exhausting and will last for two hours. You are entirely free to plan your day. Considering only your own internal "clock," which ONE of the four testing times would you choose?
12. If you got into bed at 11:00 P.M., how tired would you be?
13. For some reason you have gone to bed several hours later than usual, but there is no need to get up at any particular time the next morning. Which ONE of the following are you most likely to do?

Box 2.1 (cont.)

14 One night you have to remain awake between 4:00 and 6:00 A.M. in order to carry out a night watch. You have no commitments the next day. Which ONE of the alternatives will suite you best?
15 You have to do two hours of hard physical work. You are entirely free to plan your day and considering only your own internal "clock," which ONE of the following times would you choose?
16 You have decided to engage in hard physical exercise. A friend suggests that you do this for one hour, twice a week, and the best time for him is between 10:00 and 11:00 P.M. Bearing in mind nothing else but your own internal "clock," how well do you think you would perform?
17 Suppose that you can choose your own work hours. Assume that you worked a FIVE-hour day (including breaks) and that your job was interesting and paid by results. Which FIVE CONSECUTIVE HOURS would you select?
18 At what time of the day do you think that you reach your "feeling best" peak?
19 One hears about "morning" and "evening" types of people. Which ONE of these types do you consider yourself to be?

2.2.6 Rhythm Disorders

About a quarter of people who complain of difficulty sleeping or nonrestorative sleep very likely actually have a disorder of biological rhythms; that is, their twenty-four-hour sleep-wake schedules have been desynchronized with their social zeitgebers or social obligations. In the so-called delayed sleep phase disorder (DSPD), sleep-wake schedules are advanced at least two hours later than habitual or conventional sleep times. So if most people in the local culture go to sleep by midnight, sleep onset times at around 2 A.M. would be considered possible DSPD. The individual with DSPD has tried to go to sleep earlier but typically cannot, and even when he does manage to sleep earlier he still wakes up later than is culturally acceptable. Therefore, his sleep schedule conflicts with the demands of the society he lives in. He must awaken earlier than his body wants to wake. He is therefore constantly incurring a sleep debt, so he walks around sleepy and fatigued all the time. DSPD sometimes runs in families; there may be a genetic component to the disorder. Its onset is typically in adolescence.

Advanced sleep phase disorder (ASPD) is the counterpart to DSPD. While the DSPD patient prefers to go to sleep later than others, the ASPD patient prefers to go to sleep substantially earlier than others. ASPD patients become sleepy early in the evening and prefer early morning wakings. Even when they try to sleep in, they can't; they spontaneously wake in the early morning.

To treat the delayed sleep phase patient we can expose them to bright light in the morning and reduce light exposure in the evening in order to reset the master clock (which is normally entrained via daylight) so as to be entrained with conventional sleep-wake schedules. In addition, melatonin can be administered six hours before desired sleep time so as to shift the master clock backward and get it more aligned with conventional rest-activity schedules. An opposite course is followed for ASPD patients: bright-light exposure in the evening to delay sleep and melatonin release.

We all know what jet lag disorder is. When we fly across time zones our normal sleep-wake schedules get disrupted such that our circadian rhythms become out of phase with local social schedules. Your timing for sleep is out of phase with the locals as are your meal times and rest times. Therefore, your entire physiology has to adjust to local time if you are to socialize normally. Seasonal affective disorder (SAD) is a subvariant of major depressive disorder dubbed "depressive mood with seasonal pattern." People with SAD become depressed, sleepy, and inactive during dark winter months and their spirits lift with the return of sunlight and springtime. In bipolar 1 disorder, the individual's mood swings significantly within a day or across several weeks. The patient can cycle between manic episodes of sleeplessness and frenetic activity that can last several days, and suddenly crashing into episodes of dark depression, sleepiness, and inactivity. During a manic episode the patient can go without sleep for days at a time and exhibit a rapid, pressured form of speech. They are flooded with ideas, easily excitable, and sometimes hypersexual. When, however, the depression returns, there is a crash; they tend not to want to talk and there is little if any ideation. They are self-critical and depressed and appear to be somnolent – though quality sleep may elude them. SAD and bipolar 1 disorder can be treated effectively with antidepressants and mood stabilizers. Chronotherapy, or the selected use of bright-light exposure, can effectively reset circadian rhythms and thus treat phase disorders and perhaps even SAD.

2.2.7 Conclusion

The ultradian NREM–REM sleep cycle is embedded within the larger twenty-four-hour circadian cycle. The sleep cycle interacts with the circadian cycle, which in turn is controlled by the SCN master clock within the hypothalamus. The two-process model captures interactions between the circadian process and the sleep cycle. Disorders of biological rhythmicity such as delayed sleep phase syndrome and manic depression have significant but treatable effects on sleep. In the next chapter we will learn that sleep expression also changes in remarkable ways across the lifespan.

2.3 Review Questions

- What is the probable role of adenosine in regulation of the sleep cycle?
- What is the evidence that manic depression/bipolar disorder is a sleep-related disorder of biologic rhythms?
- What are two ways that social processes modulate biological rhythms?
- How does the two-process model describe the interactions of the circadian cycle and the sleep cycle?
- Why do you suppose we enter sleep via NREM stages rather than REM?

Further Reading

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CHAPTER THREE

Expression of Sleep across the Human Lifespan

Learning Objectives

- Identify the relevance of several mid-level evolutionary theories (attachment theory, parent-offspring conflict theory, hetereochrony, etc.) for the expression of sleep characteristics across the lifespan
- Evaluate evidence for the claim that REM sleep is crucial for brain development
- Describe the significance of changes in proportion to REM and NREM sleep states across the lifespan
- Identify the impact of sleep on longevity

3.1 Introduction

How should we study the typical development and expression of sleep patterns in people? The most straightforward way to do so would be to simply observe the development of sleep states in people as those people develop into maturity, reproduce, age, and die. But who, what peoples, should we study in order to get a picture of the typical human pattern?

Unfortunately, the vast majority of studies of sleep development and expression across the lifespan have been done on, and were conducted by researchers of, only a single class of people. Beebe (2016) and Henrich (2020) called them "Western, Educated, Industrialized, Rich, and Democratic (WEIRD)" peoples. Thus, the evidence on what sleep scientists have regarded as normal lifespan sleep patterns is based largely on one small and unrepresentative slice (i.e., most of the world's population is not rich) of the human family: WEIRD samples. That would be OK if sleep was known to be unaffected by social or cultural factors, but that simply is not the case. Sleep IS strongly affected by social and cultural factors, so basing our understanding of sleep development on a single sample that is not typical of the rest of the human family is problematic. For example, in non-WEIRD societies co-sleeping is the norm at all stages of life from infancy to old age, whereas co-sleeping is not universal in WEIRD societies – even for mother-infant pairs. Thus, the reader needs to keep these considerations in mind when perusing the information and

data presented in this chapter concerning norms of sleep expression across the human lifespan (Peña et al., 2016). Kocevska et al. (2020) assembled individual sleep data measured "objectively" via wrist actigraphy or subjectively via self-report (sleep diary) from 200,358 people (aged 1–100 years, 55 percent female) from thirty-six studies from the Netherlands; 471,759 people (40–69 years, 55.5 percent female) from the United Kingdom; and 409,617 people (≥18 years, 55.8 percent female) from the United States. Their basic findings can be seen in Table 3.1. At every age group females sleep slightly more efficiently than males. Sleep efficiency indicates the extent to which sleep is not interrupted by arousal, awakenings, and movements. After plateauing in the teenager years, sleep efficiency declines slightly with age and this decline is roughly equal between the sexes. Sleep duration (number of hours) for all age groups and for males and females measured objectively, runs less than eight hours per night (around seven hours). In adulthood sleep plummets to about six hours per night. Between 9.6 and 19.4 percent of these respondents reported some amount of insomnia. Among teenagers, 51.5 percent reported total sleep times (TST) of less than the Sleep Foundation's recommended eight to ten hours, and 18 percent reported daytime sleepiness.

While the data displayed gives us some glimpse as to the broad trends in sleep schedules for the otherwise healthy WEIRD population, Jonasdottir et al., (2021) analyze 11.14 million nights from 69,650 adults aged nineteen to sixty-seven from forty-seven different countries, thus including a much greater swath of the world's population than people from the WEIRD countries alone. The trends appear to be broadly consistent with those from WEIRD countries. Sleep duration decreases with age and nighttime awakenings increase with age, while sleep onset and offset times become earlier with age so that people retire earlier and wake up earlier as they age. Although men tend to sleep less than women across the lifespan, nighttime awakenings are more prevalent for women, with the greatest disparity found from early to middle adulthood, a life stage associated with child-rearing. If we look at lifespan changes in sleep in terms of sleep architecture, we find that REM sleep is abundant in early life, making up approximately 50 percent of total sleep time in infant and young children. But by the time children reach primary school age, REM sleep is reduced to around 20 percent of sleep time. With the onset of puberty REM remains at around 20 percent, but slow wave activity (SWA) starts to slowly decline until old age, where it may even entirely disappear for some elderly.

3.2 The Gut Micro-Biome and Sleep across the Lifespan

Our social-ecological theoretical framework suggests that these lifespan sleep schedules do not occur in isolation from the rest of the internal bodily

Table 3.1 Objective and subjective time in bed, total sleep time, and sleep efficiency, stratified by age and sex

	Time in Bed, Hours			Total Sleep Time, Hours			Sleep Efficiency, %		
Strata by Age and Sex	N	Actigraphy Mean ± SD	Diary Mean ± SD	N	Actigraphy Mean ± SD	Diary Mean ± SD	N	Actigraphy Mean ± SD	Diary Mean ± SD
6–13 years									
Total	900	9.2 ± 0.7	10.2 ± 0.6	900	7.7 ± 0.7	9.6 ± 0.7	900	83.8 ± 4	93.5 ± 5
Male	427	9.2 ± 0.7	10.2 ± 0.6	427	7.6 ± 0.7	9.6 ± 0.7	427	82.7 ± 5	93.6 ± 5
Female	473	9.2 ± 0.7	10.2 ± 0.6	473	7.8 ± 0.7	9.6 ± 0.8	473	84.7 ± 4	93.5 ± 5
14–17 years									
Total	486	8.4 ± 0.8	8.9 ± 0.8	486	7.2 ± 0.9	8.5 ± 0.8	486	85.9 ± 7	95.6 ± 4
Male	221	8.3 ± 0.8	8.8 ± 0.8	221	7.0 ± 0.8	8.5 ± 0.7	221	84.6 ± 6	95.9 ± 4
Female	265	8.4 ± 0.8	8.9 ± 0.8	265	7.3 ± 0.9	8.4 ± 0.8	265	87.1 ± 8	95.2 ± 4
41–64 years									
Total	1,270	–	8.1 ± 0.9	1,270	6.0 ± 0.9	6.8 ± 0.9	1,270	74.4 ± 9	84.6 ± 10
Male	557	–	7.8 ± 0.8	557	5.7 ± 0.9	6.8 ± 0.9	557	73.5 ± 9	86.9 ± 9
Female	713	–	8.3 ± 0.8	713	6.2 ± 0.8	6.8 ± 0.9	713	75.1 ± 8	82.8 ± 10
≥65 years									
Total	668	–	8.4 ± 0.8	668	6.2 ± 0.9	6.9 ± 1.0	668	73.9 ± 8	82.3 ± 11
Male	319	–	8.3 ± 0.8	319	6.1 ± 0.9	7.0 ± 1.0	319	73.3 ± 9	84.4 ± 11
Female	349	–	8.4 ± 0.8	349	6.3 ± 0.9	6.7 ± 1.0	349	74.4 ± 8	80.3 ± 10

Note. Sleep characteristics across the lifespan in 1.1 million people from the Netherlands, United Kingdom, and United States: a systematic review and meta-analysis by Kocevska et al. (2021); prevalence was not calculated if there were <200 participants in a cell. Abbreviations: N = sample size; SD = standard deviation.
Used with permission from Kocevska et al. (2021).

systems and physiology, or the surrounding social and ecological environment. With respect to the relation of sleep schedules to other bodily systems, take for example that other brain located in the human body: the gut microbiome. The microbes that colonize the small and large intestines activate and help to active the gut microbiome and build it up over time. This microbiome has its own genome, its own enteric nervous system (with the vagus nerve playing a major role), and its own signaling molecules and systems. It acts as a real "mini-brain" or information processing device that helps to regulate all kinds of bodily functions, including those cerebral brain signaling systems that modulate higher cortical functions as well as sleep functions. Enteroendocrine cells (EEC) that line the gut secrete neuroendocrine and neurotransmitter signaling systems that act to modulate sleep-related sites in the brainstem as well as nucleus tractus solitarii in the medulla, and the suprachiasmatic nucleus (SCN) within the hypothalamus. At crucial developmental points (sensitive periods) marking transitions between developmental phases (weaning triggering transition to solid diet and childhood, etc.), the communications along the microbiota-gut-brain axis increase and if interfered with, cause developmental delays and dysfunction. Disruption of the mini-brain during infancy will disrupt development of sleep systems and vice versa. After the transition from infancy, a complex adult-like microbiota is established, dominated by *Bacteroides*, *Prevotella*, *Ruminococcus*, *Clostridium*, and *Veillonella* bacteria. REM sleep is dramatically reduced from about 50 percent of sleep time to 20 percent. The individual's sleep patterns become sensitive to alteration to intestinal permeability, immune system activation, inflammation, energy harvest, and the diversity of the gut microbiota. Similarly, at puberty and then at senescence, further dramatic alterations take place in the gut microbiome and in sleep wake cycles and architecture. These facts illustrate the deep embeddedness of sleep within the individual's physiology and its acute sensitivity to surrounding social and ecological influences at each stage of life (Figure 3.1). It is becoming increasingly clear that sleep is regulated by at least three brains: the mini-brain gut microbiome, the forebrain, and the information network embedded in the surrounding cultural/social and ecological matrix.

There appears to be several critical life history transitional periods when sleep is dramatically altered by these three brains (gut, forebrain, and social-cultural surround) for better or ill in response to prevailing local, social, and ecological conditions. To understand these broad lifespan trends in human sleep schedules in relation to life history transitions, we turn to evolutionary biological principles.

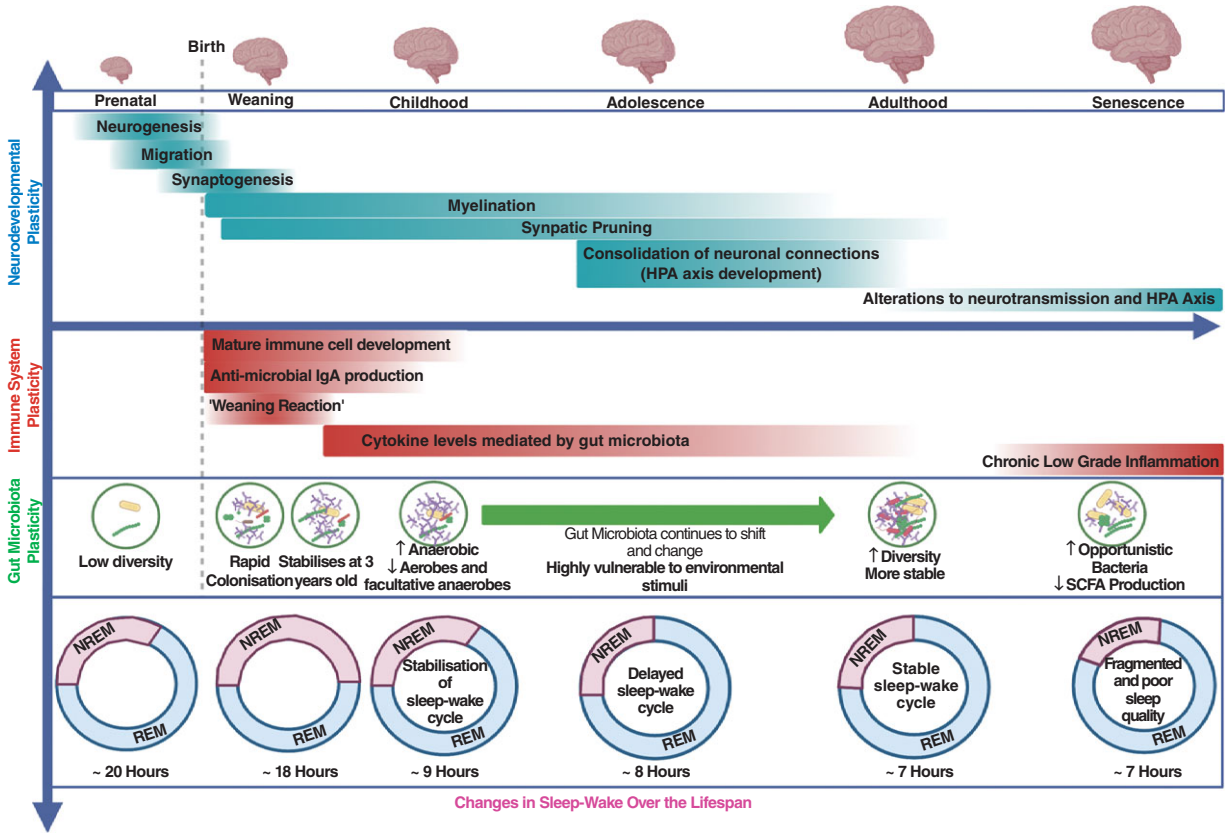

Figure 3.1 Representation of the development of the brain, immune system, gut microbiota, and sleep-wake cycle
Used with permission from Sgro et al. (2022)

3.3 Evolutionary Background to Lifespan Sleep Schedules

3.3.1 Heterochrony or Changes in the Timing of Neurodevelopmental Processes

Evolutionary innovations generally occur via alterations of developmental forms and schedules. When we examine lifespan development of sleep, therefore, we necessarily enter the territory of evolutionary biology. McNamara (1997) and others (e.g., Burns, 2007) have noted that hypermorphosis or the extension of growth times (relative to our primate ancestors) within each normal phase of development (infancy, childhood, adolescence, and maturity, etc.) captures the unique character of human development across the lifespan. We have long drawn out growth phases within each of the classical periods of primate lifespan development. That means that the brain grows larger during infancy and childhood simply because it grows for a longer period of time within the infancy and childhood phases (again, relative to our nearest evolutionary relatives). Because hypermorphosis characterizes human developmental schedules, we can expect that human sleep cycles will necessarily be affected particularly during periods of plasticity or transition between life stages. For example, if sleep functions in part to promote brain development (see more shortly) then sleep cycles may operate more intensely during the period of life where most brain development occurs – infancy and childhood. Some forms of sleep, such as slow wave sleep, decline dramatically or completely disappear in old age. Both REM and NREM sleep appear to significantly intensify for both sexes during the transition to reproductive adulthood (i.e., during adolescence). Sleep patterns shift dramatically in women during pregnancy and birth and after menopause. Hypermorphosis implies that during infancy and childhood, sleep cycles have had to either (1) become longer or (2) more intense than they are in other primates or at other times of the life cycle. Human lineages appear to have chosen both options. But hypermorphosis and developmental sleep schedules more specifically have been dramatically influenced by other evolutionary pressures, to which we turn next.

3.3.2 Executive Control Neural Networks, Technology, and Sleep

One of the major forces driving the evolution of brain function in human beings appears to be social forces (i.e., the pressures of living in groups). Pressures for social cooperation shaped human brain evolution in significant ways, but they were not the only drivers of human brain evolution.

Larger cultural forces also played a role. Cooperation requires some control over individual free riders and aggression. Thus, for social cooperation to stabilize within human groups, reactive forms of aggression had to be reined in and controlled. Brain mechanisms for control of reactive aggression thus needed to be developed and supported. Note that this enhanced control over reactive aggression would then allow for more controlled and perhaps more lethal forms of strategic aggression. Similar control mechanisms, however, were also required to support innovations in toolmaking capacities. Tool and weapon use and development are simply impossible without exquisite levels of control over attentional processes and eye-motor coordination, among other capacities. Executive control processes were also required to meet new challenges posed by the new colder and darker climates anatomically modern human (AMH) populations were facing as they moved into Europe. Not only did these populations interact with existing hominin populations, the Neanderthals and Denisovans, but they also likely needed to deal with new diseases encountered when meeting new populations and new environments. All of these matters, larger social groups requiring greater cooperation, more complex tool kits, new environmental and climate challenges, and new diseases, required new levels of cognitive control and were certainly significant drivers of human brain evolution regardless of social pressures. The evolution of brain systems of executive control likely involved expansion of inhibitory powers of prefrontal and parietal cortex. Interestingly, there is increasing evidence that REM sleep both drove the evolution of these executive control networks in the brain and were dramatically influenced and shaped by these evolutionary innovations in brain connectivity patterns. REM-related electrophysiologic oscillations in the theta band (4–8 Hz) over medial frontal recording sites are becoming increasingly established as a direct neural index of executive cognitive control. REM theta acts to coordinate multiple neural networks in disparate brain regions to process predictive errors and to update predictive models/beliefs guiding daytime behaviors. REM theta activity when coupled with gamma oscillations in prefrontal regions appears to increase when the organism encounters surprise or when daytime information conflicts with or deviates from predictive models or expectations. As such, REM theta both signals the need for model updating and coordinates that updating process. REM theta when coupled with oscillations in the gamma band functions to couple hippocampal-anterior cingulate-prefrontal cortical networks. This rhythmic synchrony or coupling between these brain regions puts analysis processes in

communication with planning and motor output processes, thus permitting optimal model updating and cognitive control. We will return to REM theta effects later when we discuss its role in memory processing. Presumably, as discussed, social and ecologic processes put pressure on our evolutionary ancestors to rely more heavily on systems of central executive cognitive control and thus, REM theta systems began to modulate brain expansion processes in our lineage.

3.3.3 Cumulative Cultural Evolution, Executive Cognitive Control, and REM Sleep

As mentioned in the previous section, human brain evolution accented development of executive control networks in the brain. Brain mechanisms of both emotional and executive control are necessary for creation of the kinds of behavioral plasticity utilized by what is now called cumulative cultural evolution (CCE). CCE observes that humans are subject to two forms of evolutionary pressure: natural Darwinian selection and cultural selection. Cultural selection entails the handing down across generations of learned social practices via cognitive processes of social imitation and learning. For example, the production processes of complex stone tool kits and socially shared symbolic knowledge within local cultures are examples of CCE. Tool kits get more effective and complex over time because they build upon already acquired knowledge and avoid reinventing the wheel with each generation. CCE requires cognitive control, technical procedural knowledge, and flexible learning skills. CCE brains require exquisitely precise and detailed inhibitory efferents running from prefrontal and parietal executive control networks to every other region of the brain. Thus, CCE will eventually influence sleep in such a way as to support build up cognitive of control networks.

Perhaps the most important form of CCE is the development of tools and technology. Neuroanatomical changes associated with the evolution of technology include the parietal precuneus, intraparietal sulcus, supramarginal gyrus, anterior insula, and visuospatial association cortex (Bruner et al., 2018). We will see all of these sites activated during REM. Instead of a reduction in size these sites are larger in AMH populations relative to Neanderthals. These regions, especially in their interactions with prefrontal cortex, are involved in executive control and attentional networks as well as integration of bodily representations with vision, eye-hand coordination, and very complex experiences such as self-awareness. Bruner argues that these are

the brain systems that facilitate integrating tools into cognitive schemes of the body.

Whether it was the need for social cooperation, a reduction in reactive aggression, unpredictable climate changes, or increasingly complex tool kits that propelled evolution of brain structure and function in anatomically modern human or AMH populations, the phenomenon we now call REM sleep and dreams found itself centrally embedded within the overall cultural evolutionary forces that were now fueling the evolution of the human race itself.

3.3.4 REM Sleep and Dreams as Critical for Cumulative Cultural Evolution

Cumulative cultural evolution took off in the upper paleolithic in the Eurasian ecologic context. Anatomically modern humans (AMH) dispersing out of Africa between 75,000 and 50,000 years ago and meeting other human populations such as the Neanderthals had to adapt to these new populations, new diseases, and colder climates. Recent analyses of Neanderthal DNA have demonstrated that migrating AMH peoples mated with some Neanderthals. Among the Neanderthal genes preserved in AMH populations are genes (e.g., ASB1, EXOC6) that enhanced REM sleep processes in several ways but also resulted in a higher risk for narcolepsy – a disorder wherein REM neurobiology is dramatically up-regulated and disinhibited, resulting in REM processes seeping into daily waking consciousness. REM neurobiology, therefore, thrust itself upon or became available in a new way for the AMH peoples of the upper paleolithic – indeed it likely became an intrusive presence for some persons predisposed to dissociative states.

3.3.5 Humans Differentially Invested in REM in the Upper Paleolithic

Nunn and Samson (2018) analyzed a large data set composed of a host of sleep, ecological, physiologic, and life history characteristics of some thirty differing primate species including humans. It turns out that humans sleep less than predicted for a primate of our body mass, predation risk, brain size, foraging needs, sexual selection, and diet. Humans were predicted to spend 13.8 percent of their TST in REM. The observed value was 22.3 percent. Humans pack an unexpectedly higher proportion of REM sleep into a shorter overall sleep duration compared to other primates and do so by reducing NREM sleep (rather than increasing REM). The authors

themselves suggest that the evolutionary reduction in NREM and investment in REM produced new opportunities for learning, creating material objects, and socializing. In my view, it is reasonable to suppose that this new evolutionary opportunity for cultural learning, afforded by NREM reduction and REM enhancement. The reduction in daylight hours and the colder climate meant that AMH peoples had to adjust their sleep habits and they did so by using REM more intensely. Why is all this important for developmental sleep schedules? Well it turns out that REM may be critical for developmental creativity.

Now lets discuss narcolepsy – the developmental disorder that may have become a more insistent reality for some peoples of the upper paleolithic if the gene in question (EXOC6) was operating in the same manner as it does for modern populations (i.e., increasing risk for narcolepsy). Narcolepsy is characterized by the irruption of REM neurobiology into daytime consciousness. Its symptoms are (1) excessive daytime sleepiness; (2) hypnogogic hallucinations; and/or (3) "sleep attacks" or sudden paralysis (cataplexy) following a strong emotional stimulus such as laughing or an intense emotion. But if REM also activates the theta system, thereby increasing coupling between limbic and prefrontal cognitive control systems, narcolepsy should also carry some benefits. Lacaux et al. (2019) formally assessed links between REM neurobiology and cognitive creativity in 185 narcoleptics and 126 healthy controls. They did indeed find that narcoleptics evidence higher levels of creativity than controls as assessed in multiple ways. They were more creative on tests of past achievement and in-lab objective creativity tests that tapped "Imaginative, Innovative, and Researcher" modes of creativity as well as divergent, convergent, verbal, graphic, abstract, and concrete modes of creative thinking. Notably, 43 percent of the 185 subjects with narcolepsy were frequent lucid dreamers compared to 3 percent of the 126 normal controls, and these lucid dreamers were among the most creative of the group (Lacaux et al., 1988–1999).

While it is increasingly clear that REM is important for both evolution of the brain and its ontogenetic development within individual lifespan development, REM's and NREM's role in lifespan development of the human person needs to be appreciated within the organism's overall social and ecological environmental conditions. We also must always keep in mind that developmental schedules are aimed at optimizing long-term reproductive fitness calculations. We mentioned previously long-term evolutionary pressures impacting sleep and developmental schedule. Now we will focus in on more proximal forces influencing lifespan sleep expression. The relevant evolutionarily informed forces once again point to the clear role of social forces in shaping sleep. These

mid-level theoretical frameworks are known as parent-offspring conflict theory, life history theory, and attachment theory.

3.3.6 Parent-Offspring Conflict and Sleep

Postnatal sleep states in the infant develop in the context of mother-infant interactions. Development of mother-infant interactions, in turn, occur within the broader context of conflict between mother and child over amount and quality of resources to be invested in the infant. This conflict over provisioning of, or investment in, the infant is driven by the contrasting genetic interests of the mother versus those of the infant. The infant wants more resources than the mother can, or is willing, to give and thus the struggle ensues.

Trivers (1974) pointed out that parents are related to their offspring by a coefficient of relatedness of 0.5; that is, the child carries only half of the maternal genomic complement. Consequently, the genetic interests of mother and child are not identical and offspring will tend to want more from their parents than their parents are willing or able to give. Offspring in mammals, furthermore, may not share the same father, and, thus, their genetic interests will diverge significantly from those of their siblings. Even if they do share the same father, they are related to siblings only by 0.5 and so their interests are not identical to those of their siblings. Offspring should therefore attempt to monopolize extraction of resources from the mother regardless of consequences to the mother or siblings. The infant's strategies to obtain more resources from the caregiver include vocalizations, grasping, smiling, crying, wailing, and suckling, to name a few. Sleep represents a period of relief for the mother unless the infant suckles while asleep. Thus, night-wakings, in particular, become a battleground between the mother/caregiver and the infant. We will see in the next section that infantile night-wakings are related to the type of attachment bond created between mother and child.

3.3.7 Life History Theory and Sleep

According to life history theory (Stearns, 1992), life cycle traits such as gestation length, size and number of offspring, age at first reproduction, lactation/weaning period, ongoing reproductive strategy, and length of life are all influenced by local ecologic and social context and contribute to reproductive fitness. Individuals develop mechanisms or biobehavioral strategies that help them solve problems related to infant survival, childhood growth, adult development, and reproduction across the lifespan. Perceptual-emotional information about current environmental conditions (e.g., local

mortality rates) is used to make (unconscious) decisions about optimal allocation of limited resources.

Trade-offs have to be made between time and energy devoted to "somatic effort" (i.e., investing in growth and development of the body) versus time and energy devoted to "reproductive effort" (i.e., funneling effort toward producing and raising offspring). Since sleep functions, in part, to regulate energy budgets, sleep quotas (time devoted to sleep) will vary, to some extent, depending on somatic or reproductive aims. Reproductive effort has two further components: mating effort (locating, courting, and retaining a suitable mate) and parenting effort (i.e., gestating, giving birth, and engaging in postnatal care). Given the impact of developmental sleep processes on brain development, it is possible that sleep processes may figure in unconscious decisions concerning somatic development as well as development of behavioral strategies to support reproductive effort (parent-offspring relations and mating strategies).

Those unconscious decisions concerning developmental schedules very likely rest on an intuitive grasp of one's own genetic inheritance as well as one's current experience of the social environment. Some investigators (e.g., Belsky & Chisholm, 1999) have suggested that for the neonate and the juvenile, making a bet on future social-ecologic conditions must be based on their inherited genetic profile and current experience with their caregivers. If the caregivers invest only minimal resources in the child, then the child concludes that local, social, and ecologic conditions are adverse and he sets developmental schedules accordingly: "Grow up fast, sleep little and sire many kids as you will be dead soon." If on the other hand caregivers invest quality time and resources into the child, then the child gets the message that the social and ecologic context is favorable, whereupon the child will opt for a slower developmental schedule, longer or more intense sleep cycles, and a longer lifespan. "Grow up slowly, sleep intensely, invest heavily into one or two kids and live longer."

Dishakjian et al. (2020) tested some of these predictions concerning the impact of life history strategy on sleep schedules among 568 individuals of all different ages. They used segmented and hierarchical regression models, structural equation modeling (SEM) and machine learning to model relationships between sleep duration/quality and life history strategy (LHS). Consistent with predictions of LHS they found an age-mediated U- or V-shaped relationship between LHS and lifespan expression of sleep duration averages, sleep valuation, and sleep schedules. People who are on a fast life history track (live fast, reproduce early, and die young) evidenced more extremes of very short or very long sleep durations, and greater variability in sleep valuation; that is, these people occupied the sections of the curve with the highest mortality risk (<6.5

hours (short sleep) and >8.5 hours (long sleep)) and worse overall sleep quality (delayed sleep onset latency, more wakefulness after sleep onset, higher sleep–wake instability, and greater sleep duration variability). Interestingly, the life history measure turned out to be the strongest predictor of sleep measures in the dataset; that is, life history strategy accounted for the greatest amount of variance in sleep quality measures even after controlling for mating effort, hedonic valuations of sleep, and demographic variables. In their SEM of the relations between life history strategy and sleep outcomes, the authors were able to put some numbers on downstream effects of life history strategy on variables influencing sleep outcomes (see Figure 3.2). There appears to be a positive association between WASO (waking after sleep onset) and SOL (sleep onset latency) with sleepiness. That association is mediated by sleep-wake instability downstream of the fast LH strategy. LH slowness, in turn, is associated with less risk for STMO (short-term mating orientation) and an overall lower lifetime number of sexual partners. The path from a slow life history strategy and long-term mating strategy leads positively to stability in all other measures as well as positive hedonic valuation of sleep; these factors in turn confer some protection against sleep deficits (negative red path between deficits and stability).

3.3.8 Attachment Theory and Sleep

Bowlby (1969) and Ainsworth (Ainsworth et al., 1978) developed attachment theory. The basic idea behind attachment theory is that between birth and eighteen months infants develop a particular bond of emotional trust with their caregiver. Bowlby regarded the attachment system as a biobehavioral regulatory process that adjusted physical and emotional closeness between caregiver and child. The attachment system expects the optimal bonding style called "secure attachment" wherein the infant is consistently cared for by the caregiver and therefore develops comfort with closeness and comfort with leaving the caretaker's immediate physical presence to explore a little.

Through her "strange situation procedure" (SSP), Ainsworth was able to identify insecure attachment orientations. A child with inconsistent parenting develops an insecure-anxious orientation that is evidenced by inability to tolerate separation from caregivers without emotional crisis. These kids are difficult to console after separation and prefer to continually seek proximity to the caregiver. A child with insecure-avoidant orientation on the other hand, prefers to avoid interactions with the caregiver and appears emotionally inhibited.

Similar attachment orientations occur in adults and such attachment orientations are conceived as cognitive working models capturing perspectival relations between the self and the attachment object (see Table 3.2). That is, each of us cognitively represent attachment relations as representation of

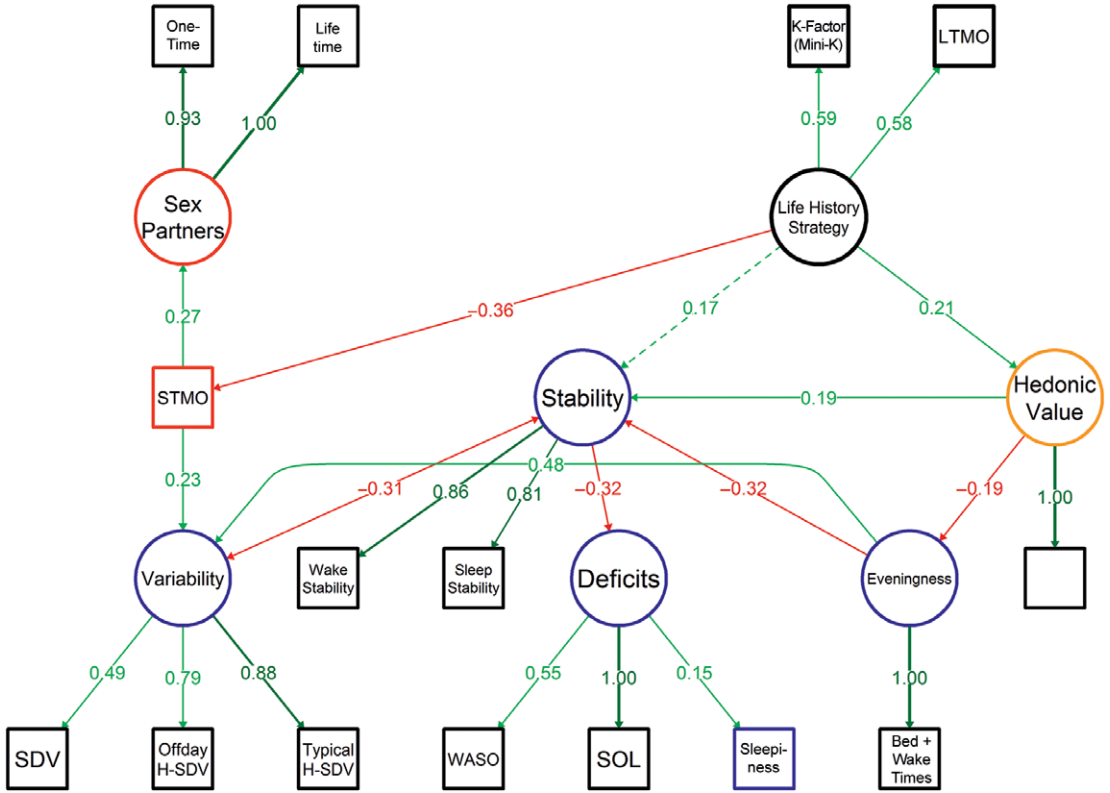

Figure 3.2 Structural equation model of life history strategy's effects on maintenance and reproduction allocations

(Paths are labeled with standardized weights. Variables with a red border indicate investment into short-term reproduction, while variables with blue borders indicate investment into long-term maintenance. STMO (short term mating orientation) is regressed (rather than loaded) on life history strategy, and sleepiness is regressed (rather than loaded) on deficits. SDV = sleep duration variability. Hedonic value = extent to which individuals value sleep. All solid line paths are significant at $P < 0.05$, while the dashed path coefficients have P values greater than 0.05.

Used with permission from Dishakjian et al. (2020)

Table 3.2 Attachment orientations as a function of internal working models of self versus other

		Self is perceived as	
		Positive	Negative
Attachment target is perceived as	Positive	**Secure** Individual is comfortable with both self and attachment target; wants to be in a relationship and is comfortable in a relationship.	**Preoccupied** Individual has negative view of self and an overly positive view of attachment target. Individual is anxious to be in a relationship.
	Negative	**Dismissive** Individual has an overly positive view of self and an overly negative view of the attachment target. Individual claims low need for intimacy.	**Fearful** Individual has negative view of both self and attachment target. Individual is anxiously avoidant of relationships.

relations between self and other. Adults with secure orientations tend to have positive views of themselves and their attachment targets and are comfortable with intimacy and independence. Individuals with insecure-ambivalent or preoccupied orientations overly value their attachment targets while devaluing themselves and are uncomfortable with independence. Individuals with insecure-avoidant orientations have negative views of themselves and their attachment targets but are comfortable with independence. And finally, individuals with dismissive orientations have inflated views of themselves and negative views of their attachment objects. There is increasing evidence that these internal working models of self and other are formed during REM dreaming. I will return to this issue in a later chapter on dreams and attachment themes. For now we are focused on the interrelationships between developmental schedules, social attachment processes, and sleep.

The attachment relationship is crucial for development of sleep-wake schedules. If the neonate/child can form a secure emotional attachment to the mother, the child will "conclude" that the local environment will support adequate resources and a long-term reproductive strategy of delayed maturity and high investment in a few high-quality offspring. In that case, infantile sleep intensity should be high, while night-wakings, nightmares, and other

sleep disturbances should occur infrequently. We will see in the next section that recent studies support this prediction.

3.4 Lifespan Development of Sleep

3.4.1 Fetal Sleep

All mammals studied to date exhibit a period of spontaneous and mixed brain activity in utero known as indeterminate sleep, which slowly differentiates into distinct sleep states by the middle of the pregnancy. Beginning at approximately twenty-eight to thirty-two weeks gestational age (ga), two forms of sleep – quiet sleep (QS) and active sleep (AS) – develop out of this "indeterminate sleep." At about twenty-eight weeks ga discrete periods characterized by rems and respiratory movements begin alternating with periods of sustained motor quiescence with no or very low numbers of eye movements. A REM–like active sleep state appears between thirty and thirty-two weeks ga and increases in amount until it comprises approximately 90 percent of fetal sleep. REM remains at about 90 percent of sleep until about one to two weeks of postnatal life; then it begins a relatively rapid decline toward adult values. No one knows why the developing fetus spends most of its time in REM, though this fact is consistent with the idea that REM functions to support brain development.

3.4.2 Neonatal Sleep

Newborns sleep about sixteen hours a day. In contrast to the adult pattern of entering sleep via an NREM episode, normal, full-term newborns enter sleep through a REM-like state rather than an NREM-like state. Infant sleep is equally divided between AS and QS, which alternate in a fifty-minute cycle. In other words, there are typically six to eight regularly occurring sleep periods in a twenty-four-hour day for a typical newborn. Significant variability exists, however, so that some newborns, for the first few days after birth, sleep for eighteen to twenty hours, while others sleep for only ten to twelve hours in a twenty-four-hour period (Burnham et al., 2002). Within the first month following birth, sleep-wake state organization begins to adapt to the light-dark cycle and to social cues.

The infantile precursor sleep states (AS and QS) begin to approximate adult forms of REM and NREM by about three to six months. With increasing maturity, the proportionate amount of time in REM sleep diminishes. The two- to three-year-old child spends approximately 35 percent of sleep time in REM sleep, while the adult spends about 20 percent in REM sleep. After three

Table 3.3 Sleep changes from neonatal to adult period

	Infant	Adult
%REM/NREM	50/50	20/80
REM/NREM cycle	50–60 minutes	90–100 minutes
Sleep onset state	REM	NREM
Temporal organization of sleep states	REM/NREM cycles equally throughout sleep period	NREM predominates in first third of night; REM predominates in last third of night
Sleep architecture	1 NREM stage	3 NREM stages

months of age, REM periods continue to recur with a periodicity of fifty to sixty minutes. However, the amount of REM sleep in each cycle begins to shift. REM sleep predominates in the later sleep cycles of the night and NREM sleep predominates during the earlier cycles, especially NREM stage N3 sleep. By three years of age, the temporal organization of sleep during the night resembles that of adult sleep except for the sleep cycle periodicity, which does not lengthen to the ninety-minute periodicity of adults until adolescence.

Studies of the effects of maternal separation on developing rats (Hofer & Shair, 1982) and monkeys (Reite et al., 1976; Reite & Short, 1978) provide a dramatic illustration of how AS/REM values are linked with infant-mother contact. These studies have conclusively shown that measures of AS/REM sleep (but not NREM or SWS) are selectively influenced after maternal separation. There is an initial increase in AS/REM times and then a dramatic reduction after separation.

In contrast to REM, where the maturation of electrographic features are quite prolonged (e.g., PGO waves are not present in the kitten until three weeks postnatally), the maturation of NREM brain activity (slow waves in the delta bands 0.5–4.0 Hz and sleep spindles at 7–14 Hz) is completed in a relatively short time. EEG slow waves are generally reported to develop as isolated slow waves in a burst suppression EEG pattern called "trace alternant" in the human infant. This pattern, in turn, is replaced later with a more continuous slow wave pattern during the course of development. Sleep spindles appear later as well.

3.4.3 Attachment and Infant Sleep

Burnham et al. (2002) reported significant variability in number of night-wakings/arousals in the infant. At one month, infants woke an average of 4.12

(SD = 2.57) times, with a range from one to eleven times. At twelve months, infants were waking an average of 2.62 times (SD = 2.03), with a range of zero to ten times. Evolutionary theory predicts that night-wakings should vary with attachment status. When nighttime awakenings are followed by an infant signaling episode such as crying they are more likely to elicit a maternal intervention than when no signaling occurs after an awakening. Several forms of nighttime signaling can influence attachment processes including crying, sucking, nursing, smiling, grasping, twitches, cooing, babbling, and other vocalizations. All of these behaviors are more likely to either occur in, or to emerge from, REM rather than NREM sleep (see McNamara, 2004 for review).

Consistent with evolutionary theory, night-wakings are known to be sensitive to attachment status. In a study of twenty infants judged insecurely attached to mother, Benoit et al. (1992) reported that 100 percent of these children evidenced severe sleep disorders. When Sagi et al. (1994) studied the effects of sleeping arrangements in Israeli kibbutzim (communal vs. home) on attachment security, they found that 52 percent of communal-sleeping infants (i.e., those who slept away from their parents' home) and only 20 percent of home-sleeping infants were later classified as insecurely attached. Scher (2001) reported that securely attached infants awaken once per night, and stay awake for about eleven minutes. A total of 43 percent of "dependent secure" (i.e., B4: secure infants who nevertheless exhibit some ambivalence as well) and 23 percent of the mothers of secure infants reported that their infants had settling difficulties. Infants with frequent night awakenings scored higher on contact maintenance in the "Strange Situation" experimental procedure than non–night waking infants. Beijers and colleagues' (2011) found that infants that were classified as insecure resistant, or insecure-anxious, evidenced the most frequent number of night-wakings at six months of age, whereas insecure-avoidant infants evidenced the least. McNamara and colleagues (2003) reported a similar pattern with insecure-anxious/resistant infants evidencing more and longer night-wakings than insecure avoidant infants at fifteen months. Morrell and Steele (2003) also found that insecure-anxious or insecure-resistant attachment in twelve-month-olds was associated with frequent night-wakings and was predictive of persistent sleep problems in a one-year follow-up. Taken together, these studies suggest that insecure-anxious attachment in infants is associated with a disproportionately high number of night-wakings between six and eighteen months, while infants with insecure-avoidant and secure orientations tend to evidence night-wakings much less frequently in that time period. Why is that? If night-wakings function to extract greater resources from the mother then infants with secure orientations will not need to utilize that behavior as they perceive themselves to be adequately provisioned. Insecure avoidant infants do not signal as often as insecure-

anxious/resistant as they have adopted the avoidant strategy. We will see in the next section that these infantile night-waking patterns make sense within an evolutionary context.

3.4.4 Infant Sleep and Genetic Conflict

Every new parent knows that it is exhausting to be a new parent. Your baby keeps you awake all night with frequent night-wakings and loud "vocalizations" or crying episodes. There is a huge industry composed of supposed experts on infant sleep that advise new parents on how to get their baby to sleep through the night so that parental sleep patterns can return to normal. Why would Mother Nature produce such a seemingly maladaptive pattern of sleep in the neonate? It does no one any good if neither the baby nor the parents get any sleep and are chronically sleep deprived. Haig (2014) and Blurton Jones and da Costa (1987) argued that infant night-wakings function to prolong interbirth intervals via nursing-induced suppression of ovulation. That is, if the mother nurses the infant she cannot get pregnant (nursing induces suppression of ovulation). If the mother does not have another baby while the current baby is struggling to survive the first couple of years of life, then that baby will get more resources from the mother, and thus its chances for survival will increase. We have seen that it is the infants with insecure-anxious/resistant orientations that experience the greatest number of night-wakings. Presumably these infants' night-wakings result in nursing episodes that suppress ovulation in the mother and thus, the infant can monopolize maternal resources for a while. If night-wakings decline over time at a slower rate for insecure resistant versus avoidant infants, then the point at which maximal benefits of contraceptive suckling are obtained may differ for infants dependent upon attachment orientation.

Optimal inter-birth intervals may differ for mother-infant dyads depending on both attachment status and social-ecologic context. In environments with high mortality rates parents pursue an opportunistic reproductive strategy aimed at greater numbers of offspring appearing at shorter ibis and receiving lower levels of investment (e.g., reduced nursing), resulting in fewer night-wakings and greater numbers of avoidant attachment orientations.

3.4.5 Sleep in Childhood

From toddlerhood up through early teens, children begin to sleep about ten hours a night. Of course, that is only an average and there is great variability around this average. Children quickly go into N3 stage SWS for about an hour and then briefly arouse, are cognitively confused ("confusional arousals"), turn

over in bed, perhaps emit vocalizations, and then return back into N3, skipping altogether the first REM period. When they do go into their first REM period, it lasts about twenty minutes and then they cycle normally between REM and NREM for the rest of the night. The amount and intensity of SWS peaks around age four in humans. This is interesting, as age four is the traditional weaning age in humans. It is certainly easier for a mother to wean a child if the child is asleep and not demanding attention. It is also around this age that night terrors may occur. Night terrors can occur between ages three and twelve, but they peak at around age four. They involve confusional arousals out of N3 SWS. The child may sit on the side of the bed and scream bloody terror. Their eyes may be wide open, but they may be unresponsive to others during the episode. It takes a lot to shake them awake.

We have seen that infant sleep is embedded within the social context of mother-infant genetic conflict and social attachment patterns. Attachment orientation in the child continues to influence childhood sleep patterns during the years of childhood. Specifically, attachment security predicts sleep patterns into adolescence and vice versa, suggesting that secure attachment promotes more stable sleep patterns. Troxel and colleagues (2013) reported that children rated high in negative emotionality in infancy (negative emotionality is often linked to insecure attachment in infancy) evidenced stronger associations between their attachment orientations and sleep patterns in toddlerhood. Keller (2011) reported that mother-child attachment security was predictive of decreased subjective sleepiness during the day for both boys and girls. Among boys, increased physical restlessness at night (indicating short arousals/awakenings) as measured by wrist actigraph in third grade predicted a decrease of mother-child attachment security in fifth grade. Additionally, a boy's emotional distress about his parent's marital relationship in third grade predicted sleep problems in fifth grade.

3.4.6 Sleep in Teens

Adolescence is associated with a decline in the percentage of total sleep time composed of SWS. Sleep scientists generally agree that teens need about ten hours of sleep per night but get only eight or less. Sleep-deprived teens make more impulsive decisions, are involved in more accidents, and generally are more moody than non-sleep-deprived teens. Sleep loss in teens appears to be related to school demands and social pressures. The widespread use of smartphones and other electronic devices with brightly lit screens is also having a negative impact on sleep times in teens. Adjusting school schedules so that teens get the sleep they need has demonstrated positive results, but the trend has not caught on.

Adolescence, of course, is associated with the onset of puberty and this biological storm both influences and is influenced by sleep. The onset of puberty is triggered by the hypothalamic-initiated release of the gonadotropin-releasing hormone (gnrh), luteinizing hormone (LH), and follicle-stimulating hormone (FSH). Under the influence of these hormones, secondary sexual characteristics emerge, including voice changes, height gains, breast development, genital development, and cellular changes in sexually dimorphic brain regions such as the amygdala and the hypothalamus. The pulsatile release of these hormones may be influenced to some extent by melatonin and sleep cycles. Virtually all of the growth hormones released during these years occur during sleep, and many of the hormones controlling onset of puberty are also influenced by sleep processes.

REM deprivation early in life is associated with later impairment in sexual functions in the adult, at least in the rat and monkey. REM is also associated with cyclic occurrence of penile erections in men and vaginal lubrication, clitoral engorgement, and pelvic thrusting in females. Total length of time (mean of 190 minutes) spent in tumescence during REM peaks in teenaged boys. Nocturnal emissions begin to occur during adolescence, probably during the REM period.

3.4.7 Sleep in Adulthood: Women

Women's sleep is influenced by the menstrual cycle. While there are no reported differences in percent of time spent in specific sleep stages, women report subjective changes in feelings of sleepiness. Progesterone release induces sleepiness in most women and this could account for the subjective sense of greater sleepiness. Pregnancy is associated with dramatic increases in the action of gonadal steroids, such as estrogen and progesterone, and the pituitary hormones prolactin and growth hormone, on the maternal brain and the developing fetus. Fetal sleep influences maternal sleep and vice versa. The steady rise of progesterone activity levels over the course of the pregnancy and up to term is associated with an increase in NREM and a decrease in REM sleep (as well as an increase in latency to REM) in the mother. The placenta stimulates an increase in estrogen and cortisol activity, particularly at term, which tends to inhibit REM. Prolactin enhances SWS with levels peaking four to six hours after sleep onset. Prolactin also promotes lactogenesis. Oxytocin interacts with melatonin activity during labor to promote uterine contractions. Levels of the placental growth hormone (GH) in the mother's blood increase throughout pregnancy and peak around week thirty-five of gestation. GH, in turn, is known to stimulate SWS.

Table 3.4 Sleep in pregnancy

	First Trimester	Second Trimester	Third Trimester	Labor/Delivery	Comments
Sleep measures relative to prepregnancy baselines	Increased total sleep time but decreased percentage of SWS	Decreased total sleep time; vivid dreams of baby animals, etc.; decreased SWS	Increased total sleep time but decreased sleep efficiency and decreased SWS and REM; vivid dreams and occasional anxiety dreams and nightmares	Decreased REM; increased delta waves with labor onset	

From the point of view of sleep state biology, pregnancy is, of course, a unique physiologic process wherein the sleep states of two genetically distinct individuals may interact. Haig (1993) has called attention to the fact that the placenta is genetically part of the fetus and not the mother, and thus, there is potential for a divergence of genetic interests between the fetus and mother. Abnormal triploid fetuses with a double set of the father's genes and a single set of the mother's have a very large placenta, while abnormal fetuses with a double set of the mother's genes and one of the father's have very small placentas and show a retardation of growth. Modeling of genetic strategies of parents and their offspring suggests that with respect to the maternal-fetal interaction, the fetus is selected to extract as many resources from the mother as possible, while the mother is selected to moderate attempts to extract her resources. Her genetic interests lie not just in the present child but in whatever future children she may bear. She needs to be discriminative when it comes to investment of valuable resources in the current child. Future offspring of the mother, therefore, are in direct competition with the current fetus for resources, and so the fetus attempts to extract as much as it can from its mother. The interplay between maternal and fetal sleep processes and growth of the fetus over the course of the pregnancy may be influenced by this genetic background (Table 3.4).

During the first ten weeks of pregnancy, human chorionic gonadotopin (hcg) levels secreted from the trophoblast of the embedded zygote maintains the corpus luteum and its secretions of estrogen and progesterone. These sex steroids together with prolactin (secreted from the pituitary) foster profound physiologic changes and growth in both the mother and the fetus throughout

the pregnancy. Increased levels of progesterone, furthermore, dampen or prevent uterine contractions. Hcg falls in the second trimester as the placenta itself increases its production of placental lactogen (PL), progesterone (P), and estrogen (E). PL is very similar in structure and function to GH. GH is a hormone whose release in males, and to a lesser extent in females, is intimately dependent on sleep: 95 percent of daily production of GH occurs in SWS during development. The relation between GH release and SWS is not merely temporal. The SWS state itself stimulates release of the hormone. SWS in females likely plays a similar role but stimulates release of other growth factors in addition to GH.

3.4.8 Sleep in the Elderly

The most consistently reported change in the sleep architecture of healthy elderly persons is a decline in the percentage of sleep composed of delta wave indexed–slow wave sleep (Bliwise, 2000). In some cases, SWS may occupy only 5–10 percent of total sleep. While there is great variability, REM percentage tends not to decline with age. Women experience the loss of NREM sleep indices about ten years after men. Elderly women evidence twice as many N2 sleep spindles relative to aged-matched men. Despite these objective differences in the sleep patterns of elderly men and women, women report poorer sleep quality than do men, particularly coincident with menopause.

3.4.9 Conclusions

Figure 3.3 sums up some of the main lifespan trends in sleep discovered to date. The time it takes to fall asleep (latency) declines until midlife and then remains about the same into old age. Time spent awake after initial sleep onset (WASO) declines across the lifespan, but its proportion of total sleep period increases. In other words, people tend to have greater number awakenings as they age. REM percentages decline with age, but the proportion of total sleep spent in REM remains about the same. This applies to N2 stage light sleep and N1 transitional sleep as well; these proportions remain about the same or slightly increase as people age. Finally, N3 slow wave sleep undergoes a steady decline with age until it almost completely disappears in old age. Throughout the lifespan, sleep evidences intimate and possibly bi-directional causal associations with socio-emotional attachment processes between child and parent during the developmental phase and then between sexual/romantic and close friends during the adult phase. These relationships between sleep processes and attachment processes once more underline the social nature of sleep.

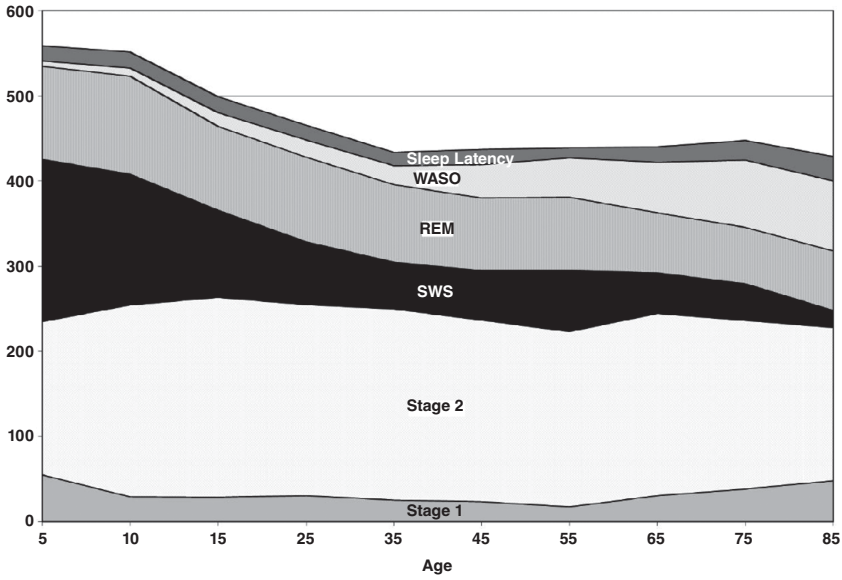

Figure 3.3 Age-related trends for stage 1 sleep, stage 2 sleep, slow wave sleep (SWS), rapid eye movement (REM) sleep, wake after sleep onset (WASO), and sleep latency (in minutes)

We have now completed our survey of the ways in which sleep is expressed across the lifespan and the ways in which evolutionary forces shape that expression. We turn next to a detailed examination of the biobehavioral and neurological characteristics of each of the major sleep states: NREM and REM.

3.5 Review Questions

- What is the significance of the differences in sleep patterns in children with insecure attachment orientations as opposed to children with secure attachment orientations?
- What is the significance of the differences in sleep patterns in adults with insecure attachment orientations as opposed to adults with secure attachment orientations?
- Why does the proportion of slow wave sleep/total sleep decline with age?
- What are the strengths and weaknesses of the evidence for social influences on sleep across the lifespan?

Further Reading

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CHAPTER FOUR

Characteristics of REM and NREM Sleep

Learning Objectives

- Describe the electrophysiologic characteristics of each of the non-REM sleep stages
- Describe the electrophysiologic and bio-behavioral characteristics of REM sleep
- Identify the key brain activation and deactivation patterns associated with NREM sleep
- Identify the key brain activation and deactivation patterns associated with REM sleep

4.1 Introduction

If we want to know about the neurobiology of sleep, we need to very briefly summarize the main features of the neurobiology of wakefulness. In Figure 4.1 the main components of the networks that activate and maintain wakefulness are diagrammed. Two major pathways are shown in Figure 4.1. One (in yellow), rooted in the ascending reticular activating system (ARAS), shows that activating impulses ascend from ARAS via the via the thalamic-relay nuclei as well as reticular nucleus of the thalamus. This input is coming from acetylcholine (ACh)-producing neuronal groups, which are located in the Pedunculopontine and laterodorsal tegmental (PPT/LDT) nuclei of brainstem. The second major group of neurons (in red) are located in noradrenergic (NA) locus coeruleus (LC), serotoninergic (5-HT) dorsal and median raphe nuclei, dopaminergic (DA) periaqueductal gray matter (vPAG), and histaminergic (His) tuberomamillary neurons (TMN). The largely cholinergic groups are in mutual inhibitory balance with the large aminergic sertoniergic/noradrenergic groups. Additional cortical input also originates from the GABA or Ach neurotransmitter containing basal forebrain (BF) neurons as well as from lateral hypothalamic (LH) peptidergic neurons that contain the melanin-concentrating hormone (MCH) or orexin (hypocretin) (ORX).

The second system influencing wakefulness is rooted in the ventrolateral preoptic nucleus (VLPO) and projects to the components of the ARAS

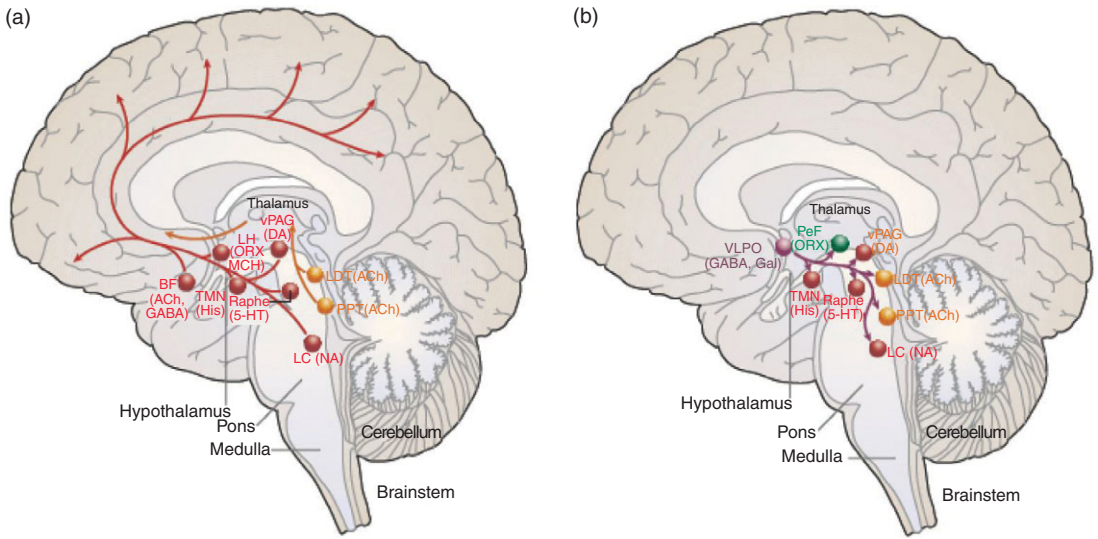

Figure 4.1 The neurobiology and physiology of sleep and wakefulness

(a) Ascending reticular activating system (ARAS): (b) projections from the ventrolateral preoptic nucleus (VLPO) to the components of the ascending wakefulness system. PPT pedunculopontine tegmental area, LDT laterodorsal tegmental area, NA noradrenergic, LC locus coeruleus, 5HT serotoninergic, DA dopaminergic, vPAG periaqueductal gray matter, His histaminergic, TMN tuberomamillary neurons, BF basal forebrain, LH lateral hypothalamus, MCH melaninconcentrating hormone, ORX orexin, VLPO ventrolateral preoptic nucleus).

Used with permission from van Someren and Cluydts (2013)

ascending wakefulness system. We will see later that the VLPO is also involved in a flip-flop switch system that turns sleep cycles on and off.

4.2 Characteristics of NREM

When we move from wakefulness to sleep, we enter the portal of sleep via NREM. Eventually we all MUST succumb to sleep – this odd, undignified, helpless period of behavioral inactivity that is composed of a bizarre mixture of oblivion and the hyper-consciousness that comes with dreaming. NREM encompasses that phase of sleep where we come closest to oblivion and nonetheless remain alive. If we follow a typical individual's path down to deep sleep, we find that we each enter into oblivion via a state of relaxed wakefulness or drowsiness. During this period, the EEG changes from the fast, desynchronized wave pattern of alert wakefulness to a slower, more regular wave pattern at a frequency of 8–12 Hz, known as alpha activity. The alpha waves are replaced by low-amplitude, mixed frequency theta waves with a predominance of activity in the 4–8 Hz range, which characterizes the N1 stage. N1 lasts only a few minutes. Next, the hapless individual headed toward oblivion alights at stage N2 NREM sleep. During stage N2, the individual experiences a form of light sleep. He is easily aroused from this stage of NREM. His EEG shows occasional bursts of higher-frequency oscillations that are called sleep spindles and K-complexes, which are very high amplitude spikes. Sleep spindles are low amplitude, 7–14 Hz synchronous waveforms that often precede the so-called K-complex waveform during stage N2 NREM light sleep. K-complexes occur randomly throughout stage N2 sleep but may also occur in response to auditory stimuli. Spindles, on the other hand, occur in all stages of sleep but "prefer" stage N2 NREM. They propagate in thalamocortical networks and exert strong depolarizing effects on projection targets in the neocortex.

Electrophysiologically and phenomenologically N1 is a drowsy state, transitional from wake to sleep. N2 is a light sleep stage with characteristic electrophysiologic signals called "sleep spindles" and "K-complexes," measured by the EEG. N3 is a deep sleep state characterized by slow wave forms and abundant delta activity. Delta (δ) activity (0.5–4 Hz) becomes increasingly dominant during the progress from light to deep sleep. Delta waves have the largest amplitudes, normally between 20 and 200 μv. Over the course of a single night of sleep there is a progressive decline in delta power (and an increase in REM). Sleep, therefore, promotes dissipation of whatever brain process delta power indexes. There is less and less of a need for delta power as sleep progresses. The magnitude of the delta power seen in sleep is partially dependent on the amount (and intensity) of wakefulness prior to the onset of

sleep. One way that "intensity of wakefulness" has been measured by researchers is via the amount of frontal lobe–dependent executive control and social cognitive tasks engaged in during the waking period. Executive-control cognitive tasks involve things like working memory, paying close attention, vigilance tasks, crunching numbers, and so forth. Social cognitive tasks involve things like attempting to read the minds of others or gauge their intentions and so forth. The link between delta power during sleep and engagement of frontal lobe processes during waking has suggested to some (e.g., Harrison et al., 2000; Anderson & Horne, 2003) that SWS functions in part to restore frontal lobe functions. While that supposition is likely true, sleep, including N3 SWS, also very likely has multiple functions – not just the restoration of frontal lobe functions.

Another EEG pattern that occurs in NREM is the so-called cyclic alternating pattern or CAP (Terzano et al., 1985). This periodic activation pattern appears to occur every twenty to forty seconds, with input-associated alternations of activation dubbed A events (A) and then generalized background (B) periods against which A events occur. A phase can come in three forms: A1, A2, and A3. A1 exhibits purely slow wave constituents (e.g., K-complexes and slow wave groups) with little autonomic and muscle changes but with signs of high homesostatic pressure. A3 exhibits the traditional arousal pattern with desynchronized fast activity, increased autonomic signs, and increases in muscle tone. A2 is a mixture of A1 and A3 features. Topographic brain–mapping studies of the CAP reveal that spectral components with anterior frontal prevalence are typically present in A1 type (0.25–2.5 Hz) events, while those in the A3 type (7–12 Hz) events have a different prevalence over the parieto-occipital areas.

Building on the distinctions between slow oscillations and delta activity and the fact that 1–4 Hz waves reflect thalamic, clock-like delta activity and cortical delta activity, while the <1 Hz component of slow wave activity during NREM is solely of cortical origin and reflects different physiological processes, Halasz et al. (2014) suggested that there were two types of slow wave activity, an instant reactive form and a more long-term nonreactive form – though both forms likely act to protect sleep and frontal lobe activity as well as cognitive functions. The quick–acting slow wave homeostatic form includes electrophysiologic events, such as K-complexes and spindles, which compensate for any potentially sleep disturbing events by providing instant "delta injections" to maintain the nightly delta level, thus protecting sleep and cognitive functions mediated by the frontal lobes.

N2 spindles and K-complexes, however, index more than just protection of sleep via suppression of incoming sensory information. Spindles arise from recurrent inhibition of cortical-projecting thalamic neurons by reticular

thalamic neurons. Spike bursts in thalamo-cortical neurons entrain cortical populations in spindle oscillations. The depolarizing effects of phasic synchronous bursts of spindling activity facilitate an influx of calcium ions (Ca^{2+}) into pyramidal neurons, a well-recognized and highly potent trigger for plastic events that potentiate synaptic sensitivity (Sejnowski & Destexhe, 2000). The fast influx of Ca^{2+} triggers an upregulation of the calcium-calmodulin-dependent protein kinase II, leading to phosphorylation of new post-synaptic plasticity-related protein receptors. Consequently, glutamatergic transmission is enhanced, leading to increased excitability within that circuit, and thus, long-term potentiation (LTP; Walker, 2005; Walker & Stickgold, 2006). LTP is an electrophysiologic neuronal marker for memory and learning. Thus, it appears that spindles may also index sleep–related memory and learning capacities.

Consistent with the plasticity-related effects of spindling activity on neocortical neuronal networks are reports of strong associations between spindling activity and memory and learning performance in both humans and nonhuman animals. Building on both the physiologic and behavioral findings, Fogel et al. (2007) have argued that spindling activity indexes an individual's overall memory or general learning capacity. A reduction in sleep spindling activity is, in fact, associated with a variety of neurodegenerative disorders including Alzheimer's disease, Creutzfeldt–Jacob disease, progressive supranuclear palsy, and dementia with Lewy bodies (Clawson et al., 2016).

Topographic EEG (electroencephalogram) analysis of N2 electrophysiologic events in humans reveals two different spindle types: a slower type that typically manifests over centro-frontal regions and a faster type that typically manifests over parietal regions (Table 4.1). The two types of spindles are differentially affected by aging, sleep deprivation, and pharmacologic agents, with the frontal type more strongly affected by sleep deprivation, aging, and dopaminergic agents than the parietal type and more strongly related to declarative and verbal learning materials than the parietal type. Given the sleep protective and plasticity-related effects associated with N2 sleep spindles, it is certain that sleep spindles will receive intensive investigation by sleep scientists in the near future.

After spending several minutes in N2 light sleep, the wayfarer then descends into the promised oblivion of deep sleep wherein "the death of each day's life . . . knits up the ravell'd sleave of care" (Shakespeare, Macbeth, scene 2, act 2). Now the EEG shows the characteristic delta waves of slow wave sleep in the band of 0.5–4.5 Hz. Now it becomes almost impossible to arouse the individual who is for all intents and purposes dead to the world.

If the individual does begin to arouse without first passing back up the three stages of sleep in reverse order (N3 to N2 to N1, etc.), you will see the strangest parasomnias emerge. For example, the individual may engage in

Table 4.1 Two spindle types

	Slow	Fast
Frequency	12 Hz	14 Hz
Topographic location	frontal	parietal
Neuropharmacology	dopaminergic	serotoninergic
Sleep deprivation	very sensitive	less sensitive
Aging	very sensitive	not affected
Cognitive correlates	effortful processing, social cognition, motor and verbal learning, declarative memory	general mental ability, performance IQ

complex motor and goal directed behaviors despite being completely unconscious. Somnambulism (sleep walking), somniloquoy (sleep talking), night terrors, sleep paralysis, and "sleep sex" all might occur if the individual partially arouses toward awakening or REM without first passing through N3, N2, and N1. In sleep walking, the individual may navigate through all kinds of barriers with apparently normal eyesight even though the brain still records slow wave activity. Some individuals go to the kitchen and eat huge amounts of food and then later, when fully awake, are puzzled by the bloated feeling they experience. While sleep talking, individuals can carry on long conversations with an invisible interlocutor. Night terrors typically occur in children who sit up on the side of the bed and scream bloody terror. Even though the child's eyes are open wide they appear unable to see you, as their brain is still in slow wave sleep. In sleep sex, the individual attempts to initiate sex with a bed partner even though both he and his partner are asleep.

4.2.1 Brain Mechanisms in NREM

NREM is initiated after adenosine levels in the basal forebrain activate GABAergic efferents that project to cortical sites that ultimately dampen activation levels in these cortical sites. In addition, thalamic efferents to cortical sites begin to fire rhythmically until they entrain cortical neurons, ultimately resulting in slow rolling waves across the cortex during N3 SWS. Most areas of the brain begin to relax metabolic activity at this point, and blood flow decreases slightly relative to waking. During N3 slow wave activity (SWA) predominates over frontal areas (Finelli et al., 2001) and travel (propagate) backward to posterior

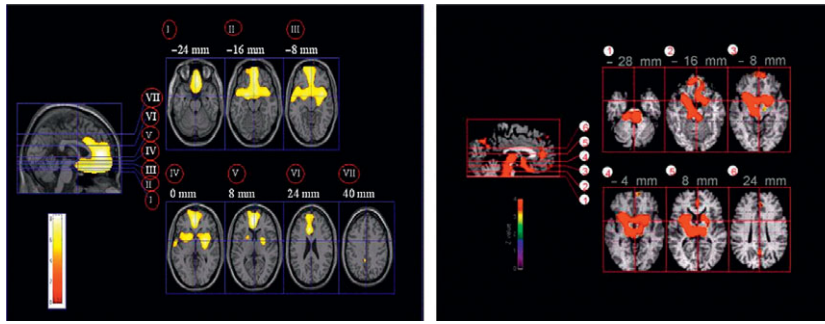

Figure 4.2 Functional neuroanatomy of normal human non-REM sleep, assessed by H2 15O PET ((Left panel) Brain areas in which regional cerebral blood flow (rCBF) decreases as a function of delta power during non-REM sleep (stages 2–3). Image sections are displayed on different levels of the z axis as indicated on the top of each picture. The color scale indicates the range of Z values for the activated voxels. Displayed voxels are significant at $P < 0.05$ after correction for multiple comparisons. (Right panel) Brain areas in which rCBF decreases during non-REM sleep as compared to wakefulness and REM sleep. Note the similarity of the regional blood flow distribution between left and right panels).
Used with permission from Dang-Vu et al. (2007)

areas of the brain. Beyond this variability, slow waves seem to systematically recruit various brain regions. The power density of delta waves (1–4 Hz) during NREM sleep is negatively correlated with cerebral blood flow in the network of the social brain – the ventromedial prefrontal cortex, the basal forebrain, the striatum, the anterior insula, and the precuneus (Dang-Vu et al., 2005). Subcortically, slow waves originate in the insula and cingulate gyrus, and propagate up through the precuneus, the posterior cingulate, ventrolateral, and medial frontal areas. Why these brain regions? Interestingly, all of these regions are associated with the network of structures that have been designated the "social brain network." Note that not all the brain regions implicated in NREM sleep overlap with or are included in the social brain network. But the interesting fact is that most are included. This network, then, is preferentially and slowly taken offline (decreases in activation) during NREM sleep phases (Figure 4.2). We will see when we discuss REM that this same network is then reconnected and taken back online during REM phases.

4.2.2 Slow Wave Sleep and Growth Hormone

Slow wave sleep of NREM is associated with a major surge in growth hormone (GH) release (Steiger, 2003) – at least during the first N3 period in

adults. It may persist beyond the first N3 period in children. When growth hormone releasing hormone (GHRH) levels rise in blood, they stimulate onset of NREM sleep. Conversely, somatostatin (SS) inhibits NREM while enhancing REM. SS also interacts with GH. Physiologic and growth-promoting effects of GH in the rat and in the human depend on pulsatile release of GH. But pulsatile release of GH, in turn, depends on SS activity. SS is released in a sinusoidal pattern. When GHRH is released during a trough period of SS release, it induces pulsatile release of GH, while a rise in SS release reduces GH release to baseline levels, thus allowing a new cycle of GH-SS interactions to begin. Fluctuating levels of SS release are, therefore, required to sustain pulsatile release of GH, which in turn influences NREM onset.

4.2.3 Slow Wave Activity and Memory

A number of important studies in the 1990s and early 2000s (reviewed in Walker & Stickgold, 2004) demonstrated that SWS was crucially important for acquisition and consolidation of certain types of new memories into long-term memory stores. For example, Plihal and Born (1997) demonstrated that learning of paired associates (word pairs like cat-dog that are semantically related to each other) and visual-imaginal, mental rotation tasks are dependent on obtaining a bout of NREM (early sleep) rather than REM (late morning sleep) after learning the task.

In addition to the role that whole bouts of NREM play in facilitation of memory processing, specific electrophysiologic events within NREM are also associated with memory and learning. For example, Huber, Ghilardi, Massimini, and Tononi (2004) showed that localized increases in SWA within NREM is associated with improved performance on tasks mediated by the frontal lobes. These sorts of findings imply that sleep homeostasis has a local component, which can be triggered by a learning task involving frontal brain regions. Interestingly, the local increase in SWA over frontal regions after learning correlates with improved performance of the task after N3 sleep. Benington and Frank (2003) point out that the activation of T-type calcium ($Ca2^{+}$) channels in non-REM sleep may promote either long-term neuronal depression or long-term potentiation, which are both processes that have been linked to hippocampal memory functions. Sleep-associated consolidation of information gathered during the wake state appears to depend on hippocampal-cortical interactions that occur during both NREM SWS and REM and involve some sort of replay of learned associations acquired while awake during REM sleep. Wilson and McNaughton (1994), for example, showed that hippocampal cells that are active when rats learn a new maze are also active during subsequent sleep.

4.2.4 Special Role of N3 SWS Sleep in Children

Given the cumulating evidence that N3 appears to facilitate learning and memory functions, one might expect that SWS plays a critical role in boosting brain plasticity or learning during childhood development. The percentage of total sleep that N3 SWS composes is very high in the developmental years of children and it gradually declines as a percentage of total sleep over the adult years, until it disappears almost entirely in old age. This developmental profile of N3 is prima facie evidence that it is important for development and especially important for brain development, given the disproportionate amount of metabolic effort that is funneled into brain development in humans. Anderson and Horne (2003) have demonstrated that N3 sleep deprivation differentially impairs frontal lobe functions. It is difficult to selectively deprive people of N3 SWS sleep, as when we do so, we inevitably also disrupt REM sleep that occurs downstream from N3 in the nightly sleep cycle. You can partially selectively deprive people of N3 by sounding a tone loud enough to disrupt N3 but not so loud that subsequent REM is disrupted. Recovery sleep from this sort of selective N3 deprivation involved making up N3 sleep first and then some REM. There are proportionally greater amounts of recovered N3 in the first few minutes and hours of N3 recovery than in the later minutes and hours of N3 recovery sleep. The fact that you can selectively deprive people of some portion of their SWS without dramatically altering REM increases our confidence in Anderson and Horne's results concerning the effects of N3 deprivation on frontal lobe functions. Frontal lobe functions include things like working memory, attention, language fluency, planning, executive control, self-regulation, and personal reflection and insight. Obtaining enough SWS during childhood may be critical for normal acquisition of these frontal lobe related skills.

4.2.5 Bodily Changes during NREM

To fully understand NREM phenomena we need to briefly touch on its ancillary effects on the body. During NREM the parasympathetic subbranch of the autonomic nervous system is more active than the sympathetic nervous system. Heart rate, blood pressure, temperature, and metabolic rate all decrease slightly from daytime baseline levels during NREM. Infectious challenges, such as getting the flu or some other infection, can cause an increase in N3 sleep duration as well as delta wave activity. Sleep deprivation, on the other hand, increases vulnerability to infection. These latter facts will be discussed more fully in the next section, but they point to a crucial function for NREM – namely immune system maintenance.

4.2.6 Fatal Familial Insomnia (FFI)

Knowledge concerning NREM related sleep disorders will help fill out the picture concerning NREM characteristics. Both NREM parasomnias and NREM-related sleep disorders, such as sleep terrors, will be discussed shortly. Here, I want to briefly discuss an extremely important NREM disorder that appears to underline the fact that if you go without sleep (at least NREM sleep) for too long, you will most certainly die. Fatal familial insomnia is an extremely rare autosomal-dominant hereditary disease caused by a mutation (missense coding at codon 178) of the prion protein gene (PRNP) (Lugaresi et al., 1986). Current knowledge suggests that only a few hundred families/patients in the world have the disease. It is characterized by loss of NREM sleep and very probably loss of some REM as well, though patients appear to be enacting REM dreams. Onset is typically in middle age and patients die typically within a year of diagnosis. After disease onset there is a progressive insomnia, somnolence (dreamy sleepiness), a somewhat apathetic stupor, motor activation (possibly REM dream enactment behaviors), and recurrent stereotypical behaviors resembling daily activities like hair combing, greeting nonexistent persons, handling objects, etc. When patients are asked about these behaviors they say they were dreaming them. After disease diagnosis, polysomonographic studies reveal fragmentation and progressive reductions in NREM spindle and delta activity until they disappear completely; then stage N1 and short bursts of REM appear sporadically, lasting only thirty to forty seconds, and are the only signs of sleep that occur for the final weeks or months of the patient's life. Postmortem analyses of the brains of FFI patients reveal degeneration and loss of cells in the thalamus and mesio-orbital areas of the frontal lobes. This terrible disease once again underlines the apparent functional connections between NREM sleep and the frontal lobes in humans.

Having completed our review of NREM sleep characteristics, we turn next to REM sleep. We will see that REM sleep is in many ways an opponent process to NREM. While the parasympathetic system is activated in NREM, the sympathetic system is activated in REM. While mesio-prefrontal systems are taken offline during NREM, they undergo gradual activation in REM. While metabolic, cardiovascular, and sexual systems are all stable in NREM, they are all unstable and fluctuating in REM. While we dream of everyday friendly interactions in NREM, we dream of aggressive and bizarre interactions in REM.

4.3 Characteristics of REM

4.3.1 The Biobehavioral Characteristics of REM

We have already discussed the peculiar and fascinating behavioral characteristics of REM. Here, we go into more of the neurobiologic detail. REM sleep

accounts for about 22 percent of total sleep time in humans. It is the form of sleep associated with vivid dreaming and rapid eye movements under the closed eyelids – hence the name. Brain activation levels within the limbic system (the emotional brain) can be higher than they are in waking state. REM is composed of tonic and phasic events. Tonic aspects of REM are the processes that occur more or less constantly during REM, and phasic aspects of REM occur intermittently. REM's tonic characteristics are a desynchronized electroencephalogram (EEG), sexual activation (penile erections/clitoral engorgement), and atonia of the antigravity muscles. Its phasic characteristics include bursts of rapid eye movements under the closed eyelids, myoclonic twitches of the facial and limb muscle groups, and increased variability in heart rate, respiration, blood pressure, and autonomic nervous system discharges. The phasic aspects of REM tend to occur in association with bursts of pontine-geniculo-occipital (PGO) waves. These PGO waves are essentially electrical spikes in visual centers of the brain. In addition, some mammals also exhibit a theta rhythm in the hippocampal formation during REM. The hippocampus is an important structure for formation of memories. REM is also associated with autonomic nervous system instabilities that become more extreme as the duration of REM episodes increases across the night. Like NREM sleep, REM deprivation results in a rebound phenomenon indicating that a certain amount of REM is required and must be made up if lost. Interestingly, after total sleep deprivation, NREM sleep is made up before that of REM.

4.3.2 REM On and REM Off Cellular Networks

REM is initiated via action of REM-on cells near the ventrolateral preoptic nucleus of the hypothalamus. Neurons in the extended VLPO area promote REM via gabaergic inhibition of nearby hypothalamic and thalamic arousal systems. In addition, cholinergic neurons originating within the laterodorsal tegmental (LDT) and pedunculopontine tegmental (PPT) nuclei (LDT/PPT) initiates REM once inhibition is lifted from these neurons. That inhibition is coming from noradrenergic (NA) and serotonergic (5HT) neurons in the locus ceruleus and dorsal raphe nucleus (DRN/LC), respectively. Activation of REM occurs after removal of inhibition exerted by these aminergic efferents on cholinergic cells in the LDT/PPT. The release of acetylcholine (Ach) from terminals of LDT/PPT cells triggers the onset of REM. As REM proceeds, cholinergic excitatory effects trigger activation of brain regions that control various components of REM, including brainstem sites, hypothalamus, limbic, amygdala, and the basal forebrain. When these activation levels reach a threshold, their continuing firing results in a feedback inhibition on REM-on cells of the LDT/PPT, thus ending the REM period. In sum, REM expression is regulated by antagonistic

cellular groups, with aminergic cell groups inhibiting expression of REM, and cholinergic groups promoting expression of REM. When cholinergic REM-on cells are activated, aminergic REM-off cell groups are inhibited and vice versa.

4.3.3 Brain Mechanisms in REM

Recently a number of positron emission tomography (PET) and functional Magnetic Resonance Imaging (fMRI) studies (see Figure 4.3) of the sleeping brain have revealed that REM (relative to the waking state) is associated with high activation in extra-striate visual regions, limbic, limbic striatum, paralimbic, anterior insula, Brodmann's Area 10 in prefrontal cortex, the ventromedial prefrontal cortex, and temporal regions, and relative hypoactivation in inferior and middle frontal gyrus and inferior parietal regions (reviews in Maquet et al., 2005; Dang Vu et al., 2010). In addition, the motor and premotor cortices are also very active during REMS (Maquet et al., 2004). Interestingly, the superior frontal gyrus, the medial frontal areas, the intraparietal sulcus, and the superior parietal cortex are not less active in REMS than during wakefulness (Maquet et al., 2005). In addition, hippocampal outflow to the cortex is blocked during REM. Instead, the hippocampus receives information from cortical networks.

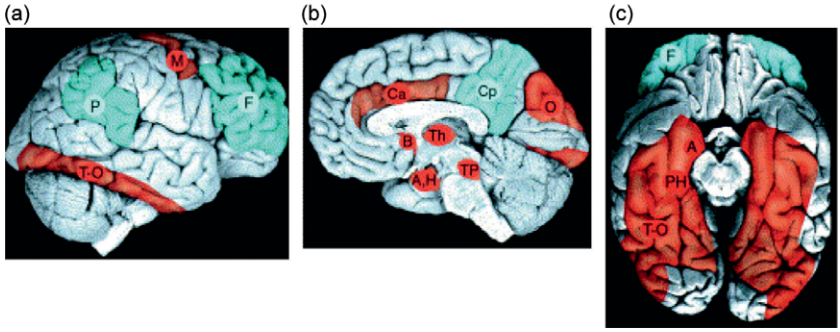

Figure 4.3 Functional neuroanatomy of normal human REM sleep, integrating data from PET and fMRI

(Regions colored in red are those in which there is a relative increase in neural activity associated with REM sleep; those in blue correspond to relative decreases in neural activity associated with REM sleep. (a) lateral view; (b) medial view; (c) ventral view. A, amygdala; B, basal forebrain; Ca, Anterior cingulate gyrus; Cp, posterior cingulate gyrus and precuneus; F, prefrontal cortex (middle, inferior, and orbitofrontal cortices); H, hypothalamus; M, motor cortex; P, parietal cortex (inferior parietal lobule); PH, parahippocampical gyrus; O, occipital-lateral cortex; Th, thalamus; T-O, temporo-occipital extrastriate cortex; TP, pontine tegmentum).

Used with permission from Dang-Vu et al. (2007)

Some authors (Domhoff, 2011; Pace-Schott & Picchioni, 2017) have noted that the collection of structures activated and deactivated during REM overlap to a significant extent with the so-called default mode network (DMN). The DMN includes posterior cingulate, precuneus, retrosplenial cortex, inferior parietal, superior temporal, hippocampal formation, and medial prefrontal cortex. These brain regions, collectively, are always active when the individual is at rest and simply thinking, letting his/her mind wander, or daydreaming. Pace-Schott and Picchioni argue that the DMN is composed of at least two major subsystems: one centered on the medial temporal lobe and the hippocampus (the simulation system) and the other centered on the medial prefrontal cortex (the self-referential system). The simulation system is operative when we imagine future or past states of affairs while the self-referential system computes or handles information about the self, including, of course, emotional information. Both are operative during dreaming. In NREM, slow wave sleep brain nodes in the DMN are disconnected, but in REM they transiently reconnect with the simulation system, displaying more stability during REM than the self-referential system. In short, the anterior portions of the DMN are reconnected during REM, but these structures, in turn, are part of the larger social brain network discussed in Chapter 1. REM appears to involve the reconnection of the interrelated set of structures that make up the social brain network after it is disassembled or disconnected during the NREM phases of sleep. We will return to the issue of the activation of the social brain network during dreaming shortly. First, we need to continue our survey of REM phenomenon/characteristics.

4.3.4 REM and Motivational Reward

Perogamvrosa and Schwartz (2012) present what they call their "Reward Activation Model" (RAM) of sleep and dreams. The authors integrate recent neurophysiological, neuroimaging, and clinical findings that point to significant activation of the mesolimbic dopaminergic (ML-DA) reward system during both NREM (N2 in humans, SWS in rats) and REM sleep. With regard to REM, dopamine bursting activity within the ventral tegmental area is elevated during REM. This is significant because it is precisely what occurs in the waking brain when it is processing stimuli, particularly social stimuli, which carries reward value.

4.3.5 REM-Related Physiologic Phenomena

REM is associated with very unusual physiologic and cognitive phenomena. In REM you get a very highly activated brain, but the body is paralyzed; the

autonomic nervous system erupts in periodic discharges or storms; thermoregulatory reflexes are reduced or absent, but the sexual system is activated and of course, you get intense dreams. Needless to say, it is not at all clear why Mother Nature would every ninety minutes or so during sleep, intensely activate your brain and your sexual system, paralyze your body, and force you to watch these things we call dreams! Let us discuss a bit more fully some of these strange characteristics of REM.

4.3.6 REM Dream Content

People dream in both REM and NREM sleep states, but some scientists believe that the dreams associated with REM awakenings are very different in type from dreams associated with the other sleep states. REM dreams tend to be more intense, more storylike, more aggressive, more emotional, and contain vivid visual detail, unpleasant emotions, and occasional bizarre and improbable events. Some dream researchers believe that most of these differences disappear when you control for number of words per dream as well as time of the night when a dream occurs. As morning draws near, the body's arousal systems begin to activate, and so dreams are more vivid when they occur toward morning. While the debate is not yet closed on whether the differences between REM and NREM are real, the bulk of the evidence appears to be consistent with the claim that the REM–NREM dream content differences are marked, stable, dramatic, and real.

4.3.7 Conclusions: REM versus NREM Sleep

It is difficult to imagine what the function of REM sleep might be, given the odd constellation of traits we have just summarized. Its biobehavioral characteristics are paradoxical in that its physiologic correlates appear to be injurious to the health of the organism, while its brain correlates suggest social-emotional functions. Unlike REM, NREM biobehavioral characteristics are slightly less paradoxical but still enigmatic. NREM's physiologic functions may be related to immune system function, while its electrophysiologic properties are clearly related to the restorative functions of sleep. Both REM and NREM sleep likely participate in memory processing but so does the waking state. The fact that NREM appears to be associated with the gradual deactivation of a select group of brain structures that are then reactivated during REM suggests that the two sleep states either work in harmony with one another to maintain optimal brain function or that NREM undoes something that REM instantiates. To understand the peculiar functions of REM or NREM and whether they operate in harmony or in opposition to one another,

we will need to assemble one more set of facts concerning disorders associated with REM and NREM sleep, and it is those facts we review next.

4.4 Review Questions

- What is the significance of the two major spindle types associated with stage N2 NREM sleep?
- What is the Reward Activation Model (RAM) of REM sleep and why is it significant?
- What is the motor paralysis associated with REM and why is it significant? Why do you think paralysis does not occur with NREM?
- What is the significance of the sexual activation that occurs in REM?

Further Reading

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CHAPTER FIVE

Sleep Disorders

Learning Objectives

- Describe the two main families of sleep disorders
- Describe the major dysommnias
- Describe the major parasomnias
- Describe the significance of brain state transition failures in parasomnias

5.1 Introduction

Forty million Americans are afflicted with chronic disorders of sleep. Sleep disorders cause 38,000 cardiovascular deaths and cost over $16 billion annually. Indirect costs of accidents, property destruction, litigation, hospitalization, and death add another $50–$100 billion. Worldwide, about 10 percent of the population meet diagnostic criteria for insomnia disorder. Obstructive sleep apnea (OSA) is becoming more ubiquitous as the obesity epidemic enhances risk for OSA. Recurrent episodes of not breathing (apnea) or reduced airflow (hypopnea) during sleep leads to an almost constant daytime state of a brain starved for oxygen. It is likely that some 10 percent of the general population has OSA but that is probably a low estimate given that OSA is likely severely underdiagnosed, particularly among African Americans, overweight individuals, and older adults. Sleep disorders also carry huge consequences for mental health. For example, 65–90 percent of adults with major depressive disorder (MDD) report sleep problems, and 90 percent of children with depression report disturbed sleep. It is not just that sleep problems result from mental health problems, sleep problems can precipitate or help cause mental problems.

When we study sleep disorders, we need to keep in mind the socio-ecological framework we have adopted to understand sleep more broadly. At the individual level, it is now clear that higher levels of education, income, and exercise, are generally positively associated with better sleep.

At the interpersonal level, secure attachment orientations within relationships predict better sleep. Supportive relationships and social networks, more generally, are associated with better sleep and can cushion negative effects of sleep disorders. At the ecological level, climate, buildings, roads, traffic

patterns, trees, and ambient environments (e.g., noise, temperature, light pollution) can affect sleep characteristics. Neighborhood social environment refers to factors such as social cohesion, safety, exposure to crime, and socioeconomic climate. All of these factors at the individual, interpersonal, social, and ecological levels impact the appearance, severity, and prospects of recovery from sleep disorders.

Sleep disorders can be divided into two very broad classes: dyssomnias and parasomnias. Dyssomnias involve changes in sleep duration such that the patient gets too much or too little sleep. Parasomnias involve partial arousals from within a REM or NREM sleep state. A third class of sleep-related disorders involves changes in the circadian pacemaker system such that the daily sleep period is displaced (delayed or advanced) from its normal slot within the twenty-four-hour day. Since I discussed circadian-related sleep disorders in a separate chapter, I confine discussion here to dyssomnias and parasomnias.

Dyssomnias involve a change in sleep amount from normal reference values. Hypersomnolence is too much sleep, and insomnia is too little sleep. Insomnia and excessive daytime sleepiness are, in fact, the most common disorders of sleep. Changes in sleep duration, furthermore, are associated with significant risks to both physical and mental health. Persons with longer REM sleep durations (relative to the population norm), for example, experience greater risks for various medical conditions (cardiovascular disease, obesity, etc.) and mortality. They are also at greater risk for depression. Moreover, it has become increasingly clear in recent years that the restorative or homeostatic properties of sleep are dependent on an interaction between sleep amounts and sleep intensity parameters, as formalized in Borbeley's original two-process model of sleep regulation (Borbeley, 1982) and its more recent emendations (Achermann & Borbeley, 2003). When deprived of sleep, mammals typically exhibit a sleep rebound proportional to the amount of sleep lost, indicating that the amount of sleep, or some specific intensity component of sleep reflected in "amount of sleep," is physiologically obligatory. Thus, "sleep amount or duration" is a strictly regulated physiologic need for the organism and any deviation from the amount required must be considered a disorder. Too much or too little sleep will have dire consequences for the organism. Too little sleep is a more common complaint than too much, so we begin with a discussion of insomnia, the most common dyssomnia.

5.2 Dyssomnias

5.2.1 Insomnia

Insomnia is defined as difficulty initiating sleep or staying asleep or both. It is very common, with more than 95 percent of the population claiming to have

experienced it at least once in their lives. For the vast majority of these people, the sleeplessness lasts just a few nights. For about 10 percent of the population, however, the insomnia can be persistent, resulting in very significant hits to health and well-being. The daily fatigue and exhaustion that is associated with chronic insomnia undermines all other aspects of a person's life, making them irritable, unhappy, and vulnerable to all kinds of minor and major health complaints. Insomnia, in short, is a major public health crisis in the world today.

There are two forms of insomnia: primary and secondary insomnia. Primary insomnia is sleeplessness due to intrinsic sleep-related causes, while secondary insomnia is sleeplessness due to non-sleep-related causes such as anxiety, illness, stress, etc.

5.2.2 Primary Insomnia

The paradigm case of primary insomnia is fatal familial insomnia (FFI), a prion disease that damages the thalamic neurons that keep the brain awake. A "prion" is a tiny protein that gets misfolded and entangled up in the metabolic machinery of certain neurons that are important for maintaining wakefulness and sleep. Accumulation of enough of these proteins in the wrong places in neurons creates significant brain dysfunction. In FFI, the prion disease affects mostly those regions of the brain that inhibit the thalamic-alerting neurons. Thalamic alerting centers of the brain can no longer be effectively inhibited, and thus, the individual can no longer transition into non-alertness and sleep. FFI results in death in about eighteen months post diagnosis, but it is a very rare disease. After diagnosis, progression is rapid as sleeplessness persists. First there is inability to concentrate, then confusional states ensue, then panic attacks and hallucinations, and finally, weight loss. The individual fluctuates between stage N1 and REM in a kind of dreamy twilight state until death intervenes.

Aside from FFI, most sleep scientists are not in agreement as to whether there are any other intrinsic or primary insomnias. Interestingly, an individual can believe that they are not sleeping at all or sleeping well, but when we bring them into the sleep lab to test their sleep, it turns out that they are in fact sleeping perfectly well. These "insomnia" cases are sometimes called "sleep state misperception." About 4 percent of people who report insomnia are actually suffering from sleep state misperception. Most sleep clinicians believe that these individuals maintain a highly aroused baseline state of vigilance or anxiety, and therefore, their brain systems never fully relax into sleep, even though the EEG unmistakably demonstrates deep sleep.

5.2.3 Secondary Insomnia

Most cases of insomnia are in the secondary insomnia category; that is, the cause of the insomnia is usually not something intrinsic to the sleep-wake brain systems that normally regulate transitions into and out of sleep. In cases of secondary insomnia there are usually medical issues, emotional issues, or everyday stress or work-related issues that keep the person awake at night. Many medical conditions can impair sleep, such as chronic pain, restless legs syndrome, and drug side effects. In addition, medical conditions, such as hyperthyroidism (too much thyroid hormone) and hypercortisolemia (too much of the adrenal hormone cortisol), can lead to chronic hyper-arousal and therefore difficulty falling asleep or maintaining sleep. By far the most common secondary cause of insomnia is psychological or emotional problems. The everyday worries and chronic anxiety people experience on a daily basis is the enemy of deep restorative sleep.

The major psychiatric syndromes, such as major depression, schizophrenia, and bipolar disorder, are also associated with major disruptions of sleep including severe insomnia. During the manic phase of manic-depressive illness, for example, the individual can stay awake for sometimes days at a time before collapsing due to exhaustion. During active psychotic states associated with schizophrenia, individuals do not sleep normally, and during major depressive episodes, sleeplessness and early morning awakenings are common and sometimes used to diagnose depression.

5.2.4 Major Depression

Depression is almost always associated with complaints of insomnia – specifically early morning awakenings. Major depressive disorder is associated with a range of symptom clusters including the hallmark symptoms of persistent sadness and anxious and hopeless feelings. When sleep does occur, dreams are recalled as intensely unpleasant experiences with elevated levels of scenes of aggression by unknown strangers against the dreamer/self. Reduced REM sleep latency and increased REM density and REM time are commonly observed in depressed patients. REM sleep deprivation can temporarily alleviate depressive symptoms (Vogel et al., 1975). Most antidepressant drugs reduce REM sleep and degree of REM suppression is correlated with degree of symptomatic relief in responders. Thus, signs of enhanced REM sleep pressure in some depressed patients may reflect a primary symptom of the disorder rather than a mechanism compensating for the affective disturbance. Depressed people may use their dreams to work through difficult emotions (Cartwright, 1999)

5.2.5 Sleep Apnea

Sleep apnea is a very common disorder that prevents sleep because the individual's airway is blocked during sleep. About 24 percent of men and 9 percent of women develop sleep apnea, particularly as they age. Sleep apnea is responsible for 50,000 premature and preventable deaths each year from accidents, heart attacks, and strokes. The airway in a patient with sleep apnea is blocked, often because there is simply too much fat in the neck, so the collapse of the airway during sleep cuts off airflow to the lungs, and the individual who suddenly cannot breathe awakens briefly to catch his breath. This brief arousal or apnea episode stimulates a return to wakefulness. These brief arousals happen hundreds of times during the night, and thus, the individual's sleep architecture becomes extremely fragmented. Loud snoring usually accompanies the sleep of the patient with sleep apnea because they cannot breathe normally through the mouth. In the morning, all the individual knows is that he does not feel fully rested, and during the day he is fatigued and very sleepy.

There are two types of sleep apnea: central sleep apnea and obstructive sleep apnea (OSA). Central sleep apnea occurs when the problem is located within the central nervous system such that the respiratory muscles do not respond normally. The most common cause of sleep apnea, however, is obstruction of the airflow. The barriers to airflow are the soft tissues of the mouth, nasal cavities, and throat. These tissues can begin to occlude the airway if the musculature of the jaw and chin is such that it promotes occlusion. By far the most common cause of occlusion is that fat deposits begin to build up in the soft tissues of the mouth, nasal cavities, and throat, thus making them more prone to closure. Obesity is one of the strongest predictors of OSA.

The severity of an individual's OSA can be quantified by an apnea hypopnea index (AHI) – the number of apneas and hypopneas terminating in arousals across the night divided by the number of hours of sleep. Mild OSA is defined as an AHI between 5 and 15; moderate, between 15 and 30; severe, between 30 to 45; and extremely severe, above 45.

The most common treatment for OSA is continuous positive airway pressure or CPAP. When the patient goes to bed, he or she wears a face mask that directs positive pressure airflow into the nose. This positive pressure prevents collapse of the air passageway leading up to the trachea and allows normal breathing to continue during sleep. Most people, however, do not enjoy sleeping with a mask on, and the airflow can dry out the nasal passages.

We have now discussed the major sleep disorders involving too little or loss of sleep. Disorders of hypersomnolence or too much sleep include narcolepsy and Kleine-Levin syndrome.

Hypersomnia is excessive daytime sleepiness not attributed to lack of sleep or transient disruptions of sleep that often occur in each of us. It is a sleepiness that has more unusual causes. People with hypersomnolence have to fight or work hard during the day just to stay awake. Hypersomnolence can often be attributed to depression. When depression is the root cause of the hypersomonolence, placing the patient on antidepressants typically solves both the mood disorder and the hypersomnolence. Often daytime sleepiness can be attributed to an underlying sleep apnea. When the sleep apnea is appropriately diagnosed and treated, the daytime sleepiness lifts. If we rule out all of these potential causes of excessive daytime sleepiness – depression, sleep apnea, commonplace and transient sleep disruptions, etc., and if we rule out other well-studied hypersomnias like narcolepsy and Kleine-Levin syndrome, then we are left with "idiopathic hypersomnia" or excessive daytime sleepiness of unknown origin. Idiopathic hypersomnias can be effectively treated even though we do not know the causes. Treatment typically involves stimulant medications.

Kleine-Levin Syndrome is a periodic hypersomnia characterized by recurrent episodes of prolonged sleeping or hypersomnia and other behavioral and cognitive symptoms. It mainly affects teenage boys who display the pattern of sleeping most of the day and night for weeks at a time. The International Classification of Sleep Disorders diagnostic criteria for KLS include: (1) episodes of excessive sleepiness lasting more than two days and less than four weeks, occurring at least once a year; (2) episodes intermixed with long intervals of normal alertness, mood, cognition, and behavior lasting usually months to years; (3) episodes recurring at least every year interspersed with long periods of normal sleep; (4) episodes not better explained by a sleep disorder, a neurological disorder (e.g., idiopathic recurrent stupor, epilepsy), a mental disorder (e.g., bipolar disorder, psychiatric hypersomnia, depression), or the use of drugs (e.g., benzodiazepines, alcohol). In addition to these recurrent episodes of hypersomnia, KLS patients should experience at least one of the following symptoms: hyperphagia (overeating), hypersexuality, odd behavior, or cognitive disturbances (e.g., confusion, feeling of derealization, or hallucinations). Arnulf et al. (2005) reviewed the literature on 108 cases and found that many reported marked apathy, exhaustion, memory problems, temporal disorientation, derealization, dreamy state, and impaired speech, among other cognitive and perceptual symptoms.

5.2.6 Narcolepsy

Narcolepsy is another major disorder where affected individuals complain of too much sleepiness. It is characterized by (1) excessive daytime sleepiness; (2) hypnogogic hallucinations (vivid images at sleep onset); (3) "sleep attacks" or

sudden paralysis (cataplexy) following a strong emotional stimulus like laughing or an intense emotion; and (4) sleep paralysis or paralysis during the transition from sleep to wake or from wake to sleep. In the sleep to wake transition, the paralysis normally associated with REM sleep has not yet ceased, even though the patient is conscious or awake. Affected individuals may also exhibit reduced hypocretin in the cerebrospinal fluid, and sleep-onset REM (SOREM) on the EEG. Recall that the normal pattern is to enter sleep via NREM. SOREM indicate that the individual skips the NREM sleep stages and goes right into REM while falling asleep. Hypocretin (sometimes also called orexin) is a neuromodulator that is a peptide manufactured in the hypothalamus. Hypocretin receptors in the thalamus appear to be crucially important for activation of the awakening circuit centered on the thalamus. They are partially destroyed in narcolepsy, but it is unclear what agent or process destroys these orexinergic cells.

There is some evidence to suggest that narcolepsy may be an autoimmune disorder wherein the immune system selectively attacks certain neurons in the brain or cells in the body. In the case of narcolepsy, post-mortem examination of the brain of narcoleptics revealed damage to orexin/hypocretin-producing neurons in the hypothalamus. Genetic studies of narcoleptic individuals have revealed that narcoleptics often (though not always) share an HLA gene variant called DQB1*0602.

Let us examine the symptomology associated with narcolepsy a little more closely, as it teaches us that components of REM sleep may be affecting patient's waking behaviors. With respect to excessive daytime sleepiness, narcoleptics fall asleep several times a day. The "sleep attack" is usually for a few minutes at a time, but it can occasionally last up to an hour or two. After these "naps" or sleep attacks, the feeling of excessive sleepiness subsides for a few hours but then returns. Patients also experience microsleeps when they transiently fall into a sleep state, but it is brief and the patient is generally unaware of it. Patients can generally sense when they have a "sleep attack" coming. They feel a build-up of irresistible sleepiness and then sleep suddenly overcomes them. The cataplexy symptom involves sudden paralysis during wakefulness, usually triggered by strong emotions like laughter, surprise, or fear. They can occur during other experiences involving strong emotions such as watching a movie, having sex, or exercising. Cataplexy does not occur in all narcoleptics, but when it does it typically looks like seizure activity in that it comes on fast and the patient becomes immobilized and loses consciousness. In an attack, the patient feels a sudden weakening in the knees, the sagging of facial muscles, their head dropping forward, and arms flopping to the sides. These are all signs associated with the normal paralysis associated with REM sleep. It seems as if the patient is experiencing an irruption of REM physiology

into waking life. Consistent with this thesis is the fact that patients also report an intense dream-like state during sleep attacks, as if they are dreaming. When studied in a sleep lab, most narcoleptics demonstrate signs of REM pressure. Latency to REM is very short, and patients report that they are immediately immersed in vivid dreams after they fall asleep. When vivid dreams and images occur in the transition from waking to sleep, these dreams are called hypnogogic hallucinations. When they occur in the transition from sleep to wakefulness, they are called hypnopompic hallucinations. When muscle paralysis occurs in one of these transitional states, we call it sleep paralysis because the mind is awake but the body remains in REM paralysis. Thus, the patient is awake and aware that he or she cannot move. They may see or hallucinate some residual dream imagery – typically frightening imagery. There is a sense of threat and of an evil presence in the room – yet the patient cannot move to protect him or herself.

About one quarter of narcoleptics experience the classic tetrad of symptoms: excessive daytime sleepiness, cataplexy, hypnogogic or hypnopompic hallucinations, and sleep paralysis. The majority of narcoleptics experience at least two of these symptoms. The multiple sleep latency test (MSLT) assesses the extent to which an individual when given the chance, transitions into sleep quickly or slowly. The test also assesses whether the first sleep state the patient enters is the normal entry via NREM sleep or the abnormal entryway via REM. The MSLT can facilitate (though not definitively) diagnosis of narcolepsy if the patient falls asleep within eight minutes and preferentially enters sleep via REM. This profile is called sleep onset REM period or SOREMP. SOREMP is characteristic of narcolepsy, depression, and some other neuropsychiatric disorders.

Treatment for narcolepsy currently involves use of stimulants, such as Ritalin and Modafinil, to combat daytime sleepiness and clomipramine and imipramine (which historically were used as tricyclic antidepressants) to treat cataplexy.

5.3 Parasomnias

5.3.1 Introduction

Parasomnias are disruptions of behavior or consciousness during sleep. They typically occur in between brain states; for example, during the transition into or out of sleep, or in the transition between sleep states, such as between NREM and REM. They are divided into REM sleep parasomnias and NREM sleep parasomnias.

5.3.2 Non-Rem Parasomnias Sleepwalking

Somnambulism is the most common NREM parasomnia. Sleepwalking, which is most prevalent in children, is also seen in adults. It typically emerges when the individual transitions from N3 SWS to N2 light NREM sleep. About 20 percent of children experience at least one episode of sleepwalking up to about eleven or twelve years of age, and about 4 percent of adults occasionally sleepwalk. A family history of sleepwalking is the strongest predictor that an individual will sleepwalk. Thus, sleepwalking likely has a genetic basis, though no genetic profile has yet been found to characterize sleepwalkers. Sleepwalkers can accomplish very complex behavioral acts including preparing meals or searching for objects – even though they are only semiconscious and are likely hallucinating if their eyes are open. Efforts to wake them up frequently result in a state of agitated, confused arousal that might even involve aggressive or violent acts. There is typically disorientation such that the sleepwalker is confused as to where they are. In addition, there is typically no memory of the sleepwalking episode. Occasionally a sleepwalker develops a habit of getting out of bed, stumbling to the kitchen, and preparing and eating favorite dishes. This is called sleep-related eating disorder.

The EEG of sleepwalkers demonstrates unstable delta activity and frequent arousals during the early part of the sleep period, and it is out of these arousals that a sleepwalking episode begins. Sleepwalking can be treated effectively with clonazepam or alprazolam (Xanax).

5.3.3 Sleep Sex

As in the case of sleepwalking, sleep sex is a condition that involves sexual activity while asleep or in a confusional arousal out of NREM sleep that exhibits nonstable delta activity. Sleep sex can occur when the sleeper is alone or with a bedpartner. It can involve a very wide variety of sexual behaviors including masturbation, touching, fondling, sex talk, and outright intercourse. Interestingly, there is typically no kissing. Sometimes these sleep sex behaviors can involve overpowering the bed partner, and then it becomes dangerous. Accordingly, sleep sex is a clinically significant problem and potentially a legal issue. There is little reliable data on the disorder, probably because most people with the disorder are ashamed to talk about it. From what little we know, most patients (80 percent) with sleep sex disorder are men. Many also exhibit sleepwalking behaviors. The sleep sex is typically robotic because the initiator is asleep. If you wake him up he is confused and disoriented and sometimes

ashamed or angry. There is often amnesia for the act. This disorder is associated with major personal and sometimes legal consequences for the patient. Bedpartners of these patients are often perplexed and sometimes shocked by the patient's behaviors. When the sex is forced, of course, there are legal consequences. If the patient is in a new relationship, the bedpartner will be shocked by the unconsciously initiated and often aggressive sleep sex.

5.3.4 Night Terrors

Like sleepwalking and sleep sex, night terrors emerge when the patient (typically a child) is attempting to arouse out of N3 SWS. There is unstable delta activity and frequent arousals usually, in the first couple of sleep periods. The child sits upright and screams as if in terror. Night terrors run in families. Night terrors may not be recalled, however, and it is very difficult to awaken the child. Episodes can last between a few minutes and a half hour, and there is amnesia for the episode after awakening.

5.3.5 Sleep-Related Binge Eating

This involves an individual awaking from a NREM episode and then going to the kitchen in a somnambulant state and bingeing on some sweets or very rich foods. Most cases (75 percent) of sleep-related binge eating involve girls or women. Risk factors for development of this NREM disorder include a family history of sleepwalking or other sleep disorders and a history of eating disorders (e.g., anorexia or bulimia). Although it appears to be a NREM parasomnia, an arousal can begin in a NREM state but then switch into a REM state while the patient is bingeing. Level of consciousness can vary during a bingeing episode with some patients having no awareness and no memory of the episode and others being partially awake but feeling no control over the behaviors. Often the binge eating co-occurs with classic sleep walking disorder and when both disorders run in families. The binge eating episodes can occur regardless of whether or not the patient had a full meal before bedtime and regardless of hunger. In the morning, there is always a feeling of being bloated and not hungry. The foods the patients choose to binge on are typically high-carbohydrate foods mixed with very unusual items reflecting the clouded consciousness they operate under during an episode. For example, patients have reported eating raw meat and bacon, salt sandwiches, soap or hand cream, dog food, coffee grounds, ketchup, and mayonnaise. Patients may also go through elaborate food prep rituals and attempt to cook these items and then forget to turn off the stove. Other negative consequences of sleep-related

binge eating are not surprising: weight gain, increased risk of food poisoning, daytime appetite changes, and dental and other health complications.

5.3.6 Sleep Talking

Although the available data suggests that sleep talking can occur in any stage of sleep, it may be that like sleepwalking, sleep sex, sleep-related bingeing, and sleep terrors, sleep talking typically begins as a confusional arousal out of N3 slow wave sleep after some unusual delta activity and then proceeds to unfold in a hybrid or mixed form of REM sleep. About 50 percent of children sleep talk and some 4 percent of the adult population sleep talks on a consistent basis. Like the other NREM parasomnias, sleep talking runs in families and typically co-occurs with one or more of the other NREM parasomnias. The content of sleep talk is typically mundane. It most often involves interactions with hallucinated or dreamed interlocutors. The speech is most often linguistically well formed and grammatical, but its semantic content can often be meaningless. Bilinguals most often prefer to sleep talk in their native first learned tongues. Interestingly, when two sleep talkers sleep in the same bed they can often carry on largely meaningless conversations, all while they are asleep! As in the other NREM parasomnias there is typically amnesia for the sleep talking event in the morning. Although sleep talking is harmless in and of itself, it can sometimes weakly predict (in a middle-aged adult) later onset of a neurodegenerative disorder such as multiple system atrophy.

5.3.7 "Exploding Head Syndrome"

This is a real disorder! It is characterized by the sense of a flashbulb sound or explosion going off inside your head. Typically it occurs in the transition from waking into N1 sleep, and the explosion sound wakes you up. The explosion is usually experienced as deep within the center of the brain, but there is no pain. It lasts a second and disappears upon awakening. It typically occurs only a few times a lifetime in most people, but may be more common in the elderly. Aside from being startled, there are no apparent negative clinical effects of the experience. Nobody knows what causes this sleep-related experience.

5.3.8 REM Parasomnias

The major parasomnias of REM sleep are nightmares, REM behavior disorder (RBD), and isolated sleep paralysis.

5.3.9 Nightmares

The DSM-5 defines Nightmare Disorder (DSM-5 307.47 (F51.5)) as a parasomnia involving repeated awakenings from extremely frightening dreams that do not occur in the context of some other mental disorder. Upon awakening, the individual is oriented and alert and has clear recall of the content of the dream, which in turn is associated with clinically significant distress and impairment in daytime functioning. Similarly, the 2016/17 ICD-10-CM Diagnosis Code F51.5 defines nightmare disorder as a sleep disorder characterized by the repeated occurrence of frightening dreams that precipitate awakenings from sleep. Upon awakening, the individual becomes fully alert and oriented and has detailed recall of the nightmare, which usually involves imminent danger to the individual.

Nightmare disorder typically involves distressing nightmares that occur at least once a week for a month or more. Epidemiological studies indicate that 2–6 percent of the adult American population (about 15–20 million people) experience nightmares at least once a week. Between one half and two thirds of children experience recurrent nightmares. Recurrent nightmares in children significantly predict later adolescent and adult risk for psychosis. If we can effectively treat nightmares in children, we may be able to prevent later onset of psychosis in at least some of these kids. Indeed, experience of frequent nightmares in both children and adults is associated with a host of neuropathological and neuropsychiatric risk factors and disorders including anxiety, depression, stress, and suicidal ideation (Spoormaker, Schredl, & van den Bout, 2006). Severely distressing and repetitive nightmares are a hallmark of post-traumatic stress disorder (PTSD), REM behavior disorder (RBD), and several other chronic and disabling neuropsychiatric syndromes. Development of an effective treatment of nightmare disorder will improve treatment outcomes of nightmare-related distress in these other disorders as well.

5.3.10 REM Behavior Disorder

RBD is characterized by loss of the atonia normally associated with REM sleep. Patients therefore often act out dreams normally associated with REM sleep. They appear to experience visual and auditory hallucinations in association with violence-themed dream narratives. RBD may herald by decades the onset of one of the synucleinopathies such as Parkinson's disease (PD), Lewy body dementia (LBD), or multiple system atrophy (MSA). Dream enactment can last for minutes and be quite violent, with the individual physically doing battle with imaginary foes. These patients exhibit complex exploratory and often defensive motor activities during REM sleep, usually in association with

dreams. Common behaviors include screaming, punching, grasping, kicking, or jumping out of bed in pursuit of or in flight from a supernormal or monstrous foe. Fantini, Corona, Clerici, and Ferini-Strambi (2005) studied the REM sleep dreams of forty-nine patients with polysomnographic-confirmed RBD and seventy-one healthy controls. Compared to controls, patients with RBD reported a striking frequency of aggression wherein the dreamer was threatened or assaulted by monstrous beings or unfamiliar strangers with intent to harm the dreamer or his family. This signature RBD dream interaction was captured by various standardized Hall/Van de Castle dream content indicators, including a higher percentage of "dreams with at least one aggression" (66 percent vs. 15 percent); an increased aggression/friendliness interaction ratio (86 percent vs. 44 percent); and an increased aggressions/characters (A/C) ratio (0.81 vs. 0.12).

5.3.11 Sleep Paralysis

Isolated sleep paralysis (ISP) is a relatively common experience, typically characterized by an inability to move or speak after waking up as well by the eerie sense that someone or something who is somehow evil or malignant and threatening you is in the room with you. The individual/patient is really still in REM sleep, although he might believe he has fully awakened, or at least his mind is awake. It is legitimately categorized as a dream because the individual often experiences both auditory and visual hallucinations as well as the muscle atonia or paralysis that normally accompanies REM.

The experience is caused by fragmentation of the brain state associated with REM such that one does not move entirely out of REM when one transitions from REM to wake. The potential causes of the failure to completely transition out of REM into waking consciousness are multiple. They include stress, disease, injury, illness, chemical imbalances, and sleeplessness among many other causes. Cheyne (2002) of the University of Waterloo has collected some data on the phenomenology of these sleep paralysis dreams/visions. He used a survey to tabulate the frequency with which these various features occur in these experiences. Of 2,397 respondents, the mean age of onset was seventeen, but the experiences can happen at any age. About one quarter report experiencing sleep paralysis weekly or more frequently. Almost 80 percent experienced a sensed presence; that is, when they are awake but paralyzed they sense that someone or something is in the room with them and usually intends them harm. So the individual attempts to scream for help but cannot. Approximately 60 percent reported auditory and visual hallucinations, whereas only 41 percent reported tactile hallucinations. They hear the sensed presence breathing or moving toward them and can actually feel them touching or pressing in on

them. One-third (33.4 percent) of respondents were able to assign a sex to the sensed presence. Of these, most (82 percent) perceived the presence to be male, with women significantly more likely than men to see the sensed presence as male. Slightly more than half of the auditory hallucinations involved hearing voices. Approximately two thirds of respondents reported feelings of pressure. Almost as many reported feeling that they might die. Approximately 30 to 40 percent of respondents reported out-of-body experiences or OBEs, where they could see themselves from a point above their bodies, while only about 20 percent reported spinning sensations or autoscopy (seeing a double). Almost 100 percent (96 percent) of respondents reported fear. But substantial percentages also reported feeling erotic feelings and even bliss.

Parker and Blackmore (2002) used the Hall/Van de Castle dream content scoring system to quantitate the characteristics of ISPs that differentiate them from standard dreams. In ISP reports there were four times as many references to the body than in standard dream reports. In addition, there were higher levels of physical aggression against the individual than that found in run of the mill dreams. Unlike dreams that depict a variety of emotions, fear predominated in ISP reports. Parker and Blackmore found that the typical ISP

takes place in a familiar, indoor setting (the bedroom). Often a presence or person not known to the dreamer is present. The presence is more often male (if gender is reported), but most common of all is that a sexless "creature" or "form" is sensed. Interactions with the presence are predominantly aggressive "Dreamers" often feel victimized by these interactions. They also report a much greater awareness of their body, particularly torso, which is accompanied by increased reports of negative emotion (fear). They often struggle to overcome the situation (paralysis) and meet with equal degrees of success or failure (sometimes they are able to overcome the paralysis or not) (Parker & Blackmore, 2002, 57)

Box 5.1 Sleep and the law

Can someone perform all of the complex acts of killing another person while "asleep"? Rosalind Cartwright is a sleep scientist who has studied the issue and testified in court cases concerning sleepwalking and homicide. Cartwright says that in rare cases this is possible. In her 2010 book *The Twenty-Four-Hour Mind* (Cambridge University Press), she presents in detail the tragic case of Scott Falater murdering his wife of many years. A neighbor heard the cries of the wife and saw Falater pushing the body of the bloodied woman into the family pool. Falater had stabbed his wife dozens of times and then calmly left the scene of the murder (apparently to place the bloodied

Box 5.1 (cont.)

knife and clothes into a tupperware container). He then returned to the body to push it into the pool. After pushing the body into the pool, Falater apparently went back into the house. Cartwright thinks Falater went back into the house to go back to bed/sleep, just as most other sleepwalkers do unless they are awakened by a loud sound. Falater was convicted of murder despite the defense's claim that the defendant had been sleepwalking. Cartwright claims that a genuine sleepwalker who engages in complex criminal acts while asleep is utterly amnestic for the episode in the morning and attempts no cover-up of the deed. There is an individual and family history of sleepwalking, and then after the tragedy there is intense grief, remorse, and efforts to cooperate in the investigation. Siclari et al. (2010) suggest other factors that can be used to establish a sleep disorder as a causative factor in sleep-related violence. First, as Cartwright says, there should be medical evidence (e.g., from a polysomnographic sleep study) of an underlying sleep disorder as well as a family or individual history of sleep symptoms. Next, the violent act itself should conform to other independently established norms concerning sleep-related violent acts. For example, most such acts occur without apparent motivation and they are bitterly regretted upon awakening. There must also be precipitating factors to trigger a parasomnia, such as prior sleep deprivation, and thus, increased homeostatic drive (see summary).

Presence of an Underlying Sleep Disorder
Presence of solid evidence supporting the diagnosis
Previous occurrence of similar episodes

Characteristics of the Act
Occurs on awakening or immediately after falling asleep Abrupt onset and brief duration
Impulsive, senseless, without apparent motivation
Lack of awareness of individual during event
Victim: coincidentally present, possible arousal stimulus on return to consciousness
Perplexity, horror, no attempt to escape amnesia for event

Presence of Precipitating Factors
Attempts to awaken the subject
Intake of sedative/hypnotic drugs
Prior sleep deprivation
Alcohol or drug intoxication precludes the use of disorder of arousal in forensic cases

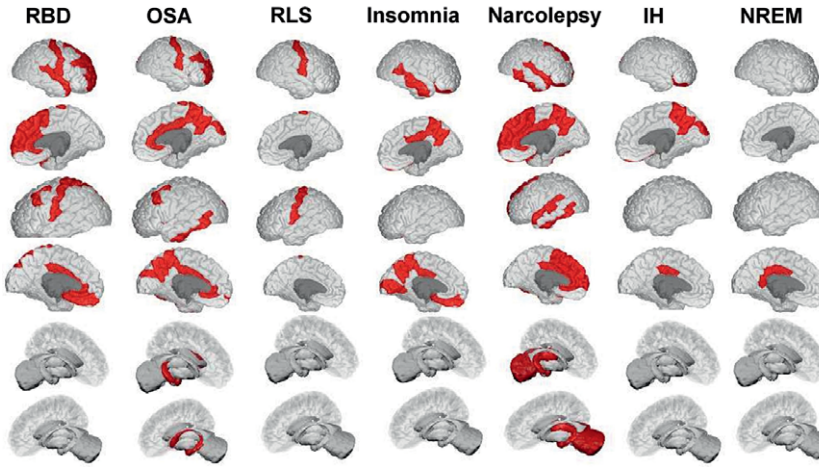

Figure 5.1 GM thickness changes across different sleep disturbances
(RBD – rapid eye movement (REM) sleep behavior disorder; OSA – obstructive sleep apnea; RLS – restless legs syndrome; IH – idiopathic hypersomnia; NREM – non-REM (parasomnias).
Used with permission from Paulekiene et al. (2022)

5.3.12 Conclusions

If we look across several sleep disorders, we can measure the toll these sleep problems take on measurable brain systems by examining Figure 5.1. This figure depicts forebrain gray matter volume losses associated with each of several sleep disorders including RBD – rapid eye movement (REM) sleep behaviour disorder; OSA – obstructive sleep apnea; RLS – restless legs syndrome; IH – idiopathic hypersomnia; NREM – non-REM (parasomnias). Gray matter volume loss is depicted in red. Please notice the striking difference in loss volume and pattern for the REM-based sleep disorders (RBD and narcolepsy) versus the NREM disorders.

The neuroscience of sleep and dreams teaches us that there are three basic brain states: waking, REM, and NREM sleep. What determines or creates and maintains each of these three states is a differing mixture or profile of brainstem-generated neurotransmitter (aminergic and cholinergic modulation) activity levels as well as differing forebrain activation and deactivation patterns, which were discussed in previous chapters. The three different brain activity profiles that give rise to the three different brain states must be thought of as probabilistic profiles. Each brain state's profile can be fully engaged or only partially engaged. Most importantly for understanding the experiences associated with parasomnias, the transitions between the brain states can be

complete or only partial. When one state ends, another state begins if the transition between states is complete. But because the mechanisms that control brain states are probabilistic, transitions between states are virtually never entirely complete. When transitions between states are partial, we get a hybrid brain state; for example, a mixture of REM and waking, or a mixture of NREM and waking, or REM with NREM. When these hybrid states occur, we get the classic parasomnias.

For example, sleep paralysis represents a hybrid of REM and waking. The patient is conscious and awake, but he is paralyzed and cannot move because the muscle atonia associated with REM is persisting into the wake state. In addition, the patient hallucinates seeing an intruder, possibly because many REM dreams are about potential threats, and so on. Similarly, lucid dreams (to be discussed later) are a hybrid of REM and waking states because the dorsolateral prefrontal cortex is partially activated in lucid dreaming. In normal REM, the dorsolateral prefrontal cortex is deactivated. But when it is activated during REM, the individual gains some awareness of self and so becomes aware that he is dreaming. Sleepwalking/talking and other NREM parasomnias represent a hybrid between NREM and waking. In these cases the individual remains in N2 or N3 sleep but can nevertheless engage in complex behavioral actions without awareness. Here, the brain can coordinate complex behaviors without awareness because the waking state (presumably mediated by an activated dorsolateral prefrontal cortex) has intruded upon a brain not fully disengaged from slow wave activity. In REM behavior disorder the muscle atonia associated with REM is abolished due to disease and thus, the sleeping brain is not fully in REM and we get dream enactment behavior – once again complex actions and behaviors but this time under the control of the sleeping brain. The REM/wake and NREM/wake hybrid states suggest that consciousness requires participation of dorsolateral prefrontal cortex and its connections, as whenever that brain network is activated, self-awareness and critical insight ensue.

We have discussed the hybrid states of REM plus waking and NREM plus waking. But what about the hybrid of REM and NREM? Most sleep scientists believe that the REM/NREM hybrid just yields unconsciousness. But it is possible that some parasomnias may involve the REM/NREM hybrid. Although nightmares typically arise out of REM, trauma-related nightmares can occur outside of REM. The phenomenon of sleep terrors suggests that the individual experiencing the NREM parasomnia is also experiencing an intense nightmare, as the individual typically screams in terror. Theoretically, I see no reason why some REM features, such as intense amygdalar activity, cannot temporarily occur with slow wave activity in many other parts of the brain. Such a state would produce a hellish nightmare.

Each brain state and their hybrids produce differing experiential worlds, with the waking state being the state we choose to identify with. All other human cultures choose likewise, but some add the REM state as an additional state of being that, while not equal to the waking state, is nevertheless considered ontologically real and is sometimes privileged vis-à-vis the waking state. These same cultures privilege some hybrid REM/wake states (e.g., waking dreams/visions). Apparently, no cultures privilege the NREM state, but many cultures produce religious, magical, and medical treatments to prevent hybrid NREM /wake states (e.g., sleep walking) and some REM/wake states (e.g., sleep paralysis). While the unusual states of consciousness associated with the parasomnias clearly interact with culture, sleep science has yet to develop a clear evidence-based theory of these hybrid brain states, leaving it as a task for future sleep scientists.

5.4 Review Questions

- What might failures to transition from one brain state to another teach us about consciousness?
- What is the significance of abnormal N3 delta wave activity for NREM parasomnias?
- How does failure to transition out of REM explain sleep paralysis nightmares?
- What are the major causes and consequences of insomnia?

Further Reading

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CHAPTER SIX

Theories of REM and NREM Sleep

Learning Objectives

- Identify and evaluate the evidence for the claim that NREM promotes enhanced immune responses
- Evaluate strengths and weaknesses of the restorative theory of sleep
- Identify and evaluate the evidence for sleep–related memory consolidation processes
- Describe functional significance of REM–NREM physiologic interactions

6.1 Introduction

Although we know a lot about sleep, there is no scientific consensus on its function or functions. Its functions, however, must be extraordinarily significant, given that it renders us vulnerable to predators each time it overcomes us. It is involuntary. Everyone must eventually succumb to sleep or die. We must have it as surely as we must have oxygen, food, and water. But we do not know why.

It is possible and even likely that sleep has more than one function. Indeed, NREM sleep may have separate functions from REM sleep, and the two sleep states may have complementary or contrasting functions. The functions of sleep may have arisen in concert or separately during sleep's evolutionary history. If functions arose sequentially during evolutionary history, then later functions would likely utilize physiologic systems designed to meet earlier functional needs. For example, although lungs likely evolved for gas exchange, they now can also handle speech and language vocalization functions. Similarly, although mammalian sleep may have originally evolved for thermoregulatory purposes, it is now crucial for brain and cognitive functions as well.

Yet, we can find simple quiescent-activity patterns in even very simple organisms like the worm (C elegans), which has only 302 neurons. If sleep can occur in these simple organisms without complex nervous systems, then the primordial form of sleep must be an emergent property of small neuronal assemblies.

6.2 NREM

As discussed in previous chapters, NREM is composed of three stages, but there are no evidence-based theories of the potential functions of N1 or N2 stage sleep. Therefore, we will focus first on potential functions of stage N3 and specifically slow-wave activity and then REM.

A tried and true method for discovery of function of a particular system in neurophysiology is to block or delete the system and then see what physiological functions are lost. In the 1980s, Allan Rechtschaffen and colleagues (Rechtschaffen et al., 1989) at the University of Chicago studied the consequences of total sleep deprivation in rats. Using a variant of the platform method for sleep deprivation whenever the experimental rat fell asleep, it was woken up. Basically the platform method involved the rat sitting on a platform that dipped into water if the rat ever fell asleep. The water would then awaken the rat so the rat could never sleep. Control rats were given the same platform stimulation but were allowed to sleep some of the time. Of course, this is an incredibly stressful procedure for rats, so it is difficult to factor out the effects of stress from the effects of sleep deprivation, per se. While the control rats were normal with no health consequences noted, the sleep-deprived rats died after sixteen to twenty-one days without sleep. The sleep-deprived rats lost weight even though they ate voraciously. Their fur became oily and matted, and they developed sores on their skins and feet. They could no longer regulate their metabolic and internal heat systems. Most died of septicemia, indicating immune system collapse.

6.2.1 NREM Sleep Promotes Optimal Immune System Responses

These results and others reviewed shortly suggest that sleep, and SWS in particular, very likely contributes to regulation of immune system response. In humans and other animals, sleep loss leads to the increased production of pro-inflammatory cytokines. Chronic sleep loss leads to chronic inflammation. Sleep durations of laboratory animals increase after an infection when mounting an immune response. Presumably, evolved increases in sleep duration allow animals to channel more metabolic resources into their immune system components – maintenance and repair. Short-term increases in sleep duration appear to be triggered by immunomodulatory cytokines that are released by white blood cells during immune reactions (Opp & Krueger, 2015). For example, administration of interleukin I (IL1) is associated with immediate production of slow-wave sleep. IL-1 stimulates the synthesis and/or release of growth hormone-releasing hormone, prostaglandin D2, and adenosine. Each of these substances is implicated in regulating or modulating

NREM sleep, and antagonizing these systems attenuates or blocks IL-1-induced increases in NREM sleep. Adenosine production, as reviewed in previous chapters, is known to be directly related to slow-wave sleep homeostatic mechanisms. If larger numbers of white blood cells produce a greater immune response to antigenic challenge, and hence a greater release of sleep promoting cytokines, this could potentially drive evolutionary increases in sleep durations.

Preston et al. (2009) assessed the correlated evolution of sleep and the immune system. To do so, they extracted data on sleeping times for different mammalian species from the published literature and matched these data where possible with white blood cell counts reported by the International Species Information System (ISIS). White blood cell counts were used as a proxy for immune system investment, as they are central to all immune responses and are a validated measure of immunocompetence. White blood cells originate in bone marrow and are derived from the same hematopoietic stem cells that produce red blood cells and platelets. As these latter cells have no direct immunological function, the investigators used them as natural controls to test the specificity of any relationship between sleep and the immune system. If a key selective advantage of NREM sleep is that it allows greater investment in the immune system, then species that sleep for longer periods of time should have increased numbers of immune cells in circulation, but there should be no similar relationship with control cells. After matching species values from each database, the authors were able to analyze data for twenty-six mammalian species while controlling for confounding factors (e.g., body size and activity period). As expected, species that engaged in more sleep had higher numbers of white blood cells circulating in peripheral blood. A fourteen-hour increase in sleeping times across species corresponded to an additional 30 million white blood cells in each milliliter of blood (a 615 percent increase). Crucially, no similar patterns were evident with either red blood cells or platelets. These relationships were also tested using phylogenetically independent contrast methods to account for the lack of statistical independence in species data. These analyses identify evolutionary change in relation to other variables (in this case change in sleep durations of different lineages in relation to immunity variables). Preston and colleagues found that when lineages evolved longer sleep durations, they also increased their white blood cell counts. Again, this relationship was specific to immune cells, leading to an increase in the ratio of immune cells to other blood cell types when species evolved longer sleeping durations. The lineages and species that developed longer sleep durations also turned out to be less parasitized. That is, those species' parasite loads were far lighter than species with shorter sleep durations, even after accounting for potential confounds like body weight, size, ecologic niche, and activity

schedules. These data strongly support the contention that one evolutionary function of NREM sleep is that it promoted immunocompetence.

6.2.2 Sleep Is for Rest/Replenishment: Sleep Restores Energy

The most commonsense explanation for sleep is that it restores our energy level. These energy stores get depleted during wake and then replenished during sleep – or so the theory goes. Certainly, overall metabolic rates decline during sleep, relative to the quiet resting state during wake. But the energy saving one gets during sleep-related declines in metabolic rates relative to wake-related rest periods turns out not to be that substantial. So the feeling of restoration after a good night's sleep must be related to more than just the energy saving one could get from quiet rest.

The brain accounts for about 20 percent of the entire body's energy expenditure, even though it is only about 2 percent of the total body mass. The brain's only energy source is the sugar glucose. Sugar in the brain is manifested as glycogen. Glycogen molecules are long, branched chains of glucose molecules. There are two major neural cell types in the brain – neurons and glial cells. Glia make up about 90 percent of brain tissue, and they synthesize and store glycogen. When the activity in a brain area suddenly and rapidly increases and the glucose in the blood flowing through that area is not sufficient to fuel that activity, the glia rapidly break down their glycogen and supply fuel to the neurons.

Glia also forms a kind of lymphatic system for neurons. Just as the lymph nodes in your body remove toxic byproducts of the metabolic machinery in your body, so too does the glymphatic system help to remove toxic byproducts of the neuronal metabolic machinery. During NREM N3 this glymphatic system steps up its activity ten- to twenty-fold. relative to its daytime activity. During N3 the glial cells shrink to half their size during NREM, thus allowing CSF to bathe the neurons and flush out toxic byproducts from the system. One of these toxic byproducts of neuronal activity is amyloid proteins, which are known to entangle and kill neurons building up clumps of dead tissue in the brain that, in turn, can ultimately contribute to neurodegenerative disorders such as Alzheimer's disease.

As mentioned, glia also participate in the breakdown of glycogen in order to produce fuel for the brain. One potential major function N3 SWS is to enable the restoration of the brain energy reserves in the form of glycogen. The brain and body's key metabolic machinery to produce energy is adenosine triphosphate (ATP). The accumulation of extracellular adenosine is a signal that cells or tissues are running out of energy. In the brain, adenosine opens potassium channels in the neuronal cell membrane. Opening those channels increases the

movement of positively charged potassium ions out of the neurons. This movement of positive charges leaves behind negative charges, so the electrical potential across the membrane becomes more negative. We say that the neuron is hyperpolarized. When the calcium channels open, calcium rushes into the neuron, it becomes depolarized, and the neuron generates action potentials. It is these action potentials that are measured as slow waves in the EEG. Bennington and Heller (1995) demonstrated adenosine activity levels covaried with brain slow–wave activity. When they injected a molecule that mimics the action of adenosine into the blood or brains of rats, they showed what appeared to be normal sleep for several hours with very high slow–wave activity. During sleep, adenosine levels gradually fall in all brain areas, but during waking, adenosine levels gradually and significantly rise predominantly in two brain areas – the cerebral cortex and the basal forebrain. Adenosine acts in the basal forebrain to influence the switch from wake to sleep and to maintain this flip-flop switch in the sleep position; second, it acts in the cortex to produce and sustain the slow-wave activity that seems to be the homeostatically regulated variable of sleep.

Glycogen breakdown (known as glycogenolysis) is accomplished through the actions of an enzyme called glycogen phosphorylase. Glycogen synthesis is accomplished through the actions of an enzyme called glycogen synthase. The neurochemicals that promote wake, such as norepinephrine, activate the glycogen breakdown enzyme and inhibit the glycogen synthesis enzyme. When those neurochemicals are withdrawn – resulting in less activity in the brain stem nuclei that promote wake – the glycogen breakdown enzyme is inhibited, and the glycogen synthesis enzyme is activated. In addition, ATP inhibits the glycogen breakdown enzyme; thus, if the cell has plenty of energy and its ATP levels are high, it does not use its glycogen reserves. During wake, the glia are poised to give up their glycogen at a moment's notice if energy levels start to fall, and during sleep, the glia are put into the mode of replacing their glycogen stores.

Adenosine seems to have dual actions: working at the level of the basal forebrain to promote the transition to sleep and working at the level of the cortex to promote the intensity of sleep. It turns out that activity in different regions of the cortex – activity that could deplete the glycogen in those specific areas – result in regional sleep responses. Cortical regions that work harder, such as the ventromedial portions of the frontal lobes, have greater slow-wave activity during subsequent sleep.

One of the strongest pieces of evidence that N3 sleep functions to restore energy is the phenomena of sleep rebound after sleep deprivation. Periods of enforced waking lead to increased NREM sleep drive or sleepiness. This sleep need can be relieved by subsequent sleep, thus supporting the restorative

theory for NREM sleep. Interestingly, the recovery sleep typically occurs first for NREM, and only after NREM is made up is REM made up. Sleep deprivation, in other words, produces compensatory increases in both NREM sleep time (specifically NREM delta activity) and REM sleep time during recovery sleep, but NREM is made up first. NREM delta activity has been shown to accumulate during normal periods of consolidated wakefulness and it discharges or declines during subsequent NREM sleep. Apparently, mammals need a certain amount of SWS in order to function properly.

Many theorists of sleep function have argued that NREM functions to restore physiological functioning. Certainly the subjective feeling of being refreshed after a good night's sleep supports these restorative theories, as do the findings of sleep rebound after sleep deprivation. A potential problem for the restorative theory of N3 SWS is that there is a rebound of delta sleep (slow-wave NREM sleep) after hibernation in hibernating animals. Hibernation, of course, is a low-energy state and should not need a process to restore energy or repair tissue since little or no energy was consumed and little or no tissue was damaged during the hibernation period. Indeed, the standard explanation for hibernation is that it is a state designed to conserve energy! Yet animals undergoing hibernation or torpor generally have to arouse periodically in order to engage in slow-wave activity.

6.2.3 NREM Restore Optimal Cognitive Performance Particularly for Frontal Lobes

Sleep deprivation leads to performance decrements on a variety of cognitive tasks – especially tasks that depend on integrity of the frontal lobes (Achermann et al., 2001; Anderson & Horne, 2003). The degree of impairment is wake dependent and use dependent. The greater the use of the frontal lobes for tasks that depend on the frontal lobes, the greater the degree of compensatory delta activity in the frontal lobes. Normal performance can be restored by sleep in a sleep dose–dependent manner – the more intense the delta activity, the better the subsequent performance on the cognitive tasks. Clearly, then, sleep can function to restore cognitive performance, particularly for tasks dependent on the frontal lobes.

6.2.4 NREM Promotes Optimal Neuronal Connectivity

Perhaps the most commonly proposed function for sleep put forward by neuroscientists is that sleep promotes neuronal connectivity (Benington & Frank, 2003; Huber et al., 2004). Synapses between neurons and via extracellular signaling regulatory molecules form functional networks that are the physiologic basis of the brain/mind. Synapses are formed on the basis of

use; that is, they are activity dependent. The greater the activity, the greater and the stronger the synaptic connectivity. Use and reuse of synapses leads to strengthening of synaptic efficacy of active synapses and therefore reuse. But this strengthening of connections leads to problems for brain dynamics. The eventual consequence of use and reuse of synaptic circuits would be an overly connected set of rigid networks and ultimately a totally connected brain without any residual brain plasticity. Without plasticity, of course, learning would be impossible. Negative feedback mechanisms are needed to disturb rigidly connected networks and to continuously introduce plasticity. Sleep, in these connectivity theories, paradoxically does two things: It performs that function of introducing disruption of rigidly connected networks while also strengthening use-related synapses. It both strengthens some synapses and weakens others. How does sleep simultaneously strengthen synapses in a use-dependent fashion while also introducing mechanisms that avoid creation of rigid networks? We do not yet know the answer to this question.

6.2.5 NREM Promotes Certain Forms of Memory Processing

The active systems consolidation theory suggests that during NREM sleep, slow-wave activity facilitates processing of various forms of memory via the transfer of incoming memories from the hippocampus, and via the thalamus and into long-term cortical stores. This process is orchestrated via synchronization between hippocampal sharp-wave ripples, nested in thalamic spindles that are nested in cortical slow oscillations (see Figure 6.1). Hippocampal memory episodes or engrams encoded during wake via neural firing patterns are literally

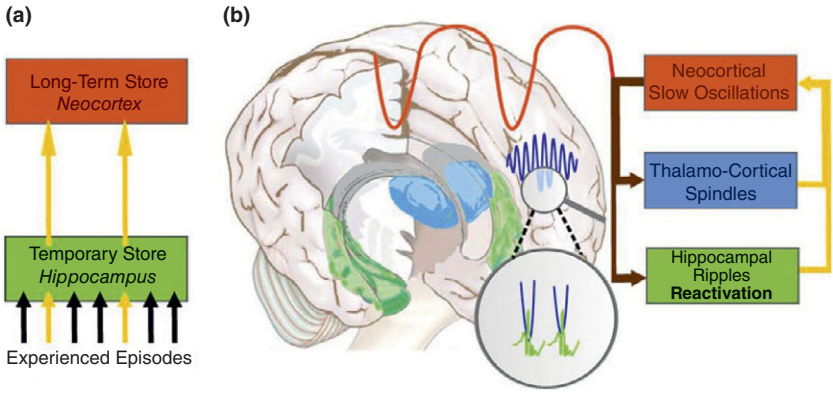

Figure 6.1 The active systems consolidation theory
Used with permission from Born & Wilhelm (2012)

reactivated in the same firing pattern during subsequent non-REM sleep. The replay facilitates stabilization of the memory and its long-term storage. This "replay" has been associated with hippocampal sharp–wave ripples.

The active systems consolidation theory, a dominant theory of sleep-dependent memory consolidation, suggests that slow-wave oscillations, sleep spindles, and hippocampal sharp-wave ripples support memory consolidation during non-REM sleep (Diekelmann & Born, 2010).

6.3 REM

The case for a functional role for REM is more complicated than that of NREM. In the case of NREM, we have seen that deprivation of NREM produces a clear compensatory rebound effect of recovery NREM sleep that apparently makes up for some of the lost NREM sleep. After decades of experimental work involving hundreds of REM sleep deprivation (RSD) experiments (mostly in rats, cats and humans), there is still no consensus on whether the compensatory REM rebound represents a form of sleep that must be made up (i.e., obligate sleep; see Horne, 2000). Aside from a partial REM rebound effect, no significant psychologic or biologic effects are noted with REMD – at least for short-term REMD in humans. With prolonged (sixteen to fifty-four days) REM deprivation there appears to be a heavy price to pay; in rats, prolonged REM deprivation leads to death (Rechtschaffen et al.,1989). But it is questionable whether death is due to selective REM deprivation, since SWS deprivation results in death as well (in twenty-three to sixty-six days). In addition, it is difficult to separate out the aversive/stressful effects of REM deprivation from effects of REM deprivation, per se. It is very difficult to selectively deprive an animal of a given sleep type over a long period of time. Death in the prolonged SWS/REMD studies is believed to be due to a significant decline in core body temperature (as much as 2C), with compensatory attempts to increase temperature through increased energy expenditure. Another potential cause of death in rats after REMD may be a systemic bacterial infection. On the other hand, administration of antibiotics to REM-deprived rats did not prevent death.

REM probably does not function to restore energy. It does not appear to replenish stores of metabolic energy; instead it dissipates significant amounts of energy. Neuroimaging studies of REM show that it reaches brain activation levels and brain glucose utilization levels exceeding those of the waking state. If the restorative theory is correct, then the energetic gains that accrue from sleep must be greater than what can be achieved by simple daytime rest. Another problem with energy replenishment or energy conservation theories is that body temperature does not decrease during REM, and metabolic activity

does not decrease during REM. Nor do metabolic rates correlate with REM times. If we recall that REM alternates with NREM throughout the night, then energetic savings would have to accrue selectively during the NREM periods. The energy savings must somehow be maintained across REM periods to result in any significant net reduction in metabolic rates across the night, or else any savings accrued in a given NREM period would be immediately dissipated in a subsequent REM period, resulting in no net gain for the animal.

6.3.1 Memory Consolidation

A number of sleep researchers have proposed that REM functions to consolidate various types of memories (Smith, 1995; Stickgold, 2005). REM times, for example, increase after intense learning episodes, particularly after procedural types of learning tasks. Consolidation of learning of Morris water maze tasks in rats can be blocked if the animal is deprived of REM during a critical time window, which typically occurs sometime (on the order of hours) after the learning trials. By comparison, Crick and Mitchison (1983) postulated a kind of reverse learning function of REM to rid relevant neural networks of unnecessary bits of information. Crick and Mitchison, however, did not consider the role of NREM. An empirically supported theory of REM-based memory consolidation is the sleep-to-forget, sleep-to-remember hypothesis (see Figure 6.2; van der Helm & Walker, 2009). This suggests that REM sleep offers a preferential window for emotional processing through the reactivation of emotional memories. Through each REM episode over the course of the night, episodic memories are stripped of their emotional or arousing valences to leave the semantic informational content that will then be more easily stored. The high-cholinergic, low-aminergic modulation of brain circuits during REM facilitates brain plasticity processes while REM theta oscillations between hippocampal and neocortical sites promote memory consolidation, facilitation of task-oriented cognitive control capacities, and belief updating. Emotional memories, which rely critically on the amygdala for their consolidation, do indeed appear to benefit most from REM sleep. In a typical study of the role of sleep in emotional memory consolidation, Wagner et al. (2001) found that three hours of late-night REM-rich sleep (but not three hours of early night slow-wave–rich sleep or three hours of wakefulness) facilitated memory for narratives with intensely negative emotional content.

What about the role of REM dreams in memory processing? It may be that dreams do not affect memory consolidation processes at all. Thus, there is nothing much to say about dreams and memory consolidation processes except that the two have nothing to do with one another. Another possibility is that dreams do not causally participate in the memory consolidation process, but

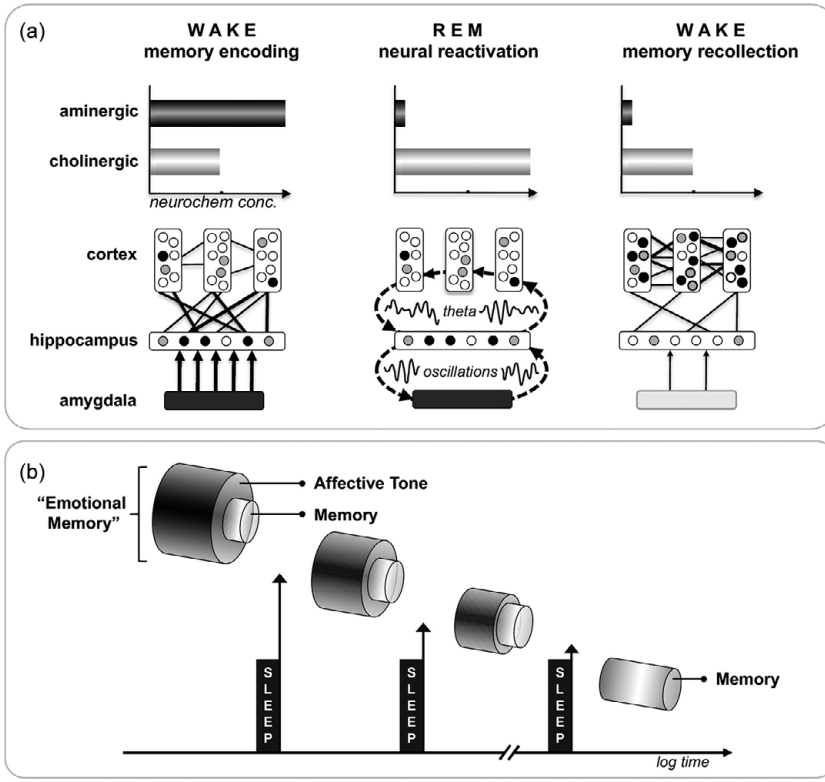

Figure 6.2 The sleep-to-forget, sleep-to-remember hypothesis for REM
(The hypothesis suggests that the declarative content of emotional memories is strengthened over repeated episodes of REM sleep, while the affective tone is gradually reduced).
Used with permission from Walker and van der Helm (2009)

they do reflect it – they are byproducts of the process in this view (e.g., Foulkes, 1985). We can learn about memory consolidation by studying the ways in which dream content changes over a single night of sleep and over time within a person's life. A third school of thought asserts that dreams do not merely reflect memory consolidation processes but instead are causally implicated and may even be required for the consolidation of certain types of memories – especially emotional memories. Nielsen's et al.'s (2004) work on the dream lag effect is instructive. The dream lag effect refers to the empirical finding that items being encoded into long-term memory sometimes appear in one's dreams (usually) the night after the event was experienced and then subsequently five to seven days later. If dreams were mere reflections of ongoing memory consolidation processes, then one would expect dreams to

reflect ongoing memory experiences each night, but instead we get this interesting lag effect, which suggests a more important role for dreams than previously expected. Emotional experiences may be reactivated during REM sleep over a time-course of at least a week. In another study, participants (n = 20, 10 female, mean age 21 years) were woken from PSG-recorded overnight sleep (REM and SWS) in the laboratory and were again asked for immediate dream reports. The frequency of references to recent waking events in REM dreams was significantly associated with REM theta activity (Eichenlaub et al., 2018). Theta rhythms have been previously associated with emotional memory consolidation (Hutchison & Rathore, 2015). Therefore, these results also suggest that recent events are replayed and consolidated during REM sleep. Another line of evidence that suggests an important role for dreams in memory consolidation processes comes from Hartmann's work (Hartmann, 1998) on central images in nightmares that apparently facilitate encoding of intense emotions into long-term memory.

6.3.2 Dialogue between the Hippocampus and Cortex during REM

It has been known for years that damage (via stroke, head injury, or surgery, etc.) to the hippocampus impairs the acquisition of new memories. Recent advances in neuroplasticity research has demonstrated that one of the areas of the brain that can generate new neurons (neurogenesis) is the hippocampus. Sleep deprivation can impede neurogenesis in the hippocampus.

Most models of how sleep facilitates memory processes involves communication between the hippocampus, amygdala, and the cerebral cortex, where long-term memories are stored and formatted for later use in cognitive processes. In these models, the hippocampus is considered a temporary repository for abstracting context from memories; the amygdala separates context from emotions, and the cortex is the site where context-free memories are stored over long periods. Both the hippocampus and the amygdala (along with critical subcortical sites such as the dopaminergic "salience system" centered in the ventral striatum) act as selectors, gatekeepers, or sieves for what information ultimately gets encoded into long-term stores versus what information gets discarded. During sleep, external input is reduced so memory consolidation processes can occur "offline," free of the potentially interfering effects of ongoing incoming information from the external environment. Each of the two major sleep states are believed to play a specialized role in the preparation (e.g., the encoding and formatting) and storing of long-term memories, with NREM playing a central role in stripping context from episodic and procedural memories and REM playing a central role in stripping context from emotional memories.

As the brain cycles between NREM SWS and REM, across the night there occurs a complex interaction between activity patterns in the amygdala/hippocampus subcortically and the cortex. The communications between the hippocampus and cortex involve an interesting choreography of different brain waves. The slow waves of non-REM sleep – the sleep spindles – that originate in the circuits involving the thalamus and the cortex, and the brief sharp-wave ripple events that originate in the hippocampus, all appear to index key steps in the memory consolidation process. Sleep spindles index activation of the thalamus and cortex while the slow-wave oscillations index synchronizing activity in the hippocampus so that hippocampal activity also occurs in frames synced up with the spindling activity in the thalamus/cortex. As a result, sleep spindles and sharp-wave ripple events in the hippocampus occur together. It is believed that these spindle-ripple events reflect communication of information between the hippocampus and the cortex. In short, during non-REM sleep, there is a dialog between the hippocampus and the cortex that is coordinated by slow waves, sleep spindles, and sharp-wave ripples.

While information appears to flow from the hippocampus to the cortex during SWS and is indexed by sleep spindles and sharp-wave ripple activity, theta rhythms are thought to support the transfer of information in the opposite direction during REM sleep. Theta waves enhance hippocampal long-term potentiation (LTP), a candidate mechanism for memory formation. Interestingly, this synchronization with theta wave activity during REM sleep appears to shift from in-phase (i.e., correlating with the peak activity of the theta wave) to out-of-phase (correlated with the troughs of inactivity) over four to seven days of daily exposure to a new environment. Such a shift could produce a switch from LTP and memory consolidation to long-term depression (LTD) and memory erasure. The time course of this shift is similar to the dream-lag effect mentioned previously and suggests that this is the time required for hippocampally dependent memories to be transferred to the neocortex for long-term storage.

Sejnowski and Destexhe (2000) provided a model of the sleep-related consolidation process that depends on spindle oscillations during early SWS and an alternating pattern between slow-wave complexes and brief episodes of fast oscillations as SWS deepens. Together, these considerations suggest a model of sleep–dependent sequential memory processing in which different types or aspects of memories, including emotional memories, are processed progressively over the course of the night. In this model, specific memories from the recent past could be identified at sleep onset for subsequent reprocessing and then stabilized (via context stripping) and/or strengthened, possibly during NREM sleep and integrated into cortical networks during REM. The alternating REM and NREM periods would then permit several cycles of stabilization and integration.

Box 6.1 Targeted memory reactivation in sleep

Targeted memory reactivation (TMR) refers to a new technique to get at memories during sleep. Researchers present cues or stimuli, such as a smell, that was associated with events or information or materials that the subject learned prior to sleep. Then when the subject is asleep, the subject is reexposed to the associated cues (e.g., a smell) and then later tested (after awakening) for memory of the original materials. When the cues are presented to the subject during sleep there is better retention of the original materials; for example, if you studied a foreign language in the presence of the smell of roses and then went to sleep. You would do better on a test of that foreign language after awakening if you had smelled roses during sleep. The smell facilitates consolidation of the memory during sleep. TMR has been shown to result in a performance gain of around 20 percent, as compared to natural sleep. This gain in performance could make a huge difference for people who are experiencing memory loss. Essentially, TMR is a kind of priming or activating of the memory system using specific sensory cues. Priming effects in social psychology are generally small effects and have proven difficult to replicate. Experimental data in TMR studies are not consistent across studies and extensive research is warranted to address many open questions before TMR can be considered established, effective, and safe (Schouten, Pereira, Tops, & Louzada, 2017).

In summary, while REM clearly plays a crucial role in memory consolidation, it does so in concert with NREM sleep states. Does REM exhibit any functions not also exhibited by NREM sleep states? The answer appears to be yes.

6.3.3 REM Functions to Promote Brain Development

Brain development relies on both experience and genetics. Genetic elements guide development via information encoded in genes. Experience is collected from the surrounding social and ecological environments as translated by levels of activity of steroid stress hormones and memory systems. In chaotic or harsh environments with high mortality rates among conspecifics, brain development will be canalized along rapid trajectories and favor only essential systems and defensive postures. Functional neural elements and systems are established via pulses or phases of intense neuronal proliferation and migration beginning embryonically and extending into the postnatal period. Certain

periods during development appear to be time windows where certain brain circuits, for example, aggressive defensive circuits, are particularly receptive to surrounding social and ecological. These are sometimes called "critical periods." These critical periods are phases of heightened neuronal openness or plasticity where lifelong structures are installed and then become resistant to change. Juveniles spend the bulk of their time in sleep with particularly abundant periods of REM. Why is sleep, particularly REM, so abundant early in life during this critical period for brain development? The short answer is that sleep enhances critical period plasticity and promotes experience-dependent synaptic pruning in the developing cortex. Therefore, normal plasticity during critical periods depends on proper sleep, particularly REM.

Dumoulin Bridi et al. (2015), for example, presented evidence that REM sleep in early life promotes brain development by facilitating release of a kinase critical for neuronal plasticity, so-called extracellular signal–regulated kinase or ERK. Kinase phosphorylation in the primary visual area of the cortex, area V1, requires REM sleep, because it will not occur in sleep-deprived animals. REM-induced ERK phosphorylation is essential for plasticity changes in the visual region of the brain. REM sleep deprivation after monocular deprivation (suturing one eye shut) profoundly and selectively inhibited ERK phosphorylation in V1 in kittens. Preventing REM sleep after monocular deprivation reduced ocular dominance plasticity (normal visual development) and inhibited activation of ERK, the kinase critical for this plasticity.

Formation of new neuronal circuits in the brain is accomplished by experience-dependent selective pruning and elimination of an initial exuberant overgrowth of synapses during development. REM deprivation prevents selective pruning and elimination of neuronal synapses. Li et al. (2017) have shown that REM sleep promotes pruning of newly formed postsynaptic dendritic spines of layer 5 pyramidal neurons in the mouse motor cortex during development and motor learning. Dendritic calcium spikes arising during REM sleep are important for this pruning and strengthening of new spines (Li et al., 2017). Despite this kind of evidence of REM facilitation of brain development, it is difficult to understand why, if REM's function is purely to promote brain development, it should persist into adulthood or have the properties it has both in the juvenile and adult. Some juvenile aquatic mammals may not have REM at all, yet their brain growth is normal. Jouvet (1999) has suggested yet another version of the brain development hypothesis for REM. He proposed that REM is important for maintenance of synaptic circuits that mediated inherited behaviors. If the cells that mediate the muscle atonia associated with REM are destroyed in cats, you see them engage in instinctual predatory behaviors when they go into REM sleep. The penile erections associated with REM are another example of instinctual behaviors manifested in REM.

6.3.4 REM Regulates Expressions of Emotions and/or Emotional Balance

Many studies involving deprivation of REM have resulted in enhanced motivational states and changes in emotional state. Thus, some investigators have suggested that REM functions to inhibit or modulate emotional arousal and motivational striving. Cartwright (2010) argued that daytime mood is associated with nighttime sleep amount and quality. Vogel demonstrated that REM deprivation improves depressive mood states in depressed patients (Vogel, 1999). REMD presumably alleviates depression by enhancing drives and other motivated behaviors (i.e., REMD removes REM's tonic inhibitory effects on drives and emotions). Hartmann (1998) has argued that REM values and REM dream content vary with a person's stress levels and emotional history, and that REM dreaming functions to rebalance emotional responses. Neuroimaging studies are consistent with the emotional regulation view of REM, as REM is associated with high activation levels in the limbic system and the amygdala. Van der Helm and Walker (2011) have produced experimental evidence that supports a role for REM in emotional memory consolidation and emotional regulation. Sleep periods rich in REM sleep as opposed to NREM sleep are associated with significantly greater amounts of emotional memory consolidation and recall. REM sleep is rich in acetylcholine activity (which is known to be crucial for memory encoding processes) and may act to decontextualize negative or fearful memories and thus make them easier to consolidate into long-term memory stores.

6.3.5 NREM–REM Interactions and Genetic Conflict

We have seen that REM and NREM may sometimes interact in an antagonistic fashion such that REM undoes something NREM does and vice versa. REM and NREM seem to express functionally antagonistic traits and processes across a spectrum of functional states (see Table 6.1). REM and NREM are in fact in mutual inhibitory balance – when values of NREM increase, values of REM decrease and vice versa. REM sleep is promoted by cholinergic neurons originating within the LDT/PPT and inhibited by noradrenergic and serotonergic neurons in the locus coeruleus (LC) and dorsal raphe (DR), respectively. NREM sleep is promoted by turning off the cells that promote REM or wake. For example, inhibiting (via GABA-ergic-mediated processes) firing of cells in the reticular activating system, LC, DR and thalamus, results in synchronous bursts of thalamic and then cortical neurons, the loss of wake-related alpha waves, and gradual slowing of the forebrain EEG frequency. Removal of inhibition exerted by aminergic efferents on cholinergic cells in the LDT/PPT results in reentry into REM.

Table 6.1 REM–NREM characteristics suggesting opposing functional states

	REM	NREM
Mouse strains indicating separate genetic influences	C57BL and C57BR are associated with increased REM and short SWS episodes	BALB/c is associated with short REM and long NREM episodes
Prader-Willi syndrome (paternal deletions / maternal additions on C15) Excessive sleepiness	decreased	increased
Angelman syndrome (paternal additions/ maternal deletions on C15) Sleeplessness	increased	decreased
Nursing (rat)	neonate typically in REM during nursing and REM % increases during milk ingestion up to 4% of body weight	mother must be in NREM-SWS for milk ejection to occur
Percent of total sleep time in adult	20–25%	75–80%
Distribution during sleep phase	predominates in last third of night	predominates in first third of night
Response to infection	decreased	increased
Sleep deprivation–related rebound (both REM and NREM)	repaid after NREM debt paid	(repaid before REM)
Cerebral blood flow	increased; social brain network structures re-connected and activated	decreased
Arousal thresholds	+	++
Eye movements	rapid eye movements with occasional bursts or clusters of rems	slow rolling eye movements
EEG	"desynchronized" Phasic events: REM bursts, muscle twitching, middle-ear muscle activity, hippocampal theta waves Tonic events: muscle hypotonia or atonia, especially in the antigravity muscles, penile tumescence	synchronized

		stage NI (light falling asleep), vivid dream-like imagery stage N2 (light sleep with K-complexes and spindles) stage N3 slow-wave sleep with delta waves
Muscle tone	decreased	intact
Metabolic rate under thermoneutral conditions	increased	decreased
Thermoregulatory reflexes	decreased to absent	present
ANS	increased variability/ANS storms/increased HR and BP	no significant changes
Neurochemistry	cholinergic REM-on cells 5-HT and NE REM-off cells	GABA important for NREM onset
Parasomnias/sleep disorders	nightmares, narcolepsy; depression	sleep terrors, sleep walking (i.e., confusional arousals)
Mentation	vivid dreams with narrative structure	N3 ruminative; N2 less storylike dreams; N1 vivid imagery
Hormones	somatostatin prolactin	growth hormone GHRH

Why are there only two major sleep states, and why do they interact in this antagonistic fashion? The simplest explanation for this state of affairs is that REM and NREM are regulated by separate sets of genes with opposing genetic or evolutionary interests (McNamara, 2004). Inbred mice strains C57BL and C57BR are associated with increased REM and short SWS episodes, while the BALB/c strain is associated with short REM and long NREM episodes, indicating separate genetic influences on REM and NREM sleep amounts. Some strains (e.g., AKR/J mice) are known to show high rebound after six hours of sleep deprivation, while other strains (e.g., DBA/2J mice) show only a

mild response. Reciprocal crosses of these AKR/J and DBA/2J lines produce sleep rebound (after experimental sleep restriction) effects as a function of parent of origin or imprinted gene expression.

Some neurodevelopmental sleep syndromes express strikingly opposite profiles in terms of sleep and related clinical characteristics, and these data suggest that the ultimate cause of these syndromes lies in a set of genes that control expression of these contrasting clinical phenotypes. For example, Kleine-Levin syndrome affects mainly young males and, like Prader-Willi syndrome (PWS, described shortly), is characterized by bouts of hypersomnia, compulsive eating, cognitive changes ("dreamy" states and derealization), signs of dysautonomia, and, unlike Prader-Willi, episodes of hypersexuality. Anorexia nervosa, on the other hand, affects mainly young females and is characterized by bouts of sleeplessness, self-starvation, cognitive changes, and hyposexuality.

Like anorexia and Kleine-Levin, Angelman and Prader-Willi syndromes are neurodevelopmental syndromes that involve opposite and contrasting sleep state changes. Prader-Willi syndrome is associated with maternal additions/paternal deletions of alleles at chromosome 15q11–13 and is characterized by poor sucking response, temperature control abnormalities, and excessive sleepiness. Sleep architecture changes have also been noted in children and young adults with PWS, most specifically REM sleep abnormalities such as sleep onset REM periods, REM fragmentation, intrusion of REM into stage 2 sleep, and short latencies to REM. Conversely, Angelman syndrome is associated with paternal additions/maternal deletions on chromosome 15q11–13 and is characterized by prolonged sucking, severe mental retardation, and insomnia or reductions in sleep. These children may sleep as little as one to five hours a night, with frequent and prolonged night wakings.

These syndromes can be construed as disorders related to the phenomenon of genomic imprinting (Haig, 2002; Isles et al., 2006; Ubeda & Gardner, 2010). Imprinting involves the inactivation or silencing of one allele of a gene, depending on its parental origin. Expression of the associated allele likewise depends on whether it was inherited from the father or the mother. Haig's (2002) evolutionary conflict model of the way genomic imprinting works suggests that paternally derived alleles act to enhance growth of a developing fetus or child regardless of its effects on the mother or siblings of the child, and that maternally derived alleles act to restrain growth or transfer of resources to any given offspring. The mother wants to restrain growth as it becomes easier to care for that child while also caring for the other children (siblings) as well. It is in her genetic interests to raise as many offspring as possible, so she has to divide up her investment across all her young. When the child is asleep, the mother can turn her attention to other siblings or perhaps get some sleep

herself. The father's genetic interest is in having the current child get all available resources without limit. So, the father wants the child awake and demanding more resources from the mother at all times. The reason the father wants to invest without limits in the current child is because he is much more certain of paternity (that he is the real biological father) for the current child than he is of future possible children. Independent studies have shown that "extra-pair couplings" that cuckold the father occur at significantly high rates in most mammalian lineages and in many avian species as well. The father has to assume that the next child the mother has will not be his, so he wants the mother to invest all she has in his current child and thus, increase his fitness accordingly.

Tucci and colleagues (2016) showed that loss of expression of paternal Snord116 results in enhanced REM sleep. Double expression of maternal Gnas (due to loss of imprinting) results in decreased REM sleep, which might imply that the normal role of imprinted single dose Gnas is also to decrease REM (assuming additive effects of expressed gene dosage of Gnas on REM). Gnas, an important imprinted gene, also plays a role in thermoregulatory processes. Moreover, a recent GWAS study exploring genetic associations with insomnia complaints in 113,000 individuals identified GNAS as the strongest candidate gene enhancing risk for sleeplessness (Hammerschlag et al., 2017).

Imprinted genes expressed in the brain can shape brain function and behavior. Garfield et al. (2011) studied the gene Grb10, which is expressed from the maternal allele during embryogenesis. Grb10 encodes an intracellular adaptor protein that can interact with several receptor tyrosine kinases and downstream signaling molecules. Tyrosine kinase is a rate limiting enzyme important in the metabolism of several neuromodulators including dopamine – the central neuromodulator involved in reward and motivational behaviors. Garfield et al. demonstrated that loss of the peripherally expressed maternal allele leads to significant fetal and placental overgrowth and that in the adult, allogrooming and social dominance behaviors increase in paternal Grb10-deficient animals. Thus, Grb10 influences both fetal growth and adult behavior, owing to the actions of the two parental alleles in different tissues at different times in the life cycle. Using fifteen single–cell RNA sequencing datasets, Higgs et al. (2021) systematically investigated imprinted gene overrepresentation at the organ, brain region, and cell-specific levels. They first established that imprinted genes are indeed overrepresented in the adult brain, and in neurons particularly, compared to other brain cell-types. They then examined brain-wide datasets to test enrichment within distinct brain regions and demonstrated overrepresentation of imprinted genes in the hypothalamus, ventral midbrain, pons, and medulla. Finally, using datasets focusing on these regions of

enrichment, they identified hypothalamic neuroendocrine populations and the monoaminergic hindbrain neurons as specific hotspots of imprinted gene expression. The authors concluded that imprinted genes are selectively and strategically involved in the neuronal regulation of motivated behaviors such as feeding, parental behavior, and sleep.

REM sleep may involve relatively selective activation of the brain network known as the "social brain network." The social brain network includes subcortical hypothalamic, amygdalar and limbic sites, and ventromedial prefrontal sites. All of these sites may be differentially influenced by paternal line genes. Maternal line genes, conversely, may influence the parietal-prefrontal network centered on the dorsal striatum and caudate nuclei with fibers projecting to dorsolateral prefrontal lobes. Keverne and colleagues (Keverne & Curley, 2008) showed that functionally distinct regions of the brain may reflect the distinct contributions of the maternal and paternal genomes with paternal line genes influencing a circuit centered on hypothalamus and limbic regions, and maternal line genes being expressed more often in neocortex.

If NREM during the course of the night involves gradual deactivation or taking offline the key structures of the social brain network, and if REM gradually reactivates these structures and imprinted genes control these deactivation and reactivation sequences during each sleep cycle, then sleep may be construed as a theater of evolutionary genetic conflict, with maternal line genes battling paternal line genes each night over control of the organism's brain structures in the social brain network and daytime social (ultimately reproductive) behaviors.

Noting that the genes within an individual do not necessarily agree on how their carrier should interact with social partners, Faria et al. (2019) used mathematical modeling to create a theoretical framework within which to understand social pressures on sleep. The authors arrived at the conclusion that genomic imprinting effects should regulate expression of sleep patterns. Because an individual can be more related to social partners through one parent than the other, then genes inherited from each of the two parents may differ with regard to the social behavior they favor. Staying awake versus falling asleep may be altruistic if it is to protect conspecifics during the night. Or staying awake may be selfish if it is used to pursue selfish opportunities including mating opportunities. Duration-of-sleep decisions, then, are truly a site for social (evolutionary) conflict. Maternal-origin genes and paternal-origin genes may then disagree on how much an individual should sleep. If individuals altruistically sacrifice sleep in order to protect their group mates from danger, then we expect that the genes for which relatedness between social partners is higher will be more strongly favored to sacrifice their carrier's sleep. Assuming female biased dispersal (arguably the human default

pattern where ancestral females left the local group to find reproductive opportunities in another group where they were genetically unrelated to others) – which reduces genetic relatedness between social partners with respect to their maternal-origin genes – leads to paternal-origin genes favoring less sleep and maternal-origin genes favoring more sleep. In contrast, if individuals sleep to selfishly increase their mating success, then the genes for which relatedness is higher will favor more sleep. Specifically, given that relatedness is higher for paternal-origin genes, maternal-origin genes favor more sleep and paternal-origin genes less sleep, if sleep is selfish. On the contrary, if sleep is altruistic, then maternal-origin genes favor less sleep and paternal-origin genes more sleep. What determines when an individual chooses altruistic sleep durations or selfish sleep patterns? Although Faria et al. do not discuss possible answers to this question, one possibility may be that local social-ecologic conditions determine sleep-duration choices. In social-ecologic conditions of threat and high mortality rates, it may be that sleep choices become biased to be more altruistic (people are required to reduce sleep to maintain vigilance during the night, etc.). Maternal-origin genes may be overexpressed in such conditions. If local conditions, on the other hand, are not threatening or harsh then sleep choices can become more selfish and paternal origin genes may be overexpressed. In short, the genetic conflict theory of sleep fundamentally explains why sleep must be "social" through and through.

6.3.6 Conclusions

In all of the foregoing theories of REM and NREM functions we have been assuming that the sleep state is doing something for the wake state; that is, that NREM SWS restores energy for waking consciousness, or that REM supports emotional memory consolidation for waking consciousness. But it is also possible that the functions of REM and NREM have more to do with the sleep states themselves than with waking consciousness. REM may be undoing something that NREM is doing, since REM typically follows NREM in the sleep cycle. Or conversely, NREM SWS may be doing something important for the organism (e.g., immune system repair), but that function is costly, so REM functions to complete, complement, repair, or undo something that NREM had to do to accomplish its primary functions. In this scenario, nightly SWS sleep repairs the immune system, but that is so onerous a job that NREM then requires REM to restore NREM's functional capacity so that it can do its immune system repair again the following night.

These sorts of scenarios raise fundamental questions about REM and NREM: How are these two forms of sleep related? Do they support or oppose

one another's effects? Are they antagonistic or complementary? Let us take the scenario just mentioned where REM undoes something or repairs something with NREM that NREM needs to accomplish its primary function (immune system repair). In this scenario, the wake state creates a need for non-REM sleep, and the expression of non-REM sleep (not something during wake) creates the need for REM sleep. In other words, the longer you are awake, the greater the intensity and duration of NREM will be, and likewise, the longer or more intense the NREM, the more intense the subsequent REM episodes will be. Delta waves index the intensity dimension for SWS, and eye movement density indexes the intensity dimension for REM. If REM indices are in reaction to or oppose those of SWS indices, then the need for REM sleep might be due to or related to the delta waves occurring in SWS during NREM. Benington and Heller (1994) noted that percentage of REM (during a twenty-four-hour period) was significantly associated with percentage of NREM, not percentage time spent awake.

If NREM sleep is homeostatically regulated, but REM need depends on NREM need, then the relationship between the two sleep phases should be partially but not entirely opposing. REM need should build up during NREM sleep, and NREM need should build up during waking. As the REM need accumulates during NREM sleep, it is more likely to intrude into waking state or interrupt the NREM episode. The drive to enter REM sleep – in other words, the magnitude of the need for REM sleep – would then be another indicator of the homeostatic regulatory process, which aims to maintain balance between the two sleep states. NREM sleep undoes something that occurs during waking life and REM undoes something that occurs during NREM in this theoretical approach to the two sleep states. In this scenario, however, REM is in service to NREM, while NREM is in service to waking consciousness.

Yet another theoretical possibility is that while NREM may be in service to the waking state (e.g., immune system repair), REM is not in service to NREM but actually opposes NREM physiology. This sort of scenario could happen if sleep states are influenced by evolutionary genetic conflict as discussed previously (see also McNamara, 2004). In this case, one set of genes would shape characteristics and functions of NREM, and a separate set of genes with opposing interests would shape characteristics and functions of REM (see Table 6.1). Tucci et al.'s (2016) work on the association of imprinted genes with REM partially supports this scenario.

REM sleep was discovered in 1953 and its association with dreams was discovered soon thereafter. Its role in the production of dreams has yet to be considered its primary functional role, but that is a function we will explore in another chapter.

6.4 Review Questions

- What is the theoretical significance of REM–NREM interactions?
- What are the strengths and weaknesses of the evidence that REM undoes something that occurs in NREM?
- Why are there only two major sleep states?
- Compare and contrast sleep and biobehavioral characteristics of Prader-Willi Syndrome and Angelmann syndrome.

Further Reading

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PART II

Dreams

CHAPTER SEVEN

What Are Dreams?

Learning Objectives

- Evaluate the strengths and weaknesses of the definition of dreams as sleep-dependent cognitions
- Identify formal properties of dream phenomenology and distinguish these formal properties from content of dreams
- Evaluate the significance of the role of emotion in shaping dream narrative form and content
- Evaluate story or narrative structure in creation of dream properties and phenomenology

7.1 Introduction

The most common definition of dreams among scientists appears to be that dreams are sleep-dependent cognitions (Table 7.1). They are thoughts and mental images that occur during sleep. If, however, dreams are sleep-dependent cognitions, then they require sleep if they are to occur. If REM sleep can erupt or invade daytime consciousness and REM-related mentation can occur with that daytime eruption, then we can get dreams or dreamy thoughts and images while awake. Indeed, that is what we call daydreams. Even if we cannot strictly claim that dreams are always sleep dependent, they nevertheless typically occur in association (even when daydreaming) with a brain state (REM) that is normally activated during sleep.

Since dreams typically occur during sleep, and since we claimed in our definition of sleep that sleep is "a restorative process that is brain state-regulated, reversible, homeostatic, embedded in both a circadian and social-physiologic organization and involving a species-specific quiescent posture, some amount of perceptual disengagement and elevated arousal thresholds"; then dreaming refers to the cognitions that take place within that biologic framework, that is, within a restorative process that is brain state–regulated, reversible, homeostatic, embedded in both a circadian and social-physiologic organization, and involving a species-specific quiescent posture, some amount of perceptual disengagement, and elevated arousal thresholds. That

Table 7.1 Hall/Van de Castle social interaction content ratios

Aggression/Friendliness percent	Dreamer-involved aggression/(dreamer-involved aggression + dreamer-involved friendliness)
Befriender percent	The percentage of all dreamer-involved friendly interactions in which the dreamer befriends some other character. Dreamer as befriender/(dreamer as befriender + dreamer as befriended)
Aggressor percent	The percentage of all dreamer-involved aggressions in which the dreamer is the aggressor. Dreamer as aggressor/(dreamer as aggressor + dreamer as victim)
Physical aggression percent	The percentage of all aggressions appearing in reports whether witnessed or dreamer involved that are physical in nature. Physical aggressions/all aggressions
Aggression (A/C) index	Frequencies of aggressions per character (all aggressions/all characters)
Friendliness (F/C) index	Frequencies of friendly interactions per character (all friendliness/all characters).
Sexuality (S/C) index	Frequencies of all sexual encounters per character (sexual encounters/all characters)

definition of sleep then tells us something about the cognitions that occur during sleep: They will occur within a brain state that is homeostatically regulated, reversible, constrained by circadian and social physiologic variables, and characterized by some amount of perceptual disengagement. In short, dreams will be products of the sleeping brain, will be sensitive to social factors, and will not be constrained by some forms of external perceptual stimuli.

We do not know if dreams themselves are homeostatically regulated, though they appear to be so. Individuals who are on medications that suppress REM sleep for long periods of time report less dreaming during that time and then abundant dreaming when the medications are discontinued. We do not know if dream rebound occurs with NREM rebound, but dream rebound does appear to occur with REM rebound. Similarly, when we experience sleep deprivation over a period of a few days, we normally experience very vivid and abundant dreams when we are allowed to sleep again.

Box 7.1 Dreams are sleep-dependent cognitions

-vivid images and increased emotion in dreams
-increase memory access during dream, so hypermnesia but decrease recall for dream after awakening (amnesia)
-mind reading
-visual sense predominates
-decreased taste
-decreased smell
-decreased pain, despite painful scenes

NARRATIVE STRUCTURE
-thematic discontinuities
-quick changes in plot
-occasional improbabilities (defy laws of physics)
-incongruities (plot elements don't fit together)
-characters can be real or fantastic
-lapse in self-reflectiveness
-hyper-creativity (mental simulation)
-scene is hyper-real
-effortlessly created and combined
-some problem-solving
-automaticity
-perceptual disengagement
-metaphor

DREAMWORK (Freud)/literary tropes (White)
-condensation: two figures resolved into one (metaphor)
-displacement: affect appropriate for one character is directed onto a more neutral characters (metonomy)
-symbolization: (metaphors are used to express meanings)
-secondary revision (irony)
-presentation (synecdoche)

This dream phenomenology is realized most completely in REM dreams and in N2 dreams when N2 occurs in the early morning hours. It is realized partially in N2 dreams and when N2 occurs in the first part of the sleep cycle. N3 dreams probabilistically realize a few or several of these characteristics depending on the time of night, circadian factors, and individual difference factors.

So, dreams are cognitions that depend on sleep and occur in sleep and presumably would not occur without sleep, but what do we mean by cognitions? As a first pass, the definition of the term cognitions includes everything that is part of

mental life: thoughts, mentations, affects, images, emotions, visual simulations, memories, etc. But we will immediately need to qualify this claim a bit because not all forms of mental activity occur regularly in dreams. Dream researchers have shown, for example, that instances of reading, writing, arithmetic, and reflective thought are relatively infrequent occurrences in dreams as compared to their frequency in waking life. And conversely, some forms of mental experience occur in dreams more frequently than they occur in waking consciousness. For example, Freud pointed out that some dreams allow us to engage in vivid hallucinatory wish fulfillment. If the child is hungry he will dream of some delicious foods. In addition, a long line of experimental research demonstrates that dreams allow us to more easily make disparate connections between otherwise unconnected concepts. That ability to make connections where the waking mind sees no connections underlies the ability of dreams to promote creativity. Similarly, dreams allow us, or better, force us, to interact with supernatural beings from monsters and demons to angels and gods, thus demonstrating that dreams involve a unique form of cognition that is probably related to spiritual and religious ideas. These are only a few examples of how dreams differ from the waking mind in terms of the cognitions they exemplify.

The philosopher Jennifer Windt (Windt, 2015) has argued that the phenomenal core and distinctive experiential properties of dreams can be captured by her immersive spatiotemporal hallucination or ISTH model of dreams. Dreams for Windt are essentially immersive spatiotemporal hallucinations. In her view, dreams involve a shift in the spatiotemporal location of the self from a veridical perception-based reference frame to a non-veridical hallucinatory reference frame. Windt's term "immersive" refers to the ways in which the experience of self is always indexical: The self inhabits a specific place and time relative to other selves or objects in the same reference frame. Dreams are immersive in that they involve the experience of spatiotemporal situatedness of the self. As in waking experience, there is a self as well as a world in dreams, and the boundary between the two is felt or taken for granted in dreams. In waking experience according to Windt, the situated self is related to a real word, whereas in the dream, the situated self is related to a hallucinated world. The immersive self need not be composed of a history, emotions, or memories for ISTH. It is simply a point in space and time relative to other such points. Windt is even willing to entertain the idea that the self in dreams can involve nothing more than an unextended point of awareness or a disembodied point or entity. She points to reports of lucid dreamers who claim to have experienced dreams without images or emotions. She also cites the experiences of Buddhist meditators who report the experience of absence of sense of self and the experience of

dreamers who cannot recount details of dreams but report "just knowing" that they dreamed, etc. For Windt, then, this minimal self, this point in an imagined space and time, relates to a fully hallucinated world, but this self might not form any beliefs about the hallucinated world it finds itself in. Doxastic belief formation is a problem for the dream self according to Windt. It is unclear whether Windt believes that the self itself forms the hallucinated world or some other structure in the dream forms the hallucinated world. She speaks in terms of the dream or the dreaming brain forming the hallucinated world. Dreams (not the self, apparently) integrate distal memory sources and internally generated imagery into a hallucinated world for the self to relate to. But the self in dreams does not form a fully fledged first-person perspective according to Windt, and therefore the self has difficulty forming beliefs or opinions about the dream world it inhabits. In fact, according to Windt, a cogitative self is prevented in dreams. Windt claims we need this account of the dream as an experience of a minimal self in a hallucinated world that does not construct and does not evaluate anything, in order to capture the core phenomenology of dreams, which involves an unstable, unreflective, and disoriented self as well as those rare accounts of dreams without images, emotions, or events.

Nir and Tonini (2010) are also impressed with how labile the sense of self is in dreams. For these authors, the self changes mostly in negative ways during the dream. For them, dreams are characterized by a sense of self that has reduced voluntary control during the dream (we supposedly cannot pursue goals in dreams), reduced self-awareness (we uncritically accept bizarre occurrences in dreams), reduced reflective thought (we typically do not know we are dreaming), heightened emotionality, and altered (mostly increased) access to memories during the dream despite being largely amnestic for the dream after awakening. But as both Nir and Tononi (2010) and Windt (2015) note, there are many exceptions to their rule of the impaired self in dreams. We can have dreams where we vigorously pursue and attain goals, where we have self-awareness and self-reflection (we deliberate about options and puzzle over bizarre encounters in dreams), and where we are appropriately emotional and not particularly amnestic about the dream upon awakening.

In fact, dreams are not mere chaotic assemblages of bizarre images that occur in the presence of a minimal and impaired self. Rather, dreams mostly involve perceptually and thematically organized material in the form of narratives containing appropriate images, themes, and simulations of the dreamer's life-world. Nevertheless, dreams are not like waking cognitions in all respects. The following cognitive characteristics are the key phenomenologic features of dreams (see Table 7.1).

7.2 Increased Emotions in Dreams

Emotions occur in virtually all dreams (Merritt et al., 1994). When using the Hall/Van de Castle scoring rules, negative emotions appear in about 80 percent of dreams for both men and women. Strauch and Meier (1996) reported that the emotions evidenced in dreams are appropriate to the action occurring in dreams. If sadness is reported then the dream scene contains a sad event, and when fear is reported the dream scene contains some scary events, etc. It is a remarkable fact that almost all dreams contain emotions, as not all waking states contain emotion and certainly not emotional states that are as intense as they typically are in dreams.

7.3 Hypermnesia within the Dream and Amnesia for the Dream

Hobson (1988) pointed out that when we dream, we sometimes have access to memories that our waking mind does not have access to – yet we are often amnestic for dream content when we wake up. Foulkes (1962) reported that non-contemporaneous images (images dating back to the person's more distant past) were more likely to occur in REM than in NREM dreams. Offenkrantz and Rechtschaffen (1963) and Verdone (1965) reported a relation between the date of memory images in dreams and time spent in sleep such that as the night progressed the dreamer was more likely to report older personal memories. So, although the dreaming mind/brain is digging through older memories as the night progresses, upon awakening the waking brain/mind often forgets that the dreaming mind/brain was doing all that memory rummaging during the night.

7.4 The Visual Sense Predominates in Dreams

Dreams are composed mostly of vivid visual images. Within a dream, the dreamer feels as if he or she is seeing a scene or a life-world. Touching, smelling, and tasting things in the dream-world do not occur as often as they do in waking life. Auditory experiences occur fairly frequently in dreams, but the visual sense predominates. Waking perception includes full color and three-dimensional views of a relatively stable world. In waking life, visual clarity varies with attentional focus of the individual and this is true also of dreams. Dreams exhibit greater clarity in the foreground of the dreamer's attention, while background details are vaguely represented. Unlike the full-color world of waking consciousness, up to 20–30 percent of dreams are achromatic. Dreams also contain a greater number of visual distortions and transformations than waking vision. For example, we may see an individual dream character's facial features change several times while we focus on those features in the dream.

7.5 Automaticity

Dreams come to us or happen whether we want them to or not. We cannot stop them and we cannot bid them to come anytime we like. They happen to us independently of our will. We can increase the chances of having a dream via certain ritual practices such as dream incubation (discussed shortly), but in general, dreams happen to us regardless of our wishes. We have dreams whether we want to or not. Dreams can therefore be construed as involuntary cognitive and symbolic events that occur during sleep and utilize perceptual and memory fragments to construct new narratives that more or less successfully simulate features of the dreamer's life-world.

7.6 Perceptual Disengagement

Virtually every scientist and scholar who has studied dreams has claimed that the key distinguishing mark of the dream is its lack of access to current perceptual information about the external world. That is why the philosopher Jennifer Windt and the neuroscientists Nir and Tononi believe that dreams are basically hallucinations and that the self in dreams is unreflective, minimal, and impaired. The self cannot check or correct its perceptions via use of incoming sensory information, and so it naively accepts what it sees as reality. In short, the common view is that the dreaming brain is totally cut off from the environment.

But it may be that the brain in REM is not simply dormant and fully disconnected from the environment (Hennevin et al., 2007). Thalamo-cortical gating of incoming sensory information is not complete during REM. During REM, evoked responses of thalamic neurons are only slightly attenuated compared with waking. Cortical neurons are still responsive to incoming auditory stimuli during REM sleep. Evidence gathered from event-related potentials (ERP) studies demonstrates that auditory discrimination, recognition of an intrinsically meaningful stimulus (e.g., the dreamer's own name), and categorization of stimuli are intact during REM. In addition, convergent evidence from behavioral and neuroimaging studies suggests that the neuronal patterns prevailing in thalamocortical systems, a burst-silence mode during SWS versus a sustained single-spike activity during waking and REM, are strongly modulated not just by subcortical sites in the brainstem, the hypothalamus, and the basal forebrain but in addition by widespread cortical networks, for example, the default mode network but including secondary sensory areas that process semantic and abstract attributes of sensory stimuli.

Although it is now clear that auditory information is processed during REM, it is also clear that the ways in which the dreaming brain processes this

information is different from the ways in which the waking brain processes the information. The processing of auditory information is slower and takes longer during REM as compared to the waking state. Brain responses to deviant tones are significantly larger during REM.

Auditory information is not the only external sensory information that maintains access to the sleeping brain during REM. Chemical, smell, somato-sensory, and kinesthetic senses all continue to be processed during REM. Indeed, the only modality that is dramatically attenuated during REM is the visual modality, but even here visual information is not completely abolished during REM. Ambient light energy is processed despite closed eyes, for example.

Fogel et al. (2022) demonstrated that people can even detect and cognitively follow a narrative when in REM sleep. They had participants attending (with eyes closed) to a rich auditory narrative that unfolds over several minutes. Because the paradigm requires no responses from the participant, they merely have to attend to the narrative; it is ideal for examining neural correlates of narrative processing across variations of consciousness, such as sleep-wake states. The authors employed simultaneous EEG-fMRI to investigate the extent to which complex processing of the auditory features of the stimulus was observed in all individuals in REM sleep (see Figure 7.1).

The authors found that no participants displayed brain activity in relation to the narrative stimulus when in any of the NREM sleep stages. Strikingly, however, one participant in REM showed specific activity in the auditory network when the suspense event in the narrative occurred. It is worth noting that methodologically speaking, obtaining fMRI data during REM is notoriously difficult. So this data is uniquely valuable.

7.7 Hyper-creativity

It is not true that dreams only utilize existing memory fragments to construct simulations and narratives. Inventories of the images and events in dreams reveal that dreams are composed mostly of images that have never before been encountered by the dreamer. Dreams, in short, are creative, productive, generative, and fecund. They are not mere reflections of waking consciousness, nor are they mere catalogs of floating memory fragments. They take specialized input by selective brain activation associated with REM, memory fragments, day residues, fleeting visceral sensations, other sources of content not yet characterized), subject that input to specialized processing algorithms using selective neural processes to do so, and then take the transformed imagery into the dream machinery system to output a unique cognitive product that we call the dream.

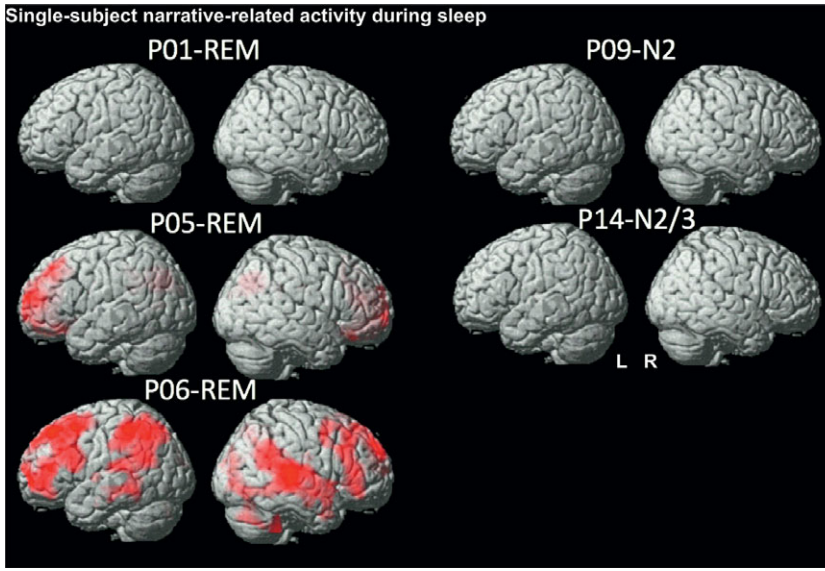

Figure 7.1 Single-subject narrative-related activity during sleep
(Significant brain activity ($p < 0.05$, FWE corrected) estimated in sleeping participants, who were presented with auditory content from the movie*Taken*, stimulus in the scanner, by the set of average suspense ratings from the awake group. The auditory and frontoparietal regions in one participant in REM (P06) and partially in another (P05) responded significantly to suspense throughout the narrative). Used with permission from Fogel et al. (2022)

7.8 Self-Reflectiveness in Dreams

Most dream researchers believe that self-reflectiveness is impaired in dreams. That means that the dreamer often accepts bizarre or incongruous dream events uncritically as if they were normal or not bizarre. The view that self-reflectiveness is impaired in dreams is supported by findings from neuroimaging studies that demonstrate downregulation of the dorsal prefrontal cortex during REM sleep. On the other hand, some investigators have pointed out that these same neuroimaging studies show clear activation of other structures known to participate in high-level evaluative and reflective processing such as the ventromedial prefrontal cortex (especially area 10). In addition, the P300-evoked potential wave is intact during REM. The P300 is elicited in the waking state whenever high-level attentional and evaluative processes are activated in response to unexpected events in the environment. It may be that levels of self-reflectiveness fluctuate considerably in the dream state, just as

they do in waking consciousness, but dreaming per se does not require impaired self-reflectiveness.

7.9 Mind Reading in Dreams

Can the dream-self read the "minds" of other dream characters – characters the dreamer himself has putatively created? You would think that since the dreamer conjured the other dream characters out of his or her own mind that the mental states of these characters would be transparent to him. It appears, however, that while the dreamer makes frequent theory of mind attributions to other dream characters, not all of their mental states are clear to the dreamer. McNamara et al. (2007) tabulated all clear references of the dreamer attributing mental states to other dream characters and found that theory of mind attributions were ubiquitous in dreams – especially REM dreams. Nevertheless, there were plenty of dreams wherein the dreamer behaved as if he did not know what other characters intended toward him.

7.10 Basic Ontology of the Dreamworld

The scientific gold standard taxonomy/ontology for dream content studies has been (for decades) the categories of the Hall/Van de Castle coding system (see review in Domhoff, 1996). Decades of research has established that its basic categories capture the essentials of dream content across cultures and demographic groups. Its coding rules and system have been validated in dozens of studies. The Hall/Van de Castle basic categories are as follows (canonical instances and definitions given in parentheses):

Characters (people, animals, mythical figures)
↓ engage in
Social interactions (aggression; friendliness and sex)
And engage in
↓
Activities (walking, talking, seeing, thinking, etc.)
And experience in those activities/interactions:
↓
Success and Failure (dreamer engages in and perseveres toward a goal and succeeds or fails in that pursuit)
And these characters can also experience /undergo
↓
Misfortune or good fortune (adverse or happy events happen to dreamer)
↓

Emotions (anger, apprehension, sadness, confusion, happiness) associated with those experiences
And all these events and interactions occur within:
↓
Settings (location, familiarity)
That also contain objects that a character notices or handles or interacts with
↓
Objects (architecture, household, implements, etc.)

Built on this basic taxonomy, Hall and Van de Castle derive the following social interaction ratios:

A major limitation of the Hall/Van de Castle system is that it is labor intensive, requiring hundreds of hours of manual coding of lots of dream reports. In order to circumvent this limitation, Bulkeley (2014) used word-search techniques to develop a word-count method that yields similar findings as the Hall/Van de Castle manual coding system for the following categories: perception, emotion, characters, social interactions, movement, cognition, culture, and natural elements.

In addition to the aforementioned Hall/Van de Castle and Bulkeley taxonomies of the dreamworld, some sleep/dream scientists would include two further categories for a complete dream ontology: cognitive processing and story structure.

On cognitive processing, McNamara et al. (2016) have used the text word–count program "Linguistic Inquiry and Word Count" to tally instance of words denoting cognitive processing. This cognitive processes category picks up words indicating insight, causation, discrepancy, tentativeness, certainty, inhibition, inclusion, and exclusion, etc. To supplement the main cognitive processing variable we added the function word category, which is made of words that function as grammatical markers (and, or, etc.). We thought that as narratives contain increased numbers of syntactical markers, so too would cognitive processing increase. Similarly, the verb category refers to "common" verbs such as walk, went, and see and includes verbs in all tenses, that is, past tense (went, ran, had), present tense (is, does, hear), and future tense (will, gonna). Not included under the verb category is auxiliary verbs (am, will, have). The verb analysis would capture additional evidence of grammatical processing, thus adding to confidence that cognitive processing was being accurately captured with these analyses. Using these word-count categories to capture indications that cognitive processing was occurring in dreams, the authors found that virtually all dreams contained an abundance of cognitive processing. Dreams appear to be doing significant cognitive work.

The last major category to characterize the typical dream world is story or narrative structure.

7.11 Narrative Structure

We saw in the previous section that REM seems so attuned to narratives that a person in REM can selectively attend to a narrative story and follow its events even when they are asleep within REM. Dreams unfurl like story plots or narratives. There is a beginning, middle, and end. The dreamer is typically at the center of the story. He or she is attempting to do something but faces obstacles or conflicts. Stories, including dream stories, tell us "who did what to whom and in what order." Remarkably, the dreaming brain creates narratives effortlessly, so it may be no exaggeration to claim that the dream's natural cognitive product may be a narrative.

REM dreams are better at creating narratives than NREM dreams are. Using a very detailed story grammar to score storylike structure in dream reports, Kuiken et al. (1983) reported that REM dreams exhibited more storylike structure than NREM reports did. Nielsen et al. (2001) reported that more stage REM than stage N2 reports contained at least one story element and a greater proportion of instances of episodic progression but only for late night reports of high frequency dream recallers. So both REM and NREM generate dreams as narratives, but REM does so more fluently. Nevertheless, the narratives that are generated often involve thematic discontinuities or disruptions in the story line and incongruous twists and juxtapositions. Some may feel that this makes for a better story, while others might deem these quirks as yielding the bizarre elements we all find in our dreams.

The dreaming mind/brain generates stories. Stories are not imposed on dream images after awakening. To construct those stories, the dreaming mind/brain very likely uses cognitive operations that can be described via Freud's "dreamwork" or by the four standard literary tropes that literary scholars claim are the engines that construct stories. For example, Hayden White (1999) has argued that people utilize the four major literary stylistic tropes metaphor, metonymy, synecdoche, and irony when cognizing their worlds, including their histories. To oversimplify White's rich tropological narrative philosophy of history: metaphor involves a comparison of materials with something familiar; metonymy involves breaking up those materials into parts; synecdoche involves reorganizing those materials into a new whole; and irony involves reflection on that new whole. White showed that these four tropes are effectively equivalent to Freud's dreamwork operations, including displacement (metonymy), condensation (metaphor), presentation (synecdoche), and secondary revision (irony). "The four operations identified by Freud function in the same way that the tropes do in allegory to mediate between the literal and the figurative levels of meanings of the text" (White, 1999, p. 103). A perusal of any corpus of dreams would yield an abundance of

instances of each of these four literary tropes, functioning to perform the dreamwork that results in dream narrative plots and structure.

It is currently unknown whether the narratives constructed during the dream experience are essentially confabulatory tales created "on the fly" by the dream itself in order to "explain" the strong emotions occurring in the dream. According to this view, dream narratives contain no inherent purpose or logic. They are constructed on the fly and after the fact, a chaotic assemblage of random images loosely strung together by an impaired cognitive system operating without the benefit of reflective thought. They are, to paraphrase Shakespeare, "tales told by an idiot and signifying nothing." If, however, the dream were a mere, after the fact, confabulation or rationalization for a series of decontextualised memory images or emotions, there would be no reason to expect consistent content across dreams. But decades of carefully conducted dream-content studies have demonstrated beyond reasonable doubt that dreams contain consistent content across groups and within individual dream series. Longitudinal analyses of dream series demonstrate clearly that dreams display intricate, thematically connected, consistent story lines, characters, events, emotions, and narratives across time.

7.12 Brain and the Phenomenology of Dreams

Hobson et al. (2000) (see Figure 7.2) offered a hypothesis on what brain networks were most important for support of a specific aspect of dream phenomenology. For example, they ascribed initiation of fictive movement in dreams and fictive movement itself to basal ganglia and cerebellum. They ascribed lack of insight to downregulation of the prefrontal cortex during REM. Activation of occipital and visual association areas supported visual scenes and so forth.

7.13 Conclusions

Dreams are cognitions that are typically dependent on sleep. However, not all forms of cognition occur during sleep. In spontaneously recalled dreams, the visual sense predominates. It is rare to remember a smell or a taste from the dream. Reading and computations (arithmetic) do not frequently occur in dreams. Many dreams contain unusual amounts of emotion and may provide greater access to older memories, especially during late-morning REM dreams. While impairment in critical self-reflective capacities may occur in dreams, it is not clear if all dreams are characterized by impairment in self-reflectiveness. The dreaming mind/brain spontaneously and automatically produces dreams in the form of narratives and likely uses cognitive operations like Freud's dreamwork to do so.

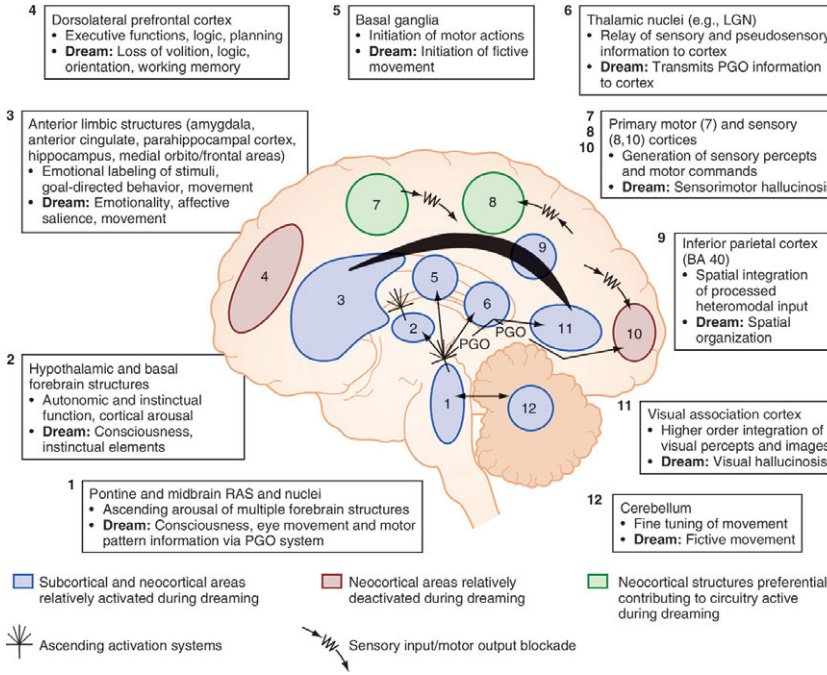

Figure 7.2 Cortical and subcortical areas in dreaming
(Areas 1 and 2, ascending arousal systems; area 3, subcortical and cortical limbic and paralimbic structures; area 4, dorsolateral prefrontal cortex; area 5, motor initiation and control centers; area 6, thalamocortical relay centers and thalamic cortical circuitry; area 7, primary motor cortex; area 8, primary sensory cortex; area 9, inferior parietal lobe; area 10, primary visual cortex; area 11, visual association cortex; area 12, cerebellum. BA40 Brodmann area 40, the temporoparietal junction, LGN lateral geniculate nucleus, PGO ponto-geniculo-occipital waves, RAS reticular activating system).
Used with permission from Pace-Schott, E. F., & Picchioni, D. (2017)

Theoretical models of the dream must be consistent with formal cognitive properties of the dream, including narrative structure, creativity, "mind-reading," hypermnesia within the dream, partial amnesia for the dream upon awakening, enhanced emotional levels for many dreams, enhanced visual (and to some extent auditory) impressions with diminished impressions from the other senses, and finally the involuntary nature of dreaming: We dream whether we want to or not.

These formal properties of dreams also make it clear that dreams are a different species of cognition than that which occurs in the waking state. While offline simulations do occur in the waking state, they are not obligatory and they do not typically involve intense emotion and are not narratively

structured. Daydreams, instead are focused on wishes, goals, and plans. They also are more often episodic and fleeting impressions rather than organized narratives. Instead, we should entertain the possibility that dreams are specialized products of the sleeping mind/brain. Dreams are one of the things that the sleeping brain is designed to produce: vivid, visually based, narrative simulations of our social worlds, with the dream self-interacting with other characters along a story line involving conflict and resolution.

7.14 Review Questions

- How is the sense of self (of the dreamer) typically experienced in dreams?
- How is mind reading (one dream character discerning the thoughts and intentions of a different dream character) possible in dreams, given that there is only one dreamer?
- What are the strengths and weaknesses of Windt's immersive spatio-temporal hallucination (ISTH) model of dreaming?
- What is the significance of the dream property "perceptual disengagement" for dream phenomenology?

Further Reading

Hobson, J. A. (1988). *The Dreaming Mind*. New York: Basic Books.

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CHAPTER EIGHT

Dreams across the Human Lifespan

Learning Objectives

- Describe how dreams of children differ from dreams of adults
- Evaluate the significance of the social content of dreams
- Evaluate the significance of the overlap in the neuroanatomy of REM with the neuroanatomy of the "social brain" network
- Describe basic dream content themes derived from the standardized Hall/Van de Castle scoring system

8.1 Introduction

Dreaming both reflects the stage of life in which we find ourselves and influences the character of that stage of life. For example, adolescence would not be adolescence without the wild, melodramatic, passionate, and swirling epic dreams teens have that center around those existential questions of "who am I" and "W\why am I here?," etc. We will see in this chapter that dreaming appears to reflect and perhaps promote waking social interactions of the dreamer, and this is true across the entire lifespan of the dreamer from toddlerhood right through to death. Of course, that is not the whole story. Dream content across the lifespan involves far more than social interactions, but it is a striking and consistent fact that simulations of social interactions are a constant feature of dream life throughout the life cycle. If we want to understand the development of dreams and dream content across an average human lifespan, we will need to first summarize the key milestones or periods of the human life cycle from the cradle to the grave. Table 8.1 displays key characteristics of each of the major human life-cycle stages and sketches corresponding dream findings.

8.2 Dreaming and the Social Brain

Why are dreams so filled with social content even in childhood? The human infant is immature neurologically at birth and thus is utterly dependent on caretakers for at least the first few years of life. That is one reason why securing "attachment" with caretakers is so crucial an aim for human children.

Table 8.1 Stage characteristics of the human life cycle

Stage	Period	Key Characteristics	Dream Life
Fetal life/ gestation	Nine-month pregnancy	Neurogenesis; fetal growth; fetal/ placental conflict	Signs of active sleep (AS) begin by second trimester and vary in response to placental changes/maternal activities. AS predominates throughout rest of pregnancy
Neonatal and infancy	Birth to end of lactation (typically 2–3 years old)	Most rapid rate of growth in body, brain, and behavior; language acquisition; attachment orientation established	First reported dreams in toddlers; static scenes of family members or animal characters; child believes dreams come from outside of self
Childhood	3–7 years	Moderate growth rates; 2nd order "theory of mind" and other social cognition skills	Frequent dreaming; clear representation of self in interactions with familiar others; occasional nightmares
Juvenile	7–12 years	Moderate growth rates; further development of social cognitive skills; peer group social interactions become important	Dreams become more elaborate; involve friends and unfamiliar characters in addition to family members; occasional nightmares
Puberty	12–16 years	Rapid growth rates; activation of sex hormones and secondary sexual characteristics; continued development of social cognitive skills	Elaborate vivid dreams; frequency of animals in dreams drops off; "wet" dreams (orgasm during dreaming); dreams of attachment or romantic objects

Table 8.1 (cont.)

Stage	Period	Key Characteristics	Dream Life
Adolescence	14–19 years	Rapid growth rates; growth spurt in height and weight; socio-sexual maturation; social cognitive skills now directed at self ("who am I?").	Elaborate dreaming; full panoply of characters (self, family, friends, strangers, romantic targets, etc.); increased physical aggression levels in male dreams and verbal aggression in female dreams
Adulthood	For women, from end of adolescence to end menopause; for men, from end of adolescence to senescence	Stable growth rates; sexual and reproductive activities; child-rearing	Elaborate dreaming around current concerns as well as counterfactual simulations of everyday social interactions; male/female differences in dream content
Senescence	Post-reproductive and child-rearing period	Gradual bodily and mental decline; generative social activities; grandparenting	Generative and reflective dreaming; scenes of loved ones living and dead
Death	End of senescence	Dissolution of bodily and mental activities	Spiritual, epic, and reflective dreaming; interactions with loved ones who have died

I discussed the role of REM sleep in attachment in previous chapters. Bodily and brain growth is rapid during infancy and followed by steady growth rates in childhood and dramatically increased growth rates again in adolescence. The major proportion of effort in growth goes to development and maintenance of brain function. Resting metabolic rate supports brain processes and development in upward of 85 percent of infants, 50 percent of five-year-olds, and 20 percent of adults. Why is all of this effort expended on brain development? While human culture and technologies have undoubtedly contributed to the need for greater resources to be funneled into brain development among humans, it also appears that large brains and prolonged development are required to succeed in complex societies.

Dunbar (1998, 2012) has articulated the "social brain hypothesis," which states that primates have larger than expected neocortical volumes (given body size) because they need to deal with the complexities of group or social life. Given neocortical volume in humans, Dunbar theorized that the average human social network should be comprised of about 150 individuals. Ethnographic and social evidence support this prediction. For example, this is the typical size of a hunter-gatherer group; of a company in a military organization; of a personal network (number of individuals a person knows directly); of a church congregation; of a small business company; and so on. Within this large personal network there are hierarchically organized subgroups; alliances, coalitions, and cliques, etc. that reflect differing degrees of familiarity with the individual at the center of the network. These coalitional alliances need to be maintained, updated, and renegotiated on a semi-constant basis by members of the alliance, all of which requires substantial cognitive resources.

Consider the cognitive demands that such a hierarchically organized network involving constantly shifting social alliances places on individuals. Human societies succeed to the extent that problems can be solved cooperatively. Cooperation is based on trust, and trust can only be earned over time and after many social interactions have taken place between the parties involved. Those social interactions have to be remembered, archived, and recalled repeatedly when evaluating current trustworthiness of one's potential partner in an enterprise. Maintaining the stability of relationships over time requires constant renegotiation of the terms of the cooperative agreement. People are capable of deception and thus, that capacity needs to be taken into account when weighing evidence of trustworthiness. It requires individuals to learn how to read the intentions or minds of others in the group so as to manage conflict, anticipate strategic moves of the other, and repair strained relationships and so on.

The capacity to appreciate that another individual has a mind like one's own, capable of cooperation but also of deception, etc., is called the "theory of

mind" or ToM capacity. Dunbar points out that this capacity involves several layers of cognitive complexity. First order ToM involves having knowledge of one's own mental states ("I believe that"). Second-order intentionality or tom involves knowledge of another person's mental states ("I believe that you understand that"). Third-order intentionality involves individual A thinking about what individual B is thinking about A's thinking ("I intend that you think that I think that we are going to") etc. Most scholars working in the area of social cognition think that human beings are capable of perhaps five orders of intentionality but no more. The computational demands on the brain for this kind of social cognition are considerable.

8.3 Overlap of the Social Brain with the REM Dreaming Brain

The areas of the brain that handle these computational demands around social cognition have been identified as the "social brain network." They are listed in Table 8.2. The amygdala is important for evaluation of the emotions of self and others. In addition, the amygdala helps to modulate hormonal levels important in social interactions such as oxytocin and vasopressin. Oxytocin has been called the trust molecule, as it enhances the level of trust and emotional closeness between people. Vasopressin appears to be crucial for social memories – especially in males. Its activity levels fluctuate in relation to testosterone activity. The fusiform gyrus supports rapid recognition and processing of faces.

Table 8.2 Overlap of social brain with default mode network

Key Nodes That Occur in Both Networks	Known Waking Functions
Amygdala	Emotion; salience evaluation
Fusiform gyrus	Face processing
Ventro and dorsalmedial prefrontal cortex	Self and other processing; theory of mind
Fronto–polar Brodmann area 10	Multitasking
Superior temporal sulcus	Mirror neurons
Temporal–parietal junction	Theory of mind
Insula	Empathy; moral evaluation
Posterior cingulate and precuneus	Self awareness; mental simulation; time traveling
Hippocampus	Memory processing

The face, of course, is crucial for social interactions, as it emits all kinds of signals concerning the intentions and emotions of the individual. The ventromedial and dorsomedial prefrontal regions are known to support processing of self-related information as well as understanding the mental states of others (i.e., tom tasks). The frontopolar region (BA 10) evidences a uniquely complex structure in humans and is one of the evolutionarily most recent regions of the brain in primates. It is involved in multitasking, working memory, and cognitive branching and therefore may support processing of 3rd and 4th, etc. orders of intentionality. The superior temporal sulcus contains mirror neurons that support social imitation behaviors and possibly emotional empathy. The temporal-parietal junction supports tom tasks and language processing. The insula supports empathetic responses as well as moral emotions, and the precuneus is involved in a range of activities from mental simulation to self-awareness. Finally, the hippocampus is involved in memory functions.

Many neuroscientists (e.g., Mars et al., 2012) have noted that this network of structures called the social brain overlaps to a significant extent with the so-called default mode network (DMN). The DMN appears to be transiently downregulated and then functionally reconnected during REM sleep. Dream investigators (see review in Pace-Schott & Picchioni, 2017) have noted that the dreaming brain during REM sleep is essentially composed of the reactivation of all of the key nodes in the DMN. It therefore appears that the dreaming brain is also the social brain. The DMN activates in a resting state when the mind is free to wander. It appears that what most people daydream about are social interactions. It should not be surprising then that night dreams, including dreams right across the life cycle, are concerned primarily with social interactions.

The theme of social concerns occurring in dreams begins right at the birth of dreaming in toddlerhood. In a previous chapter, we noted that human developmental schedules were prolonged relative to other apes, in order to enhance brain growth. The need to prolong developmental schedules and brain growth was fueled by the increasing complexities of human social life. The child needs to form stable attachment bonds with caretakers in order to survive and thrive during the long developmental phase of childhood. Dreams in children facilitate this process of attachment formation in multiple ways. Current cognitive models of the attachment formation process postulate "internal working models" of self (child) and the attachment target (e.g., mother) as key regulators of attachment formation and maintenance. Internal working models are constructed unconsciously and in dreams. In addition, children dream often of those people most important to them, such as family members and close friends. They try out all kinds of alternate scenarios of interaction with these people in their dreams.

In their review of methodologies used in research on children's dreams, Sandor et al. (2014) suggest that results from studies conducted in the child's

familiar home environment or school setting are richer in terms of representations of social interactions between the child and significant others when compared to dreams obtained from children in a sleep lab with EEG equipment and measurements. This is not surprising given that children are uncomfortable away from home and in a lab. Thus, we will focus on dreams collected in the familiar home or school environment in our review of children's dreams. There is reason to trust that children honestly share their dreams and don't make them up to please parents, etc. Children as young as three years old know what dreams are and can differentiate them from waking fantasy, events, and stories. When working with children in dream studies there are established criteria that can increase confidence that the narrative the child produces in response to a request for a dream are credible (see Table 8.3). We can therefore obtain reliable dream reports from children, just as we can from adults.

Table 8.3 Hall/Van de Castle norms on male and female dreams

	Male Norms (%)	Female Norms (%)	Effect Size	P
Characters				
Male/female percent	67	48	+.39	.000**
Familiarity percent	45	58	–.26	.000**
Friends percent	31	37	–.12	.004**
Family percent	12	19	–.21	.000**
Animal percent	6	4	+.08	.037*
Social Interaction Percents				
Aggression/friendliness percent	59	51	+.15	.014*
Befriender percent	40	47	+.06	.517
Aggressor percent	40	33	+.14	.129
Victimization percent	60	67	–.14	.129
Physical aggression percent	50	34	+.33	.000**
Social Interaction Ratios				
Aggression/character index	.34	.24	+.24	.000**
Friendliness/character index	.21	.22	–.01	.852
Sexuality/character index	.06	.01	+.11	.000**

Self-Concept Percents				
Self-negativity percent	65	66	–.02	.617
Bodily misfortunes percent	29	35	–.12	.217
Negative emotions percent	80	80	+.00	.995
Dreamer-involved success percent	51	42	+.18	.213
Torso/anatomy percent	31	20	+.26	.002**
Other Indicators				
Physical activities percent	60	52	–.38	.000**
Indoor setting percent	48	26	–.26	.000**
Familiar setting percent	62	79	–.38	.000**
Percent of Dreams with at Least One				
Aggression	47	44	+.05	.409
Friendliness	38	42	–.08	.197
Sexuality	12	4	+.31	.000**
Misfortune	36	33	+.06	.353
Good fortune	6	6	+.02	.787
Success	15	8	+.24	.000**
Failure	15	10	+.17	.007**
Striving	27	15	+.31	.000**

Note. The p values are based on the formula for the significance of differences between two proportions. The effect size derives form Cohen's h. The h statistic is determined by the following formula: $h = \cos^{-1}(1-2P_1) - \cos^{-1}(1-2P_2)$ P_1 and P_2 are proportions between 0 and 1, the $\cos^{-1}$ operation returns a value in radians. *significant at the .05 level **significant at the .01 level; Table 8.3 originally published as table 3.2 on page 73 of Domhoff, G.W. *The Scientific Study of Dreams*. Used with permission from Domhoff.

8.4 Children's Dreams and Nightmares

There have been remarkably few well-controlled studies of children's dreams. From sporadic published reports on children's dreams it appears that children can report dreams as soon as they start to speak. When studied in the lab, most kids' dreams until about middle childhood are said to be relatively static with

Box 8.1 Establishing the credibility of children's dreams

How do we know when a child is sharing a true dream versus some waking fantasy made up on the spot? Colace (2010) and Sandor et al. (2014) suggest the following guidelines:

(1) The child starts the report without hesitation
(2) The child reports the dream quickly in one go (although fulfilling these criteria could be difficult even for an adult when it comes to the recollection of possibly fragmented or bizarre dream content)
(3) The child's self definition of the story as a dream
(4) The placement of the experience itself during sleep period
(5) The coherence between the dream report and certain daytime experiences of the child in connection with the dream
(6) Good comprehension of the dream experience
(7) Consistency between the specific dream report and the general concept of a dream
(8) The last point introduces a new channel of storytelling through drawing, which should be consistent with the verbal channel, even after a certain time lapse

simple plot lines, some interactions with family members, and lots of animals. When, however, parents collect the dreams of their children, we get a different picture: Children's dreams seem to be just as dynamic and rich as adult dreams (but there are indeed more animal characters in children's dreams).

When Foulkes (1982) launched the longitudinal study of children's dreams in the 1970s he collected most of these dreams in the sleep lab; therefore, his results and analyses of the content of these dreams were colored by the lab setting. Foulkes found that young children recall very few dreams, and when they are recalled, they are static, may not contain representation of the self, and not much happens. Home-collected dreams, however, told a different story. Resnick and colleagues (1994) found no difference in dream recall frequency between four- to five- and eight- to ten-year-old age groups (56 percent and 57 percent, respectively). Nor did they find significant differences in active self-representation between four- to five- and eight- to ten-year-old (89 percent) age groups. The child was an active presence in up to 85 percent of their dreams and self-character was virtually always depicted in interaction with others in the dream story. Oberst (2005) found that boys tend to dream more about male characters (other boys his age), whereas girls dream equally often of boys and girls. Boys' dreams involve greater levels of physical aggression and

aggressive interactions with other characters (aggression/character index: 61 percent for boys and 24 percent for girls). Younger children express higher victimizations (where dreamer was the target of an aggression in the dream) than older children.

Strauch (2005) found a gradual appearance of active self in late childhood dreams and a dramatic increase in social interactions. Overall aggression/friendliness percentage declined for boys (70 percent at nine to eleven years old) and increased for girls (36 percent at nine to eleven years old) until the two plateaued at around a 50 percent aggression/friendliness percentage during the teenage years. Colace (2010) found that 68 percent of the dreams of young children (three to seven years old) contained an active self in an overall sample of home- and school-obtained dreams. Resnick et al. also found that the most frequent characters in young children's dreams were family members (29 percent of all characters) and other known children (28 percent).

Children's dreams, whether collected in the lab or at home, contain a lot of negative emotions and are often described as scary for the children. Children are more likely to experience nightmares than adults. Up to 50 percent of children between three and six years of age, and 20 percent between six and twelve years, experience "'frequent" nightmares. Persistence of nightmares during the preschool and school years (two and a half to nine years of age) is associated prospectively with psychotic experiences at twelve years of age. This association holds regardless of family adversity, emotional or behavioral problems, IQ, and potential neurological problems. Questionnaire–based nightmare and bad dream studies typically show that nightmare frequency is highest between the ages of five to ten and is related to other sleep disorders, trait anxiety, emotional problems, and behavior problems.

8.5 Adulthood: Male versus Female Dreams

The dream of adults are also intensely social in terms of their content. Adult men and women dream about their family members and close friends. They engage in a huge variety of social interactions with these close familiars. When strangers (unknown characters) appear in the dreams of children or adults, they typically indicate threat to either the dreamer or one of his close family members. Threat and aggression levels vary according to gender, with males evidencing greater levels of aggression and females evidencing greater feelings of threat (coming from strangers) in their dreams. Cross-sectional studies have documented gender differences in adult dream content. In the 1960s, Hall and Van de Castle studied the content of five dreams of each of 100 male and 100 female college students (N = 1,000), which had been collected between 1948 and 1952 (reviewed in Domhoff, 1996). They found (Table 8.3) that

unfamiliar, outdoor settings were present more often in men's than women's dreams, and that there was a higher proportion of male dream characters, unknown characters, more physical aggression, weapons, and sexuality in men's dreams. These basic cross-sectional content differences between the dreams of men and women have largely been confirmed in more recent studies.

Longitudinal studies of dream content changes in adults are rare. In examining the longitudinal effects of a cognitive processing variable on other dream content variables in dream series collected from thirty-seven men and forty-six women, McNamara et al. (2016) found that on a month-by-month basis, cognitive processing is significantly associated with markers of grammatical complexity (verbs and function words), the personal pronoun I, social processes, perceptual processes, and health and emotion (both negative and positive). All of this indicates that dreams are being used by adults to cognitively process information related to social interactions. The rate of change on a monthly basis for cognitive processes differed significantly for men versus women. Men exhibited a significantly positive rate of change in cognitive processing on a monthly basis, while women did not show a significant rate of change over time. Moreover, the difference in the rate of change in words denoting cognitive processing differed significantly in men compared to women. Pulling these results together suggests that people do in fact use dreams to "work through" or cognitively process selected types of social emotional information and the rate at which they do so appears to increase, at least for men. The topics subject to or associated with cognitive processing in dreams appears to be concerned primarily with social processes rather than health or perceptual processes. Why might men evidence increasing rates of cognitive processing around social information over time while women's rates of cognitive processing remained relatively constant over time? It cannot be due to differences in frequencies of baseline content variables, as we adjusted for baseline frequencies of words denoting cognitive processing in our analyses. Nor can it be due to age differences among males versus females, as we also adjusted for age in our analyses.

The continuity hypothesis on dream content states that dream content largely reflects waking-life. The continuity hypothesis is broadly supported by empirical evidence. It may be that women find it easier during waking lives to process emotional content, while men prefer to process socio-emotional content "offline."

Results from our analyses provide partial support for continuity but also raise significantly new issues regarding the way men and women use dreams to process emotional concerns. Men appear to engage in increasing amounts of cognitive processing in their dreams around socio-emotional information over time than do women.

8.6 Attachment Dreams

Both dream recall and dream content varies significantly in both children and adults as a function of attachment status or attachment security/insecurity (see for example, McNamara et al., 2014 and review therein). People who self-report an insecure attachment orientation tend to recall dreams that reflect their attachment orientations either in a compensatory manner (where people with anxious orientation recall more dreams focused on romantic targets) or in a reactive manner (where people with an avoidant orientation tend to not recall dreams or report dreams without affective or romantic content). Selterman and his associates (Selterman, Apetroaia, & Waters, 2012) examined partner-specific attachment representations/cognitive working models in dreams that contained significant others. Attachment orientation was measured using the "Secure Base Script Narrative Assessment" technique. The secure base script assessment takes participant responses to word cues as well as free narrative responses and then codes them for elements/scenes where the romantic target or secure base figure supports the participant's exploration, or comes to the aid of the participant, or comforts the participant, etc. Selterman and colleagues assessed their participants on this secure base script and then coded participant dreams for secure base script elements. They assessed sixty-one undergraduate students, all of whom were in committed dating relationships of six months duration or longer. Selterman and colleagues then collected two weeks of dreams from all these participants and coded all those dreams that contained a romantic partner for secure base script elements.

Results revealed a significant association between relationship-specific attachment security and the degree to which dreams about romantic partners followed the secure base script. Secure base content was identified in a significantly large proportion of dreams that contained current romantic partners. In addition, daytime attachment security as measured by the objective secure base script narrative task was significantly associated with "scriptedness," or the degree to which dreams were judged to reflect the secure base script.

These studies of dreams and attachment orientations suggest that one thing that dreams support is construction, maintenance, and adjustment of cognitive working models of attachment. As discussed in earlier chapters, those working models includes representations of the self in relation to the attachment object such that if both self and other (attachment object) are cognitively evaluated and represented in a positive manner, then we get secure attachment; if on the other hand the other is valued above the self, and the self is seen as too dependent and needy vis-à-vis the other, then an insecure and anxious preoccupation attachment orientation is formed. If the self is overly valued vis-à-visthe other, then we get an insecure avoidant orientation and so forth. McNamara et al. (2001)

showed that dream recall rates varied significantly with these attachment orientations. In addition, McNamara et al. (2001) found associations between attachment orientation, dream recall rates, and image intensity in dreams. McNamara, Pace-Schott, Johnson, Harris, and Auerbach (2011) found that people classified as anxiously attached evidenced reduced REM latencies and were more likely to have dreams containing themes of aggression and self-denigration compared to people with other attachment styles. Mikulincer, Shaver, and Avihou-Kanza (2011) reported similar findings regarding associations between insecure attachment and negative self-concept in dreams. Mikulincer, Shaver, Sapir-Lavid, and Avihou-Kanza (2009) found that both attachment-related avoidance and anxiety correlated with less dream content denoting secure attachment such as less support seeking, less support availability, and less distress relief in dreams. As mentioned previously, Selterman and Drigotas (2009) found associations between attachment insecurity (avoidance and anxiety) and conflict in dreams that contained romantic partners. Selterman et al. (2012) later reported that participants classified as secure in their current relationships tended to report dreams that contained more secure base content. Selterman, Apetroaia, Riela, and Aron (2014) later demonstrated that attachment-related dream content influenced daytime attachment behaviors. Specifically, they found that the frequency with which participants reported dreams about their romantic partners was positively associated with the extent to which they interacted with their partners and felt more love/closeness on days subsequent to dreaming about them. When people high in attachment avoidance had greater negative affect in dreams of their partners, they reported interacting less with their partners on subsequent days. For those high in interdependence, having a dream containing sexual behavior with one's partner was associated with increased love/closeness on subsequent days. In a rare EEG study of dream content across the night, McNamara et al. (2014) found that a variable capturing comfort with and actual "emotional intimacy" content increased over the course of the night as REM amount increased. While REM-dependent intimacy content tended to increase for all three attachment orientations, the rate of increase (slope) was slower for the avoidant group (0.31) relative to both the secure (0.52) or the preoccupied (0.44) groups.

8.7 Changes in Dream Content with Age

The dreams of elderly individuals have not received as much attention as the dreams of children or young adults, but they are just as important for the science of dreams. That is because as we age, slow wave sleep starts to drop out of the sleep cycle and we are left only with N2 light sleep and some REM. Thus, REM dreams no longer need to respond to or be constrained by the need

for slow wave sleep. Dream simulations should therefore be longer, more free, and wide ranging. The dreams of the elderly do appear to include a wider range of themes than the dreams of younger people, though the central theme of social interactions remain a constant – even in the dreams of the elderly.

Dale et al. (2017) reported that in a group of 231 males aged twelve to eighty-five, aggressive social interactions between the dreamer and other characters in the dreams tended to decline with age, while friendly interactions, total number of characters, and gender representation among the characters tend to remain stable over the lifespan. However, one caveat with these overall trends was the total absence of sexual content in the dreams of the last two age groups (40–64 and 65–85). This same research group reported similar findings (Dale et al., 2015) with respect to the dreams of women across the lifespan. There were no significant trends for either total characters or male characters in the dreams of women across the lifespan. But female and familiar characters decreased across the lifespan. There was a slight decrease (linear trend) with age for both total aggressive interactions and dreamer as victim interactions; a decline for total friendly interactions and the F/C index in the dreams of women from adolescence to old age.

Domhoff (2003) was given access to the dream journals of "Barb Sanders," a woman in her late fifties. Sanders had kept records of her dreams covering the years from 1970s to the late 1990s. In addition to being able to interview Barb Sanders herself, Domhoff was also able to interview four of Sanders' close female friends who had known her for many years. Thus, Domhoff was able to score the content of Sanders' dreams and study how that content changed over time and the extent to which that content reflects themes in Sanders' own life. Results showed that Sanders' dreams pretty faithfully simulated her current and past social interactions as well as desired social interactions, including those with her ex-husband, her ambivalent feelings toward her mother, her strong love for her favorite brother and her friends and children, a momentary romantic infatuation with a younger man, etc. Levels of aggressiveness versus friendliness in social interactions were remarkably stable in Sanders' dreams across decades, while the quality of social interactions with the significant people in her life changed over the years with depictions of some relationships becoming more friendly and others more distant. We will see that dreams of loved ones remain a constant feature of dream life even into old age.

8.8 Death and Dreams

Dreams have been collected from individuals who are about to die. Early studies found that the themes of dreams of the dying contained greater

numbers of supernatural agents, otherworldly settings, and images that seemed to "announce" the approach of death such as uncanny tunnels, wilting plants, and natural disasters. Some dreams seemed to ease the transition from this world into whatever awaits us after we die. Dreams of pregnancies, babies, and children appear frequently in those who are aware that they are dying. Scenes of trees bearing fruit; doors opening onto a light-filled path, and meetings with angelic and benevolent beings have been reported in the dreams of the dying. Dreams of reunions with a loved one who has died are common in all parts of the world.

8.9 Conclusions

Most dreams are filled with social interactions between the dreamer and familiar people in the dreamer's life. Dream content changes significantly according to the stage of life of the dreamer, but social interactions remain a constant in dream content across the lifespan. Much of the social interactions that occur in dreams can be characterized as attachment interactions, that is, interactions that reflect and help shape daytime attachment orientations (romantic attachments or familial attachments, etc.) of the dreamer. In old age and in death, dreams continue to simulate social interactions but new unfamiliar characters enter the dreams of the old and dying. These are supernatural beings but also include images of loved ones who have previously passed away. And so dreams that carry the child into the social world of his caretakers during early life gently escort the dreamer into the arms of his loved ones when the dreamer's time to leave this life arrives. Dreams accompany us and our loved ones, literally from the cradle to the grave.

8.10 Review Questions

- What is the significance of the experimental findings concerning associations between attachment orientations and sleep/dream measures in both children and adults?
- What kinds of evidence will increase confidence that children are really sharing their dreams rather than simply making up stories?
- Why do you suppose women dream equally often of females and males, while males dream more often of other males?
- What kind of experimental evidence would demonstrate that dream content influences daytime behavior rather than the other way round (daytime events influencing dream content)?

Further Reading

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CHAPTER NINE

Characteristics of REM and NREM Dreams

Learning Objectives

- Describe typical content of dreams associated with awakenings from NREM
- Describe typical content of dreams associated with awakenings from REM
- Describe interaction of REM–NREM dreams across a single night of sleep
- Describe role of dream content in emotional regulation

9.1 Introduction

While REM sleep is the phase of sleep from which dream reports are most reliably elicited, dream reports can also be elicited from any other stage of sleep, including sleep onset and N3 SWS. Reports after awakenings from NREM stages of sleep tend to be shorter, less emotional, and less visually vivid than reports obtained from REM – but we do get reports of "dreams" from NREM sleep states. Indeed, you can get dream reports without REM when REM is chemically suppressed via antidepressant medications. Just as dreams can occur without REM, REM sleep can occur without dream reports. About 20 percent of awakenings after a REM episode results in no dream report. Activation of REM, therefore, does not necessarily eventuate in a dream or at least a dream report. In addition, children who have abundant REM do not consistently report dreams until visuo-spatial and cognitive skills have matured enough to support reporting of visual narratives (Foulkes, 1982). Similarly, patients with lesions in the orbitofrontal cortex, basal forebrain, and near the occipitotemporoparietal junction sometimes report complete cessation of dreaming (Solms, 1997). In addition, Solms emphasized that disconnection of the ascending meso-limbic-cortical dopaminergic tracts from their termination sites in ventromedial frontal lobes could also lead to the loss of dreaming. Given that this tract is associated with instinctual appetitive drive and motivational states, it seems reasonable to conclude that this dopaminergic system may participate in the generation of some dreams. The loss of dreaming in these patients is not due simply to an inability to recall dreams, as their basic memory and recall abilities are largely intact. REM physiology as measured by sleep EEG is normal in these individuals, thus REM is still operating.

So, you can have activation of REM and no dreams, and you can have dreams without activation of REM (as in the case of NREM dreams). On the other hand, REM is the brain state that most reliably produces what human beings have for centuries called dreams. And REM dreams are somewhat different from the "dreams" obtained after awakenings from NREM sleep states.

If you awaken someone at any point in the nightly sleep cycle and ask them "were you experiencing anything just now while asleep?," they will often (though not invariably) report some sort of mentation. At the transition between wake and sleep when persons enter the N1 stage, they will likely (60–90 percent of awakenings) report vivid bizarre images, some emotion, and vivid characters, but they will not likely report dramatic scenes or story lines involving these characters. At N2, early in the night people generally report (about 40 percent of awakenings) vivid characters who interact with other characters and the dreamer, some emotion, and some plot, though the emotional intensity and story lines are not as intense or well defined as reports derived from REM sleep awakenings. N2 awakenings later in the morning hours, when REM sleep predominates, are associated with longer mentation reports with more intense emotions and more clearly defined story lines. When subjects are awoken from stage N3 or slow wave sleep they often report no mentation at all but occasionally (20–50 percent of awakenings) will report static scenes or thought-like mentation, disconnected memory fragments, and the like. However, when people are awoken from REM, at least 80 percent of the time they will report vivid "dreams" with intense emotions, vivid imagery, long well-defined story lines, dramatic social interactions, and occasional bizarre imagery. In short, early night mentation reports mostly from NREM stages are shorter, less vivid, less bizarre, less storylike, with differing social interactions than mentation reports obtained from sleep states occurring later in the sleep cycle (mostly from REM but also some N2).

What might account for the qualitative differences in sleep mentation that occur across the sleep cycle? Some scholars suggest that the two mentation types are generated by two different brain regions. This is called the two-generator model (Hobson et al., 2000). Alternatively, others (e.g., Antrobus, 1991) argue for a one-generator model with one brain network continuously generating mentation throughout the night, with differing physiologic and cerebral activation levels determining how much of that mentation can be recalled at each point in the sleep cycle. A third model, dubbed the "covert REM sleep" model suggests that while sleep mentation is tightly coupled to REM sleep processes, periods of NREM sleep can generate "dreams" or mentations to the extent that those periods borrow from REM sleep brain networks. For Nielsen, covert REM sleep processes at sleep onset (N1) and during NREM sleep in the early morning hours that occur near REM sleep

episodes produce the mentation associated with these sleep stages. In his review of REM versus NREM awakenings studies, Nielsen (2000) showed that if the NREM awakenings occurred toward the morning (when REM sleep predominates), then the NREM reports become harder to distinguish from REM reports. Another model of dream generation is Solm's motivational reward theory wherein dream generation is tied to mesolimbic prefrontal dopaminergic activity. This is a reward circuit, so Solm's hypothesizes that this mesolimbic-prefrontal dopaminergic activity supports the simulation of hallucinations (mediated by posterior cortical sites) of wish fulfilment during the dream.

Thus, in one generator theories, and in Nielsen's covert REM theory of dream generation, REM activation levels are crucial for dream generation. Dreaming depends on REM according to one generator model. But what happens to dreaming if you eliminate REM? People on antidepressants report reduced levels of dreaming but not a complete elimination of dreaming. Oudiette et al. (2012) found that long, complex, and bizarre dreams persist even after pharmacologically suppressing REM sleep, either partially or totally. Indeed, while NREM "dreams" are indeed less bizarre, complex, and storylike than REM dreams, they nevertheless still contain abundant social interactions, dramatic scenes, and intense emotions.

McNamara et al. (2005) investigated potential REM versus NREM content differences by studying 100 REM, 100 NREM, and 100 wake reports that had been collected in the home from eight men and seven women using the "nightcap" sleep/wake mentation monitoring system. The nightcap system reliably identifies episodes of REM and N2 NREM. We scored these reports for number and variety of social interactions. We found that (1) social interactions were more likely to be depicted in a dream than in wake reports; (2) aggressive social interactions were more characteristic of REM than NREM or wake reports; but (3) dreamer-initiated friendliness was more characteristic of NREM than REM. It is important to note that dreamer-initiated aggressive interactions were reduced to zero in NREM dreams, while dreamer-initiated friendly interactions were twice as common in NREM as in REM. It is therefore apparent that the lack of aggression in NREM was not due simply to fewer social interactions occurring in NREM relative to REM, as friendly interactions in NREM were more likely to be dreamer initiated than in REM (90 percent vs. 54 percent, respectively, $p < .05$). This fact, along with the total absence of dreamer-initiated aggression in NREM, suggests that an active process is operative in NREM that inhibits unpleasant and aggressive social impulses while promoting the emergence of pleasant and cooperative social impulses. Conversely, REM appears to facilitate the emergence of unpleasant aggressive impulses. We replicated

these results in a 2010 study (McNamara et al., 2010) using EEG-verified REM and NREM awakenings instead of the nightcap method.

9.1.1 NREM–REM Content Differences: Evidence from Dream Enactment Studies

Uguccioni et al. (2013) report some hard-to-get, unique data on enacted dreams, or dreams that patients appear to enact while asleep. The patient may start to vocalize while asleep as if talking to someone and then thrash around as if fighting some opponent, etc. The patient may actually get out of bed and run around the room as if trying to escape from a threat and so on. These types of dreams typically occur as part of the symptom complex associated with REM behavior disorder (RBD) or with sleepwalking or sleep terrors. Enacted dreams are uniquely important for the science of dreams because they give us a very unusual glimpse into dream content. Here we can see dreams being enacted as if on a stage while the patient is asleep and dreaming. We do not have to rely only on the patient's verbal report of a dream after he awakes.

In this study of enacted dreams, the investigators were interested in, among other things, the question of whether the content of REM dreams differed from the content of dreams that occurred in other stages of sleep – NREM dreams. The investigators could get at REM dreams via analysis of enacted dreams reported by the RBD patients and at NREM dreams via analysis of the enacted dreams reported by the sleepwalkers/sleep terror patients who typically enact their dreams in NREM stages of sleep. Thirty-two patients with sleepwalking/sleep terrors (SW/ST) and twenty-four patients with RBD were consecutively recruited and interviewed concerning their enacted dreams. These were dreams experienced during a night of sleep videosomnography as well as recent and lifetime dreams involving parasomniac events and enacted dreams. A total of 121 dreams were analyzed in both groups (sleepwalking group, n = 74 dreams; RBD group, n = 47 dreams). Dream content was then scored using primarily the standardized Hall versus de Castle criteria and Revonsuo's threats scale. There was a significantly higher level of aggression in RBD subjects than in SW/ST subjects, but these aggression levels were not correlated with daytime waking behaviors. Sleepwalkers had more frequent misfortunes in their enacted dreams than patients with RBD (28 percent vs. 8 percent, $p = 0.01$) and tended to exhibit less frequent aggression (17 percent vs. 33 percent, $p = 0.06$) during the enacted dreams. In dreams with aggression, both RBD and sleepwalker/sleep terror (SW/ST) patients were more often victims than aggressors. In dreams with misfortunes, the sleepwalkers mostly fled from a threat/disaster, while patients with RBD counterattacked when assaulted. The

settings for SW/ST-enacted dreams tended to be familiar/domestic; the settings in RBD dreams were less familiar. In summary, aggression (such as counter-attacks) is rare (8 percent) in SW/ST enacted dreams but common (reported by at least 33 percent of dreamers) in RBD. Given that SW/ST sleep parasomnias are associated with a dissociated state between N3 sleep and arousal and that RBD is associated with REM sleep, it is reasonable to suggest that the enacted dreams reflect brain state differences in these two sleep states. As the authors point out in their discussion, content analyses of thousands of dreams from healthy participants reveal strikingly similar content differences for REM versus N2/N3 sleep dreams. It is a remarkable and now fairly well-established fact that REM dreams appear to specialize in simulation of aggressive interactions, while N2/N3 dreams specialize in simulation of nonaggressive and friendly interactions.

The authors suggest that their data is at least partially consistent with Revonsuo's threat-simulation theory of dream content (Revonsuo, 2000), which states that dreams simulate daytime threats and make us better able to handle those daytime threats because both SW/ST and RBD dreams appeared to have simulated threats to the dreamer, with the RBD patients reacting with aggression and the SW/ST reacting with flight or awakenings. But note that the threat-simulation hypothesis cannot explain the key question as to why the REM dreamers responded with aggression and the SW/ST with flight. In REM sleep there is some diminution of prefrontal inhibition on limbic brain sites. While this physiologic fact can account for the mechanics of the aggressive responses in REM dreams, it does not explain the question as to why such responses should occur while we are asleep in the first place. Why are limbic circuits disinhibited in REM in the first place? The dream content data suggests that the reason is that Mother Nature wants the organism to simulate aggression-related behaviors while asleep. But again, why only for REM dreams? If activating aggression circuits while asleep is adaptive, why not do so in NREM states as well?

9.1.2 The Interaction of REM and NREM Dreams

NREM dreams very likely interact with REM dreams over the course of a single night. After the discovery of REM sleep, several authors (e.g., Trosman, Rechtschaffen, Offenkrantz, & Wolpert, 1960; French & Fromme, 1964) suggested that dreams at the beginning of the night would announce an emotional wish or emotional conflict that dreams later in the night would then pick up and work with in an attempt to contain or resolve the emotional conflict. Offenkrantz and Rechtschaffen (1963) studied the sequential sleep patterns and dreams of a patient in psychotherapy for fifteen consecutive

nights. They noted that scenes from childhood memories never occurred early in the night but did occur on eight of the fifteen nights in dreams late in the night, after 4:30 A.M. They also noted that all the dreams of a night tended to be concerned with the same emotional conflict or a small number of such conflicts. They claimed that they found evidence that the organization of a particular dream depended on the results of the dream work of the preceding dream, such that dream wishes required less and less disguise as the night progressed. Rechtschaffen et al. (1963) studied sequential NREM–REM dreams within a single night in three subjects who had previously demonstrated good dream recall from NREM sleep. They found repeated instances of dream elements recurring throughout the dream sequence. For example, the image of a street corner appeared in the first NREM dream of the night. It later appeared as the place where the dreamer met a girl. Cartwright (1999, 2010) later discussed similar findings for emotional dream content continuity across a single night of sleep.

9.2 Characteristics of REM Dreams

9.2.1 Introduction

Dream recall after awakening from the REM state is associated with theta oscillations immediately preceding awakening (NREM recall is associated with alpha oscillations). Since theta activity in the hippocampus is associated with memory encoding more generally, it is not surprising that we have better and more detailed recall of dreams after REM awakenings than we do for dreams after NREM awakenings. Most REM dreams are of ordinary social interactions occurring in familiar settings, with the dreamer and other characters talking about something of personal concern to the dreamer. Nevertheless, dreams obtained from subjects after awakenings from REM are longer in word length, evidence a greater character density and more social interactions with higher levels of aggression; more emotions, emotional intensity, and emotional memory consolidation; greater and more cohesive narrative structure (though with significant thematic discontinuities and occasional bizarre elements); and a reduced level of self-reflectiveness than dreams obtained from any other sleep state. In a previous chapter we discussed how REM dreaming involves activation and reconnectivity of the key nodes in the default mode network (DMN) or that set of structures of the social brain that are active during daytime mind wandering. We noted how DMN structures and the so-called social brain network overlap to a considerable extent. Those overlapping set of brain regions, the DMN and social brain, are essentially the set of structures most consistently activated in REM dreaming.

9.2.2 REM Dreams Are Associated with Reduced Self-Reflectiveness

Virtually all REM dreams involve the self or the dreamer interacting with other characters, and this self is usually at the center of the action. Although there is debate as to whether the dream self should be described as a full-fledged agent, it is clear that the dreamer not only intends certain actions in dreams but that in many dreams this intention is actually striving toward a goal, and this striving helps to create the narrative structure dreams typically employ. Although dreamers should be considered agents in the full sense of possessing intentional states and deploying plans to achieve those goals, the dreamer is nevertheless unable to critically reflect on the actions and interactions he or she undergoes in the dream. Most "damming" in this respect is the dreamer's inability to know that he is dreaming and the related noncritical acceptance of bizarre dream events as "normal." This reduction in self-reflectiveness is associated with reduction in activity levels of the dorsolateral prefrontal cortex during REM.

9.2.3 REM Dreams Have an Unusually Large Number of Familiar and Unfamiliar Characters

In REM dreams, women dream equally often of men and women, but men dream more of other men than of women, and usually these other men are in physically aggressive interactions with the dreamer. This latter sex difference (women dream equally often of males and females while men dream about aggressive encounters with other men) is consistent with theories of sexual selection and mating strategies in humans: Males compete among themselves for access to females.

In a study of 320 dream reports from thirty-three adults, Kahn et al. (2000) reported that 48 percent of characters in dreams were known to the dreamer. The average report length was 237 words and contained an average of 3.7 characters. According to the Hall/Van de Castle norms, only about half of the characters in dreams are familiar to the dreamer. In the background of most REM dreams there lurks unidentified characters, people that the dreamer cannot identify. Nevertheless, the dreamer could often tell that the strangers or unidentified people in the dream were males. In some dream series, up to 80 percent of characters are unknown males who are vaguely threatening to the dreamer. In an early study of more than 1,000 thousand dreams, Hall (1963) reported (1) that strangers in dreams were most often male; (2) that aggressive encounters were more likely to occur in interactions with an unknown male than with an unknown female or a familiar male or female; and (3) that unknown males appeared more frequently in dreams of males than of females.

Domhoff (2003) has recently shown that when male strangers appear in the dream, the likelihood that physical aggression will occur in that dream far exceeds what would be expected on the basis of chance. Strauch and Meir (1996) reported that in about every third dream the dreamer encountered only strangers!

9.2.4 REM Dreams Have Greater Numbers of Social Interactions and Higher Levels of Aggression

The results just summarized concerning the greater number of unfamiliar characters who turn out to be male strangers in REM dreams as well as the statistical association between the appearance of male strangers and physical aggression against the dreamer, predicts that aggression levels will be higher in REM dreams as compared to NREM dreams. While overall aggression levels may not differ between REM and NREM dreams, when the dreamer is the initiator of an aggression, then aggressive interactions are far more commonplace in REM dreams as compared to NREM dreams – even when the length of the dream report is held constant. In 2010, McNamara et al. (2010) replicated the finding of higher aggression levels in REM versus NREM with a convenience sample of 64 healthy participants (28 males, 36 females; mean age = 20.89, SD 2.56 years). The authors studied their sleep EEG in the sleep lab and conducted REM versus NREM sleep awakenings. Key results are depicted in Figure 9.1.

When we look only at those dreams where the dreamer was directly involved in (e.g., initiating) a social interaction, then clear REM–NREM differences emerge. As in our previous study with the nightcap technology, we find (with standard EEG methods) that in dreamer-involved friendly interactions, the dreamer was the befriender in only 42 percent of REM sleep dreams (N = 24) but was the befriender in 71 percent of NREM sleep dreams (n = 14; p = .070). In dreamer-involved aggressive interactions, the dreamer was the aggressor in 58 percent of REM sleep dreams (N = 12) and only 29 percent of NREM sleep dreams (N = 17).

It is important to note that these REM–NREM differences emerge only when you look at dreamer-involved social interactions. Apparently, one has to take the point of view of the dream ego to understand what is happening in dreams. When we analyzed the entire REM/NREM dream set using the standard Hall/Van de Castle categories mentioned previously, we found that among the social interaction scales, REM sleep dreams were less aggressive and more friendly than NREM sleep dreams. Of aggressive and friendly social interactions in REM sleep dreams, only 37 percent (N = 38) were aggressive compared to 57 percent of interactions in NREM sleep dreams (N = 37;

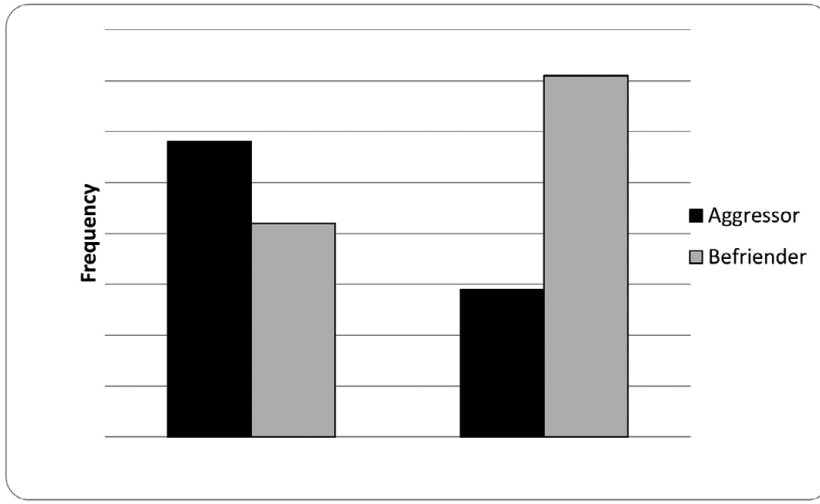

Figure 9.1 Frequency of dreamer's role in social interactions for REM and NREM dreams (This figure represents the dreamer's role in all dreamer-involved social interactions. There were thirty-eight REM dreams with dreamer-involved social interactions and thirty-seven NREM dreams. This means that in 71 percent of the dreamer's social interactions in NREM dreams, the dreamer was a befriender). Used with permission from McNamara et al. (2010)

p = .082). Of aggressive interactions in REM sleep dreams, 50 percent (N = 24) were physically aggressive (as opposed to verbal aggression or other types) as opposed to 43 percent of aggressive interactions in NREM sleep dreams (N = 23; p = .654). We believe that most of the N2 aggressions were verbal in nature and were from our female participants. However, looking at just dreamer-involved social interactions, the dreamer tended to be the aggressor in REM sleep dream interactions and the befriender in NREM sleep dream interactions.

9.2.5 REM Dreams Engage in Greater Amounts of Emotional Processing

Strauch and Meier (1996, p. 138) comment that in their dream series, "barely every second NREM dream featured the dream self emotionally related to the dream situation, whereas four out of every five REM dreams involved the dreamer emotionally in its events." Smith et al. (2004) scored both REM and NREM reports for emotion content from twenty-five dreamers. They identified eight emotions and found that most of them were rated as more intensely

expressed in REM versus NREM dreams. They then divided these eight emotions into positive and negative categories. The positive emotion category consisted of joy/elation and love, while the negative emotion category consisted of anger, anxiety/fear, sadness, and shame. They found that negative emotions were significantly more intense in REM than NREM, whereas positive emotions were not.

Van der Helm et al. (2011b) presented evidence consistent with the claim that REM sleep functions in part to facilitate emotional regulation. The authors point out that REM sleep is associated with a massive reduction in noradrenergic tone in forebrain centers, including the amygdala. (The amygdala is known to be involved in the processing of emotions – especially negative emotions like fear.) In addition, processing of emotional memories via amygdalar-hippocampal interactions takes place during REM. Thus, two events take place in REM that are crucial to daytime emotional regulation: (1) reactivity of the amygdala is downregulated due to suppression of central noradrengic tone; and (2) emotional memories are reactivated in amygdalar-hippocampal networks during REM. The latter process involves the processing of memories in absence of norepinephrine such that they are stripped of their stress-related arousing capacities before being stored in long-term networks. In short, REM is hypothesized to de-potentiate amygdalar reactivity and reprocess emotional memories in a state where noradrenergic activity is suppressed, thereby decreasing the overall intensity of negative emotional memories.

In the study that Van der Helm et al. performed, two repeat fMRI tests on thirty-four volunteers (test 1, test 2), separated by either a either a night of EEG-recorded sleep or a full waking day. During each test, participants viewed and rated the subjective emotional intensity of 150 standardized emotional pictures. Importantly, participants viewed the same stimuli at both test sessions, affording a measure of change in emotional reactivity to previously experienced affective stimuli (test 2, test 1) following wake or sleep.

Results showed that those who slept between the image viewings reported a significant decrease in their ratings of the intensity of the emotional images as well as a decrease in amygdala reactivity, while the participants who were awake between viewings/ratings demonstrated increases in both ratings and amygdala reactivity. Interestingly, the extent of overnight decrease in both amygdala reactivity and ratings was significantly correlated with the extent of reduced prefrontal EEG gamma activity (a biomarker of arousal and possibly central noradrenergic activity) during REM such that those with the lowest levels of REM-gamma expressed the largest overnight decrease in emotion reactivity. Thus, the authors have shown that amygdalar activity is decreased in REM and that this is associated with both a reduction in behavioral ratings of intensity of emotional pictures and a reduction in EEG gamma power in PFC.

The reduction in emotional reactivity associated with sleep may be a by-product of other processes occurring in REM. For example, the authors also reported a significant group (wake, sleep) by test (test 1, test 2) interaction involving altered connectivity patterns with the amygdala and the ventral-medial prefrontal cortex (vmpfc), that indicated an overnight increase in functional connectivity in the sleep group and a corresponding decrease in functional connectivity across the day in the wake group. These data suggest that the overnight reduction in amygdala activity was due to or related to an increase in vmpfc connectivity. PFC functioning is crucial for a huge array of higher cognitive functions, including, of course, dreaming. If so, then the facilitation of emotional memory consolidation during REM likely also requires cognitive products of REM, including REM dreams. During REM sleep, memories are being reactivated, decontextualized, and then integrated into long-term memory. These findings are consistent with Nielsen and Levin's (2007) proposal that dreaming normally functions to extinguish fear memories.

9.2.6 REM Dreams May Be More Storylike Than NREM Dreams

Nielsen et al. (2001) reported greater storylike complexity in REM versus NREM dreams and greater evidence for episodic progression in REM versus NREM dreams. More recently, however, Montangero and Cavallero (2015) reported that none of fourteen dream reports elicited from N2 or REM were structured like a canonical story. Nor did the REM and NREM reports differ in terms of their sequential regulation of events in the narratives.

So which account is correct? Do REM dreams exhibit greater storylike qualities than NREM dreams? Certainly, on the face of it, REM dreams are far more storylike than NREM dreams. If you wake someone up from REM, you are far more likely to obtain a story of who did what to whom and why than if you woke that same person up from a NREM episode. But to date, all studies attempting to capture the storylike structure of REM dreams have been inadequate. Nielsen and colleagues (2001) attempted to capture story content focused on the concept of episodic progression, defined as a three-step causal chain of events. An event causes a character to react, and this reaction entails another event. But stories are made of more than cause-and-effect sequences. They build to a climax and often involve dramatic tension and resolution. Episodic progressions go somewhere; they do not just continue indefinitely and aimlessly. As Montangero and Cavallero (2015) point out, about half of the dreams in Nielsen and colleagues' sample did not include a single well-defined episode. Similarly, Montangero and Cavallero's (2015) attempt to capture story qualities of dream reports used microanalysis of the linguistic

connections between successive temporal units. But stories are far more than sequential ordering of elements in a discourse. In addition, Montangero and Cavallero studied only about seven dreams from each sleep state. Finally, while Cipolli and Poli (1992) used a more sophisticated measure of story structure, they still confined their analysis to episodes and hierarchical event structures. But again, stories are more than episodes, and embedding one episode within another does not necessarily capture dramatic tension and progression within a true story.

In summary, while it is safe to say that the phenomenologic appearance of REM dreams manifestly reveals them to be well-formed stories, researchers have not yet been able to adequately measure just what makes REM dreams storylike.

Pace-Schott (2013) has suggested that the storylike quality of REM dreams is similar in some respects to confabulation seen after damage to the frontal lobes. Confabulation after prefrontal damage involves patients using whatever cues or suggestions are at hand to construct a fabrication to explain some puzzling behavior. They do this effortlessly and automatically when they find themselves needing to explain something to themselves or others. They are unaware that the explanation they just concocted is largely made-up and false. Just as in the case of REM dreams, there is a fictive narrative produced effortlessly and without insight that it is not reality. Pace-Schott also pointed out that the brain regions implicated in REM dreaming share some of the same brain regions implicated in confabulation. Spontaneous confabulation, for example, results from lesions of the anterior limbic system, including posterior medial orbitofrontal cortex (pmopfc, part of vmpfc) and its subcortical connections. REM dreams, Pace-Schott concludes, "may represent a potent, naturally occurring form of confabulation in which imaginary events are not only created and believed but are vividly experienced as organized, multimodal hallucinations" (Pace-Schott, 2013, p. 2).

9.2.7 Conclusions

While it is now clear that REM dreams and NREM dreams differ in terms of content and phenomenologies, these two dream types do not exhaust dream phenomenology. Relative to REM dreams, NREM dreams tend to be less storylike and bizarre and tend to contain fewer aggressive social interactions and greater numbers of friendly interactions, at least where the dreamer-initiated interactions are concerned. In the next chapter we will present some unusual dream types to demonstrate the amazing variety of dream experiences people undergo each and every night of every year.

9.3 Review Questions

- Some antidepressants depress some aspects of REM sleep. What might happened to dreams and their role in emotional regulation when REM is chemically suppressed?
- Evaluate the strengths and weaknesses of the evidence for greater aggression levels in REM versus NREM dreams and discuss the significance of this finding
- What role do dreams play in the consolidation of memories?
- What is the dream-lag effect, and what is its significance for a theory of dream function?

Further Reading

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CHAPTER TEN

Dream Varieties

Learning Objectives

- Evaluate the significance of the huge variety of dreaming experiences reported by people
- Evaluate the mechanisms and signficance of dream recall in dream phenomenology
- Distinguish properties and content of unusual dream types such as lucid dreaming, sleep paralysis dreams, and nightmares
- Identify the impact of the big data revolution on documenting dream variety and experiences

10.1 Introduction

It is not possible to understand dreams unless we become familiar with the whole terrain of dreams. We have discussed ordinary run-of-the-mill dreams that are associated with both REM and NREM sleep states. But there is a very large variety of dream types reported by people. To build an adequate understanding of dreams and a testable theory of dreams, we therefore need to marshal all of the salient facts concerning dreams, and those facts must include the chracteristics of a wide variety of dream types. Scientists who study dreams agree that dreams vary substantially in terms of their content and formal phenomenologic features. For example, children's dreams are very different from adult's dreams, and men's dreams differ substantially from women's dreams. There are nightmares, "big" or emotionally significant dreams, lucid dreams, shared or mutual dreams, twin dreams (dreams reported by twins), "spiritual" dreams, precognitive or prophetic dreams, visitation dreams (where deceased loved ones appear in a dream), and many other types. Dreams also vary by historical period: Dreams of the ancient Greeks and Romans are different than dreams of people in the Rennaissance period of European history. Dreams also vary by culture: Dreams of people living in traditional socieities are very different from the dreams of modernized peoples. Similarly, dreams of people living in Islamic cultures differ from the dreams of people living in cultures where other religions predominate and so on. All of this

should be pretty obvious, but dream variation is an understudied topic in the field of sleep and dream studies. While the variation in dream content and types has been documented, there is little discussion of the theoretical importance of that variation. The fundamental theoretical importance of dream variation, I will argue, is that it suggests that dream function is probably multiple. Dreams do not have only one function. No one theory can account for the huge variation in dream conent. The evident fact that there are multiple dream types is also consistent with the idea that dreams are products of the social brain and function, at least in part, to shape, alter, influence, or manipulate social relationships. Now that is NOT all that dreams do. It is likely that dreams transcend mundane social functions in multiple ways, but we simply do not know enough about these suprarational functions of dreams to comment upon them intelligently. I urge further research on so-called anomalous phenomena and dreams, but I focus here on the available empirical data we have on hand.

I will survey a number of dream types in order to give the reader a feel for the large terrain of dream phenomena any dream theory must account for. I have discussed the typical dreams of men, women, and children in previous chatpers. I will now discuss a variety of dream types as well as atypcial dream phenomena, as it is often in the study of atypical cases that we can gain insight on basical functional design chracteristics of the phenomenon under study. But first we need to consider the fundamental act of recalling a dream in the first place, as dream recall is the primary biologic constraint on dream variety.

10.2 Dream Recall

Why is it that some people seem to be able to recall dreams frequently, while others claim that they never have a dream at all? Although spontaneously or experimentally induced awakenings from REM sleep result in 80–90 percent dream recall rates, and everyone has REM sleep, we still get people claiming not to dream at all. Survey studies show that most people recall one to two dreams per week at home (Stepansky, Holzinger, Schmeiser-Rieder, Saletu, Kunze, & Zeitlnofer, 1998). About 31 percent of the population recalls dreams about ten times per month or more; 37 percent report dreaming one to nine times per month; and 32 percent report dreaming less than once per month. That break down of dream recall rates suggests that there is a normal distribution of dream recall in the general population, with about a third being high dream recallers and a third being relatively low dream recallers. A small percentage of people in the low recall wing of the dream recall distribution claim to never dream. What is going on with these people? Do they actually dream and just not recall their dreams, or do they really not dream at all?

In a recent ingenious study by Herlin and colleagues (2015), the study authors were able to show that people who claim not to have ever dreamed actually do dream, because the authors witnessed the non-dreamers actually acting out their dreams! The authors capitalized on the fact that these individuals had Parkinson's disease (PD), which is frequently (though not invariably) accompanied by REM Behavior Disorder (RBD). In RBD patients act out their dreams whenever they go into REM sleep because the cells associated with the motor paralysis that normally inhibits overt behaviors during REM are destroyed by the disease process associated with PD. The authors studied patients who reported no dream recall for at least ten years, and "never-ever" recallers who claimed never to have dreamed. These non-dreamers were compared to ordinary dream recallers. All of the individuals in these distinctive dream recall groups had RBD. Of the 289 patients with rapid eye movement sleep behavior disorder, eight patients (2.8 percent) had no dream recall, including four patients (1.4 percent) who had never ever recalled dreams, and four patients who reported no dream recall for ten to fifty-six years.

Thus, Herlin et al. really did find some individuals who would have been classified as non-dreamers in other studies. These were people who claimed to have virtually never had a dream. Then the authors watched these individuals overnight, using video-somnography or other techniques to verify they were in REM sleep. "All non-recallers exhibited, daily or almost nightly, several complex, scenic and dreamlike behaviors and speeches, which were also observed during rapid eye movement sleep on video-polysomnography (arguing, fighting and speaking)." In short, like every other person with RBD, these individuals exhibited the classical dream enactment behaviors of RBD. In typical cases of RBD when we query these patients as to whether they recall any dreams they had during the night, they will often describe dreams that match up to varying degrees with the behaviors they exhibited during their dream enactment behaviors while asleep. In the case of Herlin et al.'s poor dream recallers, however, they denied recalling any dreams when they were asked about dreams in the morning after they exhibited classic dream enactment behaviors! Neither did they recall a dream following sudden awakenings from rapid eye movement sleep. Why can't these individuals, these poor dream recallers, recall the dreams that their own dream enactment behaviors suggest that they had during the night? The eight non-recallers with REM behavior disorder did not differ in terms of cognition, clinical, treatment or sleep measures from seventeen "control" dreamers studied by the authors. So it can't be that the poor recallers could not recall their dreams simply because they have poor memory or particularly severe disease and so on.

So what prevents poor dream recallers from remembering their dreams? As this and other studies have shown, there is a small percentage of the population

(up to 3 percent) who swear that they never dream. But this study suggests that they simply cannot recall their dreams. Why?

10.3 Neural Correlates of Dream Recall

The probable answer is that the brains of poor dream recallers differ slightly from the brains of ordinary recallers. The neural correlates of dream recall has been studied using quantitative scalp electroencephalographic or EEG measures. Dream recall from stage N2 is associated with a lower level of alpha oscillatory activity in the right temporal area. On awakening from REM, dream recall is associated with a higher level of frontal theta activity. Theta and alpha oscillations are correlated with successful dream recall with involvement of temporoparietal (TPO) and ventromedial prefrontal (vmpfc) areas. Interestingly, lesions in the white matter tracts to and from the ventromedial prefrontal cortex or lesions disconnecting temporal parietal junction from other regions in the circuit that make up the social brain can result in cessation of dream recall. Regional cerebral blood flow to TPO and vmpfc is higher in frequent dream recallers compared to low-frequency recallers. Thus, people who claim never to dream very likely have reduced (relative to the rest of the population) brain activity levels in the TPO and vmpfc.

Siclari et al. (2017) reported a very interesting set of findings on the neural correlates of dreaming/dream recall. Since the 1950s, we have known that if we awakened people during REM we would reliably get reports of dreams. About 70 percent of the time, if we awakened people during the N2 light stage of sleep we would also get reports of dreams. Even if we awoke people during deep slow wave sleep states (N3), we could still get reports of dreams, though certainly not as reliably as when we awoke them from REM or N2. In short, even though REM, N2, and N3 were defined by dramatically different EEG signatures, we could still get reports of dreams in each of these sleep states. Clearly, the standard EEG sleep montage was too gross an instrument to isolate those brain states most reliably associated with dream reports.

Siclari et al. used high-density EEG recordings to isolate neural correlates of dreaming regardless of standardly identified sleep state. The authors contrasted the presence and absence of dreaming in both NREM and REM sleep. When a posterior "hot zone" showed a decrease in low-frequency EEG activity – traditionally known as "EEG activation" – subjects reported upon awakening that they did have dream experiences. By contrast, when low-frequency EEG activity increased in the same area, subjects reported that they had no dreams. Thus, those neural sites that were consistently activated whenever participants reported dreams and consistently inactivated whenever participants reported the absence of dreaming – regardless of standardly defined sleep states –

included sites within the "hot zone" that comprised the occipital cortex, precuneus, and posterior cingulate gyrus. By monitoring neural activity in this posterior "hot zone" the authors could predict when a subject would recall dreaming.

Use of high-density EEG is extremely technically challenging. That is why so few sleep studies use this technique. It is subject to all kinds of artifacts, and so researchers have to use extraordinary precautions (e.g., specially built soundproof rooms, etc.) to control noise when using these huge EEG montages. The authors not only used high-density EEG during sleep; they apparently were able to avoid artifactual contaminants even when repeatedly awakening subjects for dream reports! This is a significant accomplishment in itself.

The neural correlates for dream experience was localized to the so-called hot zone for dream experience in the posterior cortical region in this study. That region included the occipital cortex (visual center), the precuneus, and the posterior cingulate. This site seems reasonable to me to be a hot zone for dream experience. Precuneus activation, for example, has been associated with self-awareness and is part of the social brain circuit. It has been known for some time that damage to the temporal-occipital-parietal (TPO) junction can result in cessation of dream recall. The hot zone described in this book likely overlaps to some extent with the TPO.

When the authors looked at the hot zone during REM, cortical activation patterns extended far beyond the posterior cortical hot zone and into the frontal lobes. Lesions to the ventromedial frontal lobes also results in cessation of dream recall. In summary, Siclari's results are consistent with previous research on neural correlates of dream recall but adds to it the precuneus. Thus, we have the precuneus, the TPO, and the vmpfc as key nodes in a circuit responsible for generation of dreaming and subsequent recall of dreams. These nodes are also the key nodes in the social brain circuit.

10.4 Lucid Dreamers Are Specially High Dream Recallers

People who report that they frequently have lucid dreams also evidence the highest dream recall rates. This is not surprising given that you need to frequently dream and recall your dreams if you are going to have any hope of becoming aware or lucid in one of them. What is it about these high dream recallers that makes them the world champions in dream recall? Studies show that they differ from ordinary dream recallers in terms of their ability to solve puzzles, achieve sudden insight in solving a puzzle, and control their attentional skills. Presumably their brain activity levels in the social brain circuit are also high.

10.5 What Can Be Done to Boost Dream Recall Rates

Until we can directly boost brain activity levels in the social brain circuit, all you need to do to boost dream recall is set the intention to recall dreams and then use a journaling technique to record the dreams you remember. Here is where the new apps associated with smartphone devices can help.

A sleep and dream journaling app can be downloaded in two minutes on literally millions of phones. Consider, for example, if you had an app that could reliably detect REM sleep (via an accelerometer). When the movement data detected a REM profile, the phone would then emit a wake-up alarm and turn on a recording function so that you can speak your dream directly into the phone before you forget it. This can be done via autodialing a number that rings a recorder at the lab or right into the phone's recorder or an app designed for recording audio files, etc. Once the individual records his dream, the audio file is transmitted to the lab for analysis. Most dream apps can transmit the dream audio files directly onto a website where it is automatically content analyzed and aggregated into a huge database containing other dreams sent by people from all over the world!

10.6 Dreams in Traditional Societies

Nothing illustrates the social function of dreams better than examining the role of dreams in small-scale traditional societies. By traditional societies, I mean tribally based groups of perhaps a few hundred to a few thousand individuals living the way our hunter-gatherer or horticultural ancestors lived 20,000 or more years ago. There is no straightforward concordance between traditional societies and early human groups. Traditional societies are not mere replicas of early human groups. They are not "primitive" or backward. Rather, they are living the way that early human groups likely lived, and therefore we can learn something about our ancestors from studying these traditional societies. Small-scale traditional societies are the closest thing we have to a picture of what ancestral human societies were like. They can tell us something about the ways in which dreams were regarded and used by our ancestors.

We now have excellent studies of the role and status of dreams in traditional societies from several ethnographers and anthropologists, such as Steward Lincoln (1935), Eggan (1949), Devereux (1951), Kracke (1979), Kilborne (1981), and Irwin (1994). In addition, the role that dreams play in the generation of social relationships and cultural artifacts, such as rituals, sub-societies, religious myths, group alliances, medical practices, and much else besides, has been reviewed several times in the anthropological literature (see e.g., D'Andrade, 1961; Eggan, 1961; Barnouw, 1963; Grunebaum & Callois,

1966; Bourguignon, 1972; Tedlock, 1987, 1992; Irwin, 1994; Lohmann, 2003; and Laughlin, 2011). The work of these anthropologists and ethnographers has made it clear, for all who are willing to study their carefully documented ethnographies on aboriginal peoples all across the planet, that dreams were almost universally considered to be central sources for rules governing social relationships as well as sources of all kinds of cultural innovations – most especially religious innovations. In his study of the Plains Indian cultures of North America, Lee Irwin noted that "[d]reaming is a creative basis for what might be called higher knowledge in the Native American context . . . Dreams and visions constantly revealed new applications of many types such as: inventive technologies, hunting methods, warfare strategies, healing practices and herbal formulations, along with other innovations in culture. For example, the origin of fire making was attributed to visionary experience by the Lakota" (Irwin, 1994, p. 191).

In many traditional societies certain members of the population underwent initiation rites to become specialists in dream sharing and interpretation. For example, the "daykeepers" among the Quiche Maya of Guatemala not only interpret dreams, they actively encourage tribal children to remember their activities in dreams and to consider these dream activities of equal in importance to daytime waking activities. Many traditional societies (e.g., Huichol Indians of Mexico, Yansi of Zaire, many tribal groups of the Yoruba nation in central Africa, Mapuche Indians of Chile, Ojibay of North America, Lokota of North Dakota, and many others) practice daily dream–sharing rituals where groups gather in the morning to share dreams and to occasionally enact them. For example, if a dream is considered to carry significance for the dreamer's life vocation or for the tribe's welfare, the scenes in the dream will be translated into dance and ceremony and then performed. The tribe may even set aside valuable time and resources to create new garments, masks, ornaments, and other cultural artifacts to fully bring to life the scenes, characters, and drama in the dream.

Dreams in traditional societies could confer on a person tremendously important social status, yet there are no accounts of attempts to fake significant dreams in all of the ethnographies I have studied. The best studied example of this status-inducing dreaming experience is the case of the shamans of traditional societies. Shamans were the expert practitioners of the spiritual and magical arts of the traditional world. They were a mix of medicine man and sorcerer. They often did not choose to be a shaman. Instead, they had a dream one night, shared it with the group, and then were designated a shaman. They then had to apprentice themselves to an existing shaman, learn the medical and religious lore of the tribe, conduct medical interventions, direct ceremonies, chant myths, and occasionally direct hunting parties to optimal hunting

grounds. Initiatory dreams of a Siberian shaman almost always contain visions of dismemberment of the body, followed by a renewal of the internal organs and viscera, ascent to the sky and dialog with the gods or spirits, descent to the underworld, and conversations with spirits and souls of the dead shamans. These intense visceral experiences then lead to various revelations in a dream state.

The North American Ojibwa Algonguin–speaking Indians of Canada see the characters and events of dreams as carrying a significance almost equal to that of daytime events, but they do not confuse dream events with waking events. Pawaganak or "dream visitors" are nonhuman characters (what we might call supernatural beings) that come to Ojibwa dreamers in their dreams to deliver information and gifts or demands. These exchanges with the visitors establish reciprocal relations between the visitors and the dreamer. These reciprocal obligations become public once the dream is shared and must be observed to the letter. If they are not, the tribe takes note and stigmatizes the individual as engaging in bad conduct. The bad conduct injures the individual's reputation and follows him until the obligations are discharged appropriately or some other ritual intervention is accomplished. The Pawaganak are highly sought after in Ojibwa culture, not only because these relationships can be established that alter a person's social standing in the tribe, but also because the gifts often create special powers in the individual – powers to see the future, cure the ill, or obtain the best hunt or best resources. Occasionally, the dreamer obtains the power of sorcery so that he or she can inflict harm on an enemy by performing some ritual or incantation discovered in the dream. Death songs, curative songs, songs for the hunt, and other powerful songs can also come to the dreamer in dreams with the visitors. These songs then remain with the individual for the rest of his life.

Just as the Siberian shamans underwent their initiation into the shaman's life via initiatory dreams, so too Ojibwa boys found their special gifts via a dream initiation ritual. A boy about to enter puberty and become a man would undergo the dream fast. Once entered into the ritual he would be called a kigusamo. Before he could receive his initiatory dream, he had to become pure or pekize via fasting, cleansing rituals, and avoidance of women. Then a group of male relatives would take him out into the wilderness to build a platform for him to sleep in, often naked to the elements. He was not allowed food until the initiatory dream came. His male relatives might perform a song they received in dreams to strengthen the boy, but often the boy would be left entirely alone. Then the dreams would come and the supernatural beings would appear in his dreams. He would have been admonished to accept gifts from the first beings to appear to him as they were often evil or trickster spirits. The boy had to be patient and wait for a genuine dream visitor. Next he had to prove to the spirit

being that he was worthy of establishing a personal relationship and reciprocal obligations with the being. Having passed these tests, the dream visitor would then bestow gifts or exceptional powers or pimadaziwin on the boy in the form of incantations, songs, dances, or cures, etc. The boy would feel himself being transformed into the form of his now titular spirit being and then leave the site to return to the village and share his dream. The tribe might then enact the dream in full ceremonial style, thus making the boy into a man – a man with special powers to serve the tribe.

This all too brief summary of the role and power of dreams in traditional societies underlines the importance of dreams in these cultures to significantly alter social relationships for the dreamer. It should not be surprising, therefore, that dreaming taps the social brain circuits discussed previously.

10.7 Physical Symptom Dreams

Because sleep is associated with reduced visual input it is commonly believed that the brain becomes more attuned to picking up internal somatic signals and turning these signals into images that appear in dreams. But it is entirely possible that the the brain/mind can pick up faint or prominent internal somesthetic signals whether or not external sensory input is reduced. The evaluation criteria concerning these internally generated bodily signals, however, changes as a function of whether the waking or the dreaming mind first evaluates them. There may be benefits to having the dreaming mind first evaluate a bodily pain or a developing infection. Sometimes images capture more information than do words or discursive accounts. In addition, dreams have a tendency to amplify sensation such that a barely audible tone in the bedroom becomes a rising siren in the dream and so on. For example,

> [f]eeling feverish and light-headed shortly after a flu shot and aware of an exhaustion in my limbs associatd with a rapid involuntary tremor, I lay on the floor, fell asleep and found myself in my study facing a rattlesnake. Its uplifted head was oscillating rapidly (at what I realized on awakening was the same rate and quality of my shaking leg muscles). Sinking to the floor, overcome by terror I crept backwards before its steady advance. Finally, I pushed a chair against it. To my surprize the rattlesnake stopped its advance and I awoke with the brief fever gone.
>
> (Hunt, 1989, p. 80)

There are many documented cases in the literature on dreams capturing signals related to bodily dysfunction and illness, often far before the illness was detectable by conventional means. This is an area of dream research that needs to be more rigorously examined as it could be of immense clinical significance.

10.8 Dreams of Patients with Multiple Personality Disorder/ Dissociative Identity Disorder (MPD/DID)

Dreaming in dissocitaitve identity disorder is associated with many understudied but fasinating phenomena. Therapists of patients with MPD/DID report that alter personalities can appear in dreams as characters in the dream. Often a new alter will first appear in a dream and then later take over control of the behavioral repertoire of the individual and become a daytime alter. The dreamer will often experience a switch from her primary identity to an alter during a dream. Because there is amnesia for the actions of an alter during waking experience, the primary identity will occassionally experience the alter's daytime experience as a deam. Barrett presents the case where a woman with MPD/DID had recurring nightmares of catching evil cats and stuffing them in garbage bags but then awoke to find herself covered in cat hairs. She then worried that an alter had atually been capturing cats and stuffing them into garbage bags. Sometimes several alters will be in the same dreams. Alter number 1 will describe the dream detail from her position as onlooker, while alter number 2 will describe the same scene as experienced from her perspective, and so forth. The host personality will sometimes gain memories in dreams that belong to one of the alters who experienced the events in question during a waking experience.

10.9 Sexual Dreams

While Freud made us all think that dreams were about sex, quantitative studies of dream content have revealed that only 12 percent of reported dreams contain explicit sexual content. Most dreams are about mundane daily social interactions with familiars and a couple of strangers. But of course, most people do not like to share content that they find embarassing, so these numbers are probably underestimates of the number of dreams that contain explicit sexual content. "Wet" dreams or dream-induced orgasms peak for men in their twenties and for women in their forties. It may be that many people experience their first orgasm in a dream – though I could find no confirmatory data on this speculation. Contary to threat simulation theories of dreams, most people experience the sexual content of their dreams as pleasureable. As discussed in previous chapters, REM sleep is associated with genital arousal for both men and women, though this arousal is not always associated with sexual content in the dreams. Nor do sexual dreams typically reflect the sexual fantasies of the individual. We do not always dream our most common sexual fantasies – though of course, some dreams do involve the dreamer's sexual fantasies. Some sexual dreams are classic wish-fulfillment dreams, such as

when we dream of making love with someone we desire but with whom we have no relationship. Virtually all types and kinds of sexual acts have been depicted in sexual dreams – even sexual acts not performed by the individual in waking life. There are many unanswered questions concernining sexuality and dreams. The dreams of homosexual, lesbian, and trangendered individuals have not been extensively studied. The correlations between daytime sexual activities and nightly dreams have not been studied. If compensatory theories of dreams were correct we would, for example, expect an inverse correlation between the two series of activities, but we do not yet have such data.

10.10 Incubated Dreams

Ritual invoking of a dream for healing or medical purposes was practiced for thousands of years in the ancient world. The best studied case is the Asclepian rituals wherein a person with an illness would travel to a temple of the God Asclepius and sleep in that temple until he or she had a dream that spoke to the issue of the illness. The evidence for the effectiveness of the ritual comes in the hard form of inscriptions incised on stone by grateful patients who were apparently cured or at least left improved after performing the incubation rituals, obtaining a dream, and then getting well. Sometimes the temple priests interpreted the dreams and then administered treatments prompted by the dream content. Perhaps it was these treaments that cured the patients rather than anything associated with the incubated dreams. But it must remembered that priestly administration of treatments would often include items (potions, bleedings, administration of poinsons, scarifications, etc.) that would almost certainly make any patient worse, so these treatments are not likely the causes of the cures obtained in these temples. Suppliants to an Asclepian temple had to report an invitational dream from the god wherein the god invited the suppliant to the temple for a cure. Fasting, ritual bathing in cold water, and animal sacrfice had to precede admittance into the ritual area of the temple. The suppliant once entered into the temple would be shown a couch or abaton or adytum upon which to sleep. The suppliant would sleep there until an appropropriate dream came. Most propitious dreams involved the apperance of the god himself who touched the patient on the afflicted body part or delivered some information, an image, or a kind of gift the patient could use to relieve the suffering associated with the affliction.

There are many contemporary accounts of persons attempting to use incubated dreams for various purposes with varying degress of success. There is no question that dreams can be incubated to come to the dreamer. But there is no consensus on methods to promote incubation, as we cannot reproduce the religious context that supported the Asclepian rituals in the ancient world.

10.11 Lucid Dreams

Lucid dreaming has long been a topic of interest in dream research. The term lucid dreaming was coined by Frederick van Eeden in 1911. He reported on lucid nightmares among other lucidity phenomena. In a lucid nightmare, the dreamer is aware that he is dreaming and that the dream is a nightmare. The nightmare themes often involve demonic figures out to inflict terrible harm on the dreamer, who struggles to wake up but can't. The more common lucid dream is a typical dream where the dreamer is aware of the dream and has no strong desire to wake up and end the dream.

Consider the fact that the dreamer quite clearly has awareness and self-consciousness. He can discriminate the real from the unreal within the dream. What about the reality sense in lucid dreams? Here we have a paradox. On the one hand the dreamer is aware he is dreaming. On the other hand the utter reality of what he is experiencing remains. Traditional peoples cultivated lucid dreams. They too could distinguish within the dreams what was real and what was unreal, and yet they still considered spirit beings real. There can be no recourse to the credulity of the dreamer in this case. The dreamer's reason is intact and yet he or she still believes in the reality of what he is experiencing. The ability to reason and engage in logical thought is intact. Access to the dreamer's autobiographical memories is intact. The ability to take on third-person perspective is intact, so the dreamer can consider, entertain, and imagine what another character in the dream is thinking or feeling as well. Indeed, whole interactions and dialogues between the dreamer and dream characters can take place just as in waking life. The dream characters furthermore cannot be considered mere creations of the lucid dreamer, as they act as if they had full mental capacity and autonomy, and in many lucid dreams and certainly in lucid nightmares, they clearly act contrary to the wishes of the dreamer. In short, all of the constituents of the mind that we take for granted in waking life exist in the lucid dream state for both the dreamer and the dream characters.

But in addition to the normal elements of the mind present in the lucid state, the lucid dreamer and other dream characters have added mental capacities. The lucid dreamer very often can make things happen in the dream that would be considered miraculous if they occurred in waking life. The lucid dreamer may be convinced he has supernatural mental powers in the lucid state. The lucid dreamer, furthermore, can sometimes control the unfolding of the dream plot, so he stands in relation to the dream characters just as a novelist does with his fictional characters – yet at the same time the dream characters confront the dreamer as real beings, even hyperreal beings, as in the case of lucid nightmares. Here, the dream characters can make things happen in the dream

that are contrary to the will of the dream. Actions initiated by dream characters can cause everything from orgasms in the dreamer to near-death experiences in the dreamer. There are testimonies on record, for example, of a dream character shooting a dreamer in the heart and the dreamer waking up with a heart attack. Is this a case of the dreamer confabulating a story line in order to explain the pain of a heart attack? We will never know. In all likelihood dream characters have "caused" death to dreamers as well, but we will never be able to document such an event.

Most interestingly, of course, is the fact that the lucid dreamer is essentially a fully awake human person who cannot be said to be hallucinating (because he knows what is real and unreal), and yet who observes a fully realized visual world replete with settings, environments, characters, supernatural beings, unusual movements, actions, story line, plot, atmosphere, and normal actions, etc. just as occurs in the waking world. Indeed, this dream world and the characters in it are so real that they can intensely affect the dreamer's physiologic reactions even unto death.

While the lucid dream does in fact emerge very often from the REM state (and that is why it is legitimate to call it a dream), some evidence suggests that it functions in some sort of hyperreality state, given that brain activity exhibits even higher gamma-band activity than the waking brain. So is it a hybrid REM–wake state or perhaps a whole new brain state, enabled by REM but transcending REM in some remarkable manner? (see Baird et al., 2022).

Lucid dreaming is not a waking state either, however. Unlike relaxed wakefulness with eyes closed, lucid dreaming shows no evidence of stable alpha-band activity on EEG and instead is characterized by sleep-related low-frequency theta and delta activity with bursts into higher bands, peaking at the 40-Hz frequency band especially at frontal sites on the EEG (see Figure 10.1).

Dresler et al. (2012) managed to collect functional neuroimaging data on at least one out of four lucid dreamers they studied. While the dreamer was in the lucid state, bilateral precuneus, cuneus, parietal lobules, and prefrontal and occipito-temporal cortices displayed significantly greater activation as compared with the non-lucid REM state. As the authors themselves pointed out, these areas of activation (e.g., the parietal and dorsal (not ventral) prefrontal lobes overlap considerably with areas that are known to undergo deactivation during REM sleep). Thus, the neuroimaging data seem to confirm the idea that lucid dreaming is not an exclusively REM phenomenon even though it may begin in REM, and some elements of REM (e.g., muscle paralysis) remain while in the lucid state. While the fact is that the prefrontal and parietal cortical networks are reactivated in lucid dreams, this fact only helps us explain access to logical thought and awareness while in the lucid state. The lucid dream's most fascinating mysteries remain unexplained.

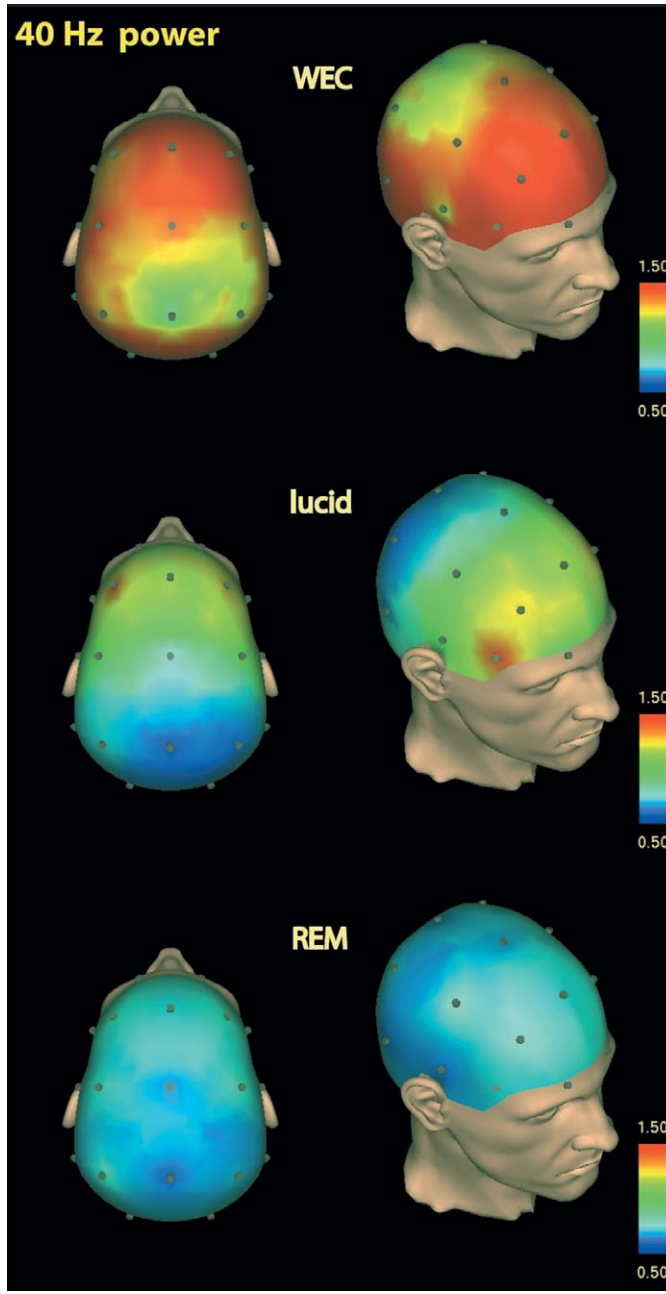

Figure 10.1 Single-subject 40-Hz standardized current source density power during wakefulness, lucid dream sleep, and non–lucid rapid eye movement (REM) sleep
(Lucid dreaming is shown to have higher than REM activity in upper frequencies, peaking in the γ band frontolaterally at 40 Hz).

Used with permission from Voss et al. (2009)

10.12 Twin Dreams

With twin dreams we have multiple case collections of twins who have dreamt the same dreams or have had precognitive dreams about the other twin. If identical twins have similar brain structures due to similar genetic endowments, then they should have similar REM sleep capacities and therefore similar dream content; similar but not identical content. Occassionally, two people claim that they had exactly the same dream. We have to take these reports seriously as we have two different people verifying that they in fact shared a dream. Twins commonly report that they share dreams. If we assume that twins do not lie about these experiences, then we have two choices: (1) the twins unconsciously and covertly, in some unknown manner, shared the information about dream content; (2) identical (or nearly identical) dreams can occur in two different (but very similar) brains.

Perhaps they had the same dreams because their brains are so similar? Yes, possibly, but then why have the same dreams on the same night? If structural brain similarities are producing the sameness of content, you would not expect that sameness of content to happen on the same night unless your view of structural similarity was so deterministic as to be laughable. We know twins' brains are not structurally identical in any case; brain plasticity makes that an impossibility. Runyan (2010) reported the following dream sharing accounts from twins:

> One twin set reported nightmares with a similar threat, tornadoes, in recent dreams. One twin set reported the same occurrence from two points of view while sleeping side by side; one twin dreamt that she came to tell the other twin that she was not getting married, and the other twin dreamt that her twin came to tell her that she was not getting married (p. 141).

In addition to these shared dreams, Runyan reported apparent precognitive dreams where one twin foresaw in his dreams a calamity that was going to befall the other twin:

> For a few weeks, I had a recurring dream (nightmare) where I was driving my car after dark. During this period of driving, I would be involved in an automobile accident where the car I was driving would flip over and over again causing my death. I was almost to the point of not driving any more after dark when my identical twin died in an auto accident where he flipped his car over with it landing on the top directly over him. This caused him to have a massive brain injury resulting in his death (Runyan, p. 140).

Runyan used the standardized Hall/Van de Castle rating scales to quantitatively compare the dream content of twins with the dream content of singletons. Some 44 percent of twin dreams contain the other twin as a character in

their dreams. Singletons do not dream that frequently of their siblings. Indeed, only 19 percent of characters in singleton dreams are family members (parents, siblings, etc.). Given that dreams of twins are often about their twin, it is not surprizing that social interactions in twin dreams are more friendly than what you see in singleton dreams. Friendliness appeared in 66 percent of the twin dreams but in only 42 percent of singleton dreams. Most interesting, perhaps, was the finding that 76 percent of the twin dreams took place in unfamiliar settings while only 38 percent of the singleton dreams did so. Why is this the case? Why should twins dream of unfamiliar surroundings far more than do singletons? We do not know. Twin dreams represent a relatively unexplored realm of the human mind and of dreams. It represents a very rich area for science as it will reveal clues as to the nature of dreaming, as well as the nature of the brain and mind relation itself.

10.13 Big Dreams

"Big dreams" are impactful, transformative dreams that typically involve direct encounters of the dreamer with a supernatural agent and that mark turning points in the lives of the dreamers. They have been important in the history of religions and are certainly important in the religious and spiritual lives of the individuals who have them.

Scholarly interest in big dreams has been increasing in recent decades. For example, Kuiken and his associates (see Kuiken & Sikora 1993) studied impactful and spiritually transformative dreams in ordinary volunteers. They identified three major types of impactful dreams, which they dubbed existential, transcendent, or nightmare dreams. All three types of dreams are experienced as very intense, memorable, and absolutely veridical/real. Each of the three types exert differing types of aftereffects. Existential dreams are followed by increased self-reflection about feelings that the dreamer was previously reluctant to accept; nightmares are followed by increased vigilance and sensitivity to threats; and transcendent dreams are followed by consideration of previously ignored spiritual possibilities.

10.14 Nightmares

The DSM-5 defines Nightmare Disorder (DSM-5 307.47 (F51.5)) as a parasomnia involving repeated awakenings from extremely frightening dreams that do not occur in the context of some other mental disorder. Upon awakening, the individual is oriented and alert and has clear recall of the content of the

dream, which, in turn, is associated with clinically significant distress and impairment in daytime functioning. Epidemiological studies indicate that 2–6 percent (about 6.4–15 million people) of the adult American population experience nightmares at least once a week. Between one half and two thirds of children experience recurrent nightmares. Nightmares are distinguished from scary or disturbing dreams by the presence of monstrous gents in the dreams. These supernatural agents evoke within the dreamer dread, terror, awe ,and uncanny fear.

10.15 Meeting One's Double in a Dream/Nightmare

The experience of meeting one's own double (the doppelganger) in a dream has been reported throughout recorded history and has most often been described by the people who experience it and in literature as a profound and dangerous encounter with one's own soul. Although doppelganger dreams are rare, they are memorable, and most people, if pressed, will report at least one such dream in their lifetime. In most doppelganger dreams, the dreamer sees him or herself in the dream – though the double usually does not perform many actions. It is as if looking at oneself in a mirror. When the doppelganger appears in a dream the dreamer almost always describes it as frightening and of special spiritual significance. One cognitive explanation of the doppelganger is that it represents a memory of seeing oneself in a mirror or even a kind of doubling of autobiographical memory.

10.16 Visitation Dreams

In visitation dreams, a loved one or an acquaintance who has died appears in a dream and looks alive and healthy and generally carries a message for the dreamer. Common themes in visitation dreams involve the deceased appearing in the dream as they did in life rather than as they did when they fell ill. In fact, the deceased often appears much younger or more healthy than when they died. The deceased might convey reassurance to the dreamer: "I am OK and still with you." This message tends to be conveyed telepathically or mentally rather than via spoken word. The dream structure is NOT disorganized or bizarre. Instead they are typically clear, vivid, intense, and are experienced as real visits when the dreamer awakens. The dreamer is always changed by the experience. There is resolution of the grieving process and a wider spiritual perspective. Often these visitation dreams can be quite emotional. See some examples of visitation dreams posted by people at my *Psychology Today* Dreamcatcher blog: www.psychologytoday.com/blog/dream-catcher.

10.17 False Awakenings and Sleep Paralysis Dreams

A false awakening is a type of dream that involves the subjective experience of waking up while remaining in the dream state. The dreamer feels as if he has woken up and he then goes about his daily routine, such as getting dressed or brushing his teeth. While performing these routine tasks, the dreamer then really wakes up! Other false awakenings contain fantastic or unrealistic elements not associated with the dreamer's waking circumstances. The dreamer may wake up in the same dreamed environment as that experienced within the dream that occurred prior to the false awakening. As in the movie *Inception*, the dreamer may have to undergo several false awakenings before he is really able to wake up. Occasionally, the dreamer may awaken into a dream containing aspects of the dreamer's past. For example, a false awakening may entail waking up in the dreamer's childhood bedroom.

In the early twentieth century, the French zoologist Yves Deluge described a dream where he was awakened by a friend knocking at his door, asking for his help. Alarmed, he got dressed and began to wash his face when he realized he was dreaming. But then he would find himself asleep again and hearing the knock on the door, hurriedly getting dressed to help his friend, and once again realizing he was dreaming. This cycle occurred several times before he finally really woke up.

Sometimes false awakenings are accompanied by an inability to move one's body. This type of experience is likely a variant of the dreams associated with isolated sleep paralysis (discussed in a previous chapter and in the next section). False awakenings and sleep paralysis both occur during transitional periods between waking and sleeping and combine characteristics of both states in unusual ways. There are also significant overlaps between descriptions of false awakenings and reports of out-of-body experiences, apparition sightings, and alien abductions.

10.18 Sleep Paralysis Dreams

Isolated sleep paralysis (ISP) is a relatively common experience, typically characterized by an inability to move or speak after waking up as well by the eerie sense that someone or something is in the room with you or is somehow evil or malignant and threatening you (Cheyne & Girard, 2007). It is legitimately categorized as a dream because the individual often experiences both auditory and visual hallucinations as well as the muscle atonia or paralysis that normally accompanies REM. The author Louis Proud (2009) provides this example:

I wake up but not completely. I can feel something touching my forehead; it is this that has drawn me kicking and screaming into a semi-conscious state. But in truth I cannot kick or scream; in fact, I can't move a single muscle in my body, even though my mind is awake. For gods sake I can't even open my eyes. All I can do is lie there while this thing attends to my forehead with delicate loving strokes. Whatever it is I can smell the stench of its presence. I can taste its mind just as it can taste mine. But its love, its child like affection is sickening me and all I want is for it to leave me in peace. I continue to lie there, engulfed by the darkness as the shadow strokes my forehead. It then moves away to the left of my body and proceeds to lie down beside me. It writhes around annoyingly until it finds a comfortable position. Then once its finished messing around it puts it arms around me. Its grip is so tight that my chest is aching. I want to scream out in fear and disgust but there is nothing I can do" (Proud, 2009, pp. 26–27)

Proud had this experience when he was seventeen and he reports in his book that the experiences returned frequently, often with other more menacing demonic figures. But almost always, the experiences involved some or most of the events described in his original experience. His mind is awake but he is paralyzed. He senses a demonic evil presence near him, which then attempts to interact with him in some way. Most often the intent of the demon is to possess him or destroy him. Often he hears voices or senses more than one presence. The presence is evaluated as something of immense evil that wants to destroy Proud. The dominant emotion he experiences is virtually always fear or terror. The experience is caused by fragmentation of the brain state associated with REM such that one does not move entirely out of REM when one transitions from REM to wake.

10.19 Musical Dreams

While very few of us regularly recall dreams with music in them, most of us have had at least one dream that could be called a musical dream – a dream where music was the main ingredient. Why do we not have more musical dreams? After all, music is a very big part of daily life for a lot of people. If the content of dreams generally reflects our everyday activities, one would expect "heard" music to show up in quite a few dreams, but this is simply not the case. While Kern et al. (2014) recently reported an association between time spent in daytime musical activities and percentage of dreams reflecting some amount of musical activity, the frequency with which musical phrases occur in dreams still appears to be quite low. Why is music so rare in dreams?

Perhaps dreams treat musical phrases and other non–self generated thoughts as "foreign" and therefore attempts to protect us against these sorts of "parasitic"

ideas. This idea goes back to the novel hypothesis concerning the function of dreams advanced by Crick and Mitchison (1986). They suggested that the REM sleep/dreams system functions as a kind of reverse learning mechanism that isolates nonessential and potentially parasitic informational elements and then eliminates them from the brain/mind. For most of us, the dreaming mind treats musical phrases as nonessential and parasitic, and it therefore prevents musical phrases from catching hold and entering long-term memory. This is not the case for musicians. For these people, music is essential and nonparasitic, so their dreaming systems allow processing of musical phrases.

10.20 Dreams of the Sensorially Limited

Blind people have a lot of dreams just like normally sighted individuals, but their dream content varies in terms of visual imagery vividness. If individuals were blind from birth they typically do not report visual images in their dreams. Instead, they will report verbal interactions and events and perhaps touch and sound images. Those individuals who became blind after around age seven still have dreams with visual images in them but over time those images become less vivid.

When amputees dream, they dream of themselves intact. They do not experience loss of the limb in dreams even years after the amputation and even when the physical handicap was congenital.

Similarly, dreams of the congenitally deaf-mute or those of the congenitally paraplegic cannot be distinguished from those of nonhandicapped subjects. This was true for most aspects of both form and content. Dream reports from deaf-mute individuals involve them talking and hearing normally. Patients with varying degrees of paraplegia report themselves flying, running, walking, and swimming.

10.21 Recurrent Dreams

Recurrent dreams are dreams that reoccur over time while maintaining the same content over time. They are relatively common, with 60–75 percent of adults reporting having had one at some point in their lives. That is a remarkable fact. We can have the same dream multiple times. Given that brain structure and activity changes constantly, how does it manage to produce the same content repeatedly? Most people who have recurrent dreams insist that the content is identical across repetitions. This means that not merely images are repeated but scenes and dramatic action events, such as being chased by a monster or being threatened by an animal and so forth. Note also that some recurrent dreams are not negative or scary dreams. Some recurrent dreams just

repeat mundane scenes or locations or events. Nevertheless, scholars who study recurrent dreams insist that they are related to unresolved emotional difficulties in the dreamer's life. Retrospective accounts of recurrent dreams experienced during childhood suggest that almost 90 percent are described as being unpleasant or of a threatening nature. But as people grow older, fewer recurrent dreams are reported as having threatening content. Up to 40 percent of adult recurring dreams were composed of nonthreatening content (e.g., descriptions of places, mundane activities, or acquaintances), while nonthreatening content occurred in only 10–15 percent of childhood recurrent dreams.

10.22 Conclusions

All of these atypical dream types must be taken into account when developing a complete account of dreams. A theory of dreams must be able to explain why people think they have mutual dreams, precognitive dreams, and visitations of loved ones from beyond the grave. Simple dismissive explanations that these people are gullible won't do, as the gullibility account does not explain the similar phenomenologies and content features of these extraordinary dreams. The fact that sensorially limited individuals, such as people with blindness, deaf-mute, and paraplegic conditions, nevertheless have dreams where none of these impairments exist must also be explained in any decent theory of dreams. Freud would claim that these dreams confirm his theory of the dream as wish fulfilment, but the wish-fulfillment explanation cannot account for the content of these dreams. The dream stories do not involve dreamers doing things they wish they could do. They involve instead everyday mundane activities. Continuity theories of dreaming also cannot explain these dreams of the sensorially impaired because dream content is clearly not continuous with the everyday lives of these individuals. They engage in walking, hearing, seeing, etc. in their dreams, but they obviously do not do any of these things in everyday life. Nor can the prediction-error theories of dreaming explain these kinds of dreams. The prediction-error account says that perception is a Bayesian process of constructing models of the world that are then corrected by sensory impressions. The model of the world is the prediction or expectation of how the world will be. Dreaming is a construction of model worlds or predictions but without normal sensory input since the visual modality is partially blocked during REM dreaming. There is always error in the model world or prediction, so sensory impressions are used to reduce the error variance around the prediction. Gradually, over time, the error variance is reduced and the models predict the world pretty accurately. The same is the case with dreams, except the model is constructed without nighttime

corrections from the visual modality. These individuals cannot use incoming sensory impressions either in the day or night to build or construct predictive models, including dreams, and yet they still dream.

In the next chapter we will discuss some of the major theories of dreaming and gauge the extent to which they can account for all of the facts concerning dreams.

10.23 Review Questions

- How do the dreams of people living in traditional societies differ from the dreams of people living in modern societies?
- Why might dreams contain indicators or early warning signals of imminent physical illness?
- How might visitation dreams or sleep paralysis dreams have influenced premodern cultural ideas of the supernatural realm?
- What is the significance, if any, of false-awakening dreams for theories of consciousness?

Further Reading

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CHAPTER ELEVEN

Theories of Dreaming

Learning Objectives

- Become acquainted with current evidence-based theories of dreaming
- Evaluate strengths and weaknesses of the social simulation theory of dreaming
- Evaluate strengths and weaknesses of neuroscience evidence in building theories of dreaming
- Evaluate the significance of the fear extinction and the affective network dysfunction model of nightmares and dreaming

11.1 Introduction

At the dawn of the twentieth century, Freud presented his theory of dreams in his landmark work *The Interpretation of Dreams*. Freud's basic claim was that the dream was a hallucinated wish fulfillment. Recent memories and imagistic fragments called day residues provide raw material for dream images that then activate motivated content and affects or wishes, and these wishes conflict with the waking ego and so must be disguised by the dream censorship mechanisms. The dreamwork mechanisms (condensation, representation, displacement, etc.) take the basic content carrying the desire or motivational wish and construct elaborate disguises around it (via secondary revision) while still attempting a hallucinated fulfillment of the wish. Up until the discovery of REM sleep in 1953, most scholars and scientists studying dreams operated within this Freudian framework. Carl Jung broke with the framework and presented his own theory of dreams as simulations that compensate for some aspect of the personality or psychic structure of the individual. Jung also postulated the appearance of mythic archetype in dreams, consistent with Freud's claims concerning reenactments of the Oedipal tragedy and transgression in dreams.

Right after World War II, Calvin Hall began to develop techniques to reliably tabulate the basic contents of dreams. He argued that to test Freud and Jung's dream theories, we needed to establish some reliable numbers around basic content indicators such as number and identity of characters,

background settings, social interactions, objects, emotions, and so on. He collected thousands of dream reports and basically counted each instance of all of these sort of dream categories. He summarized two decades of work in his *The Meaning of Dreams* (Hall, 1966). He argued that his results basically confirmed Freud's theory regarding the dream as a text or story that encodes and symbolizes some psychic conflict generated by unfulfilled wishes. Robert Van de Castle later finalized the development of the scales Hall invented and today we call the gold-standard dream-content scoring system the Hall/Van de Castle system.

After the discovery of REM sleep at the half-century mark (1953), theories of dreaming began to incorporate provisional explanations of REM biology as well. Early on, investigators proposed that REM provided an arousal or preparatory phase to a brief awakening during the night, which served a kind of sentinel or vigilance function for the animal who otherwise was vulnerable to predation while it slept. Hallucinated simulations of threat while the animal slept would also help prepare the animal for defense in case of attack. Jouvet (1980, 1999) proposed that REM supplied the endogenous stimulation necessary for a resculpting or reprogramming of synaptic circuits that support epigenetic behavioral rules/strategies. Jouvet had placed lesions in the brainstems of cats that abolished the atonia usually associated with REM. When the cats' motor systems were no longer inhibited, they appeared to act out "dreams" when electrophysiologic signs of REM appeared. These acting-out episodes typically involved primary instinctual behaviors, such as fear and rage postures, as well as orienting reflexes and the like.

11.2 AIM Theory and Hobson

In the 1960s and 1970s, Allan Hobson and his associates began to map out the neuronal networks that support REM initiation and cessation. They explicitly argued that you could derive the formal properties of REM dreams from the underlying neurological machinery of REM sleep. The activation-synthesis theory updated in 2000 by Hobson et al. (2000b) as the Activation-Input Source-Neuromodulation Model or AIM model of dreaming, summarizes decades of empirical work on the neuroscience of REM sleep and dreams. The activation-synthesis and AIM models begin with the fact that REM sleep is characterized by burst-like, random brainstem and basal forebrain cholinergic activity, while noradrenergic and serotoninergic modulation basically ceases during REM sleep. High levels of cholinergic activation accompanied by monoaminergic demodulation were hypothesized to result in the bizarre and vivid hallucinatory activity that we call dreams. The activation-synthesis name came from the idea that limbic and sensorimotor sites of the forebrain engage

in a kind of reactive attempt to produce a coherent experience from the barrage of otherwise chaotic impulses arising from brainstem activation and PGO waves. The forebrain attempt to synthesize some sort of story out of the impulses generated by brainstem REM-on networks resulted in confabulatory generation of dream scenarios. As in the activation-synthesis model, the AIM model rests on an activation component, again centered in the brainstem but now including thalamic and forebrain sites as well. Aminergic and cholinergic interactions in the brainstem LDT/PPT are retained as one factor in the regulation of REM expression. Unlike the original model, gabaergic, adenosinergic, and histaminergic influences are allowed to influence REM-on and REM-off networks. Now cortical activation ("A" in the AIM title) in addition to brainstem activation is given great weight in the AIM model. Cortical activation allows for efficient access to significant amounts of stored information during dream synthesis. Dream construction depends on access to internal ("I") a source of information. As in the older activation-synthesis model, the shift of the brain from aminergic to cholinergic neuromodulation ("M") reduces stability of cortical circuits in dream construction, thus increasing the likelihood that dreams will contain bizarre elements. The three AIM brain states can be thought of as inhabiting points in the space of a three-dimensional cube defined by these three axes. REM is situated at the high end of the activation axis, the low (internal) end of the input source and the high end of the modulatory cholinergic (low end of the aminergic) axis (see Figure 11.1). Waking consciousness shares REM's high activation level but switches to an external input source and to high aminergic modulation. AIM predicts that lucid dreaming must be a mixed or hybrid state of REM plus wake. Finally, NREM lies midway in the cube space with intermediate values along all three axes.

One drawback of the AIM model is that the activation component of the model has to somehow produce selective activation patterns in the forebrain and cortex rather than global activation levels. REM, after all, involves selective activation of limbic, amygdalar, and parietal networks and relative deactivation of dorsolateral prefrontal cortex (DLPFC). Yet, the AIM model only specifies global activation levels in its account of brain state changes. It is also unclear how global activation of the forebrain can result in reduced activation levels in the dorsal prefrontal cortex during REM. Hobson et al. appear to believe that bringing in the neuromodulatory influence (reduction of aminergic input/enhancement of cholinergic input) can account for selective deactivation of DLPFC. With regard to AIM's account of lucid dreams, there is reason to believe that lucid dreaming is not a hybrid sleep state but an entirely new, unique brain state not adequately captured by AIM – or any other account thus far.

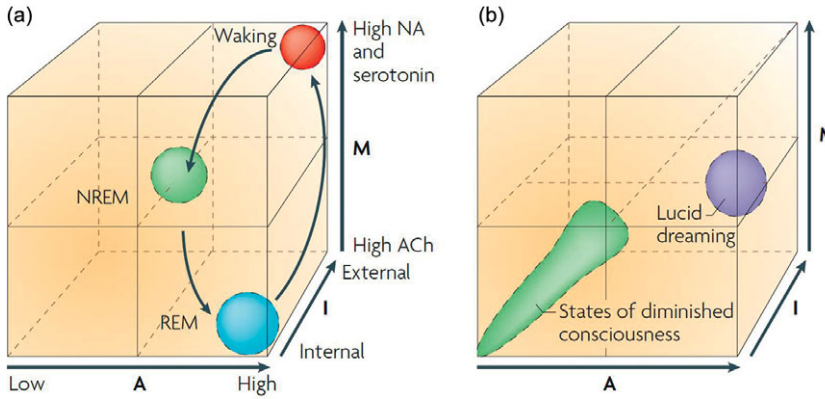

Figure 11.1 AiM model of brain–mind state control

a | The three-dimensional AIM state-space model showing normal transitions within the AIM state space from waking to non–rapid eye movement (NREM) and then to rapid eye movement (REM) sleep. The x axis represents A (for activation), the y axis represents M (for modulation) and the z axis represents I (for input–output gating). Waking and REM sleep are both in the right-hand segment of the space, owing to their high activation levels, but they have different I and M values. NREM sleep is positioned in the center of the space because it is intermediate in all quantitative respects between waking and REM sleep. During sleep, AIM values tend to follow elliptical trajectories through the space. As sleep advances in time, AIM values go less deeply into the NREM sleep domain and more deeply into the REM sleep domain. **b** | Diseases, such as those neurological conditions that produce coma and minimally conscious states, are arrayed in the left-hand segment of the space, owing to their low activation values. Lucid dreaming, which is predicted to be a hybrid state with features of both waking and dreaming, is situated in the middle of the extreme right-hand side of the AIM state space between waking and REM.ACh, acetylcholine; NA, noradrenaline.

Used with permission from Hobson (2009)

11.3 Dreams as Virtual Simulation and Predictions of Reality

More recently, Hobson has collaborated with Karl Friston (Hobson & Friston, 2012) to produce a new theory of dreams that builds on Hobson's previous work and Friston's work constructing aspects of the predictive processing framework (PPF) of brain function. Friston's version of PPF is rooted in the "free-energy" interpretation of biological systems (Friston, 2010). The Free Energy Principle asserts that living systems are compelled to resist the entropic dissolution entailed by the second law of thermodynamics, and that they achieve this goal by seeking to minimize surprise within their environment. A computation or series of computations must be performed by the brain to minimize surprise and

bind more free energy. Perception is consequently reconceptualized as a form of active inference, in which organisms try to engage in patterns of action that will enable them to remain within a set of states that guarantee survival. When applied to the brain, predictive processing offers an alternative to models of the brain that view it as a passive receiver of sensory signals. Instead, cognition is depicted as actively unfolding in hierarchically nested sets of Bayesian networks that are continuously generating hypotheses tested against inputs coming from the levels below them, all the way down to the level of sensory input (Hohwy, 2013; Clark, 2015). The brain, in short, is continuously constructing models of what to expect in the world and then correcting and updating these models by searching out errors between expected versus obtained sensory sampling. Cognitive processing is concerned with detecting discrepancies and explaining away error signals in order to construct better models of the world. Now, as Hobson and Friston note during REM sleep, some portions of sensory sampling of the external world are blocked to some extent. Thus, model updating can proceed without interference from new error detection.

The Hobson-Friston theory of dreaming formalizes a conception of the dreaming brain as a simulation machine or a virtual reality generator that seeks to optimally model and predict its waking environment and needs REM sleep processes (particularly PGO waves) to do so. The basic idea is that the brain comes genetically equipped with a neuronal system that generates a virtual reality of the waking world during REM sleep because REM sleep processes are essential to optimizing this generative model. Treatments of the mind/brain as a virtual reality machine or a prediction-error device or a "Helmholtz machine" (all roughly the same thing) are rife throughout the cognitive and neurosciences, and it makes a lot of sense to consider dreaming along these lines as well. A dream, after all, is experienced as a fully realized "world" that appears to be generated internally without benefit of current sensory input (as visual input is blocked during REM). Hobson and Friston suggest that sensory data are sampled during wakefulness to build-up a complex model of the world that can guide behavior and reduce prediction error and surprises. Then the model is taken offline during sleep and is subjected to an optimization procedure that prunes redundancy and reduces complexity, thus improving the model's fit to the world.

During waking life, changes in the model's parameters (experienced subjectively as percepts) are driven by the need to explain unpredicted visual input. During dreaming, however, there is no visual sensory input, so dreaming percepts are driven by the need to explain unpredicted oculomotor input. Dream content, therefore, is the brain's attempt to find plausible explanations for fictive visual searches triggered by oculomotor input (via rapid eye movements and

PGO waves, presumably) and by the pruning of synaptic connections that are part of the complexity reduction optimization process. The authors also suggest that their theory sheds some light on the lapse in thermoregulatory reflexes that are characteristic of REM. The reversion during REM to a poilkothermic state somehow facilitates minimization of brain entropy.

To bolster the argument that offline optimization is necessary, the authors argue that without periodic offline repair (pruning), the model will become overly complex and dysfunctional, thus reviving the old idea of Francis Crick that REM dreaming represents a purging or pruning of superfluous associations and complexity in the cognitive system. In any case, the general idea appears to be that model updating is reflected somehow in dreams. The PPF model of dreaming is very promising, as it would unite the REM dreaming field with the larger cognitive neuroscience field that has wholeheartedly adopted PPF. We have seen that dreams are highly structured narratives that exhibit typical content across all kinds of populations. If they were mere reflections of individual model updating, they would have to be far more idiosyncratic in content than they are. PPF may be able to handle this objection by saying that long established "priors" (like the sense of self, personality, long-term memories, etc.) are normally not subject to updating. Nevertheless it seems plausible that individual model updating would involve very idiosyncratic episodic memories, and thus, dreams would reflect those kinds of cognitive elements. Dreams do not merely reflect neurobiologic processes. They help shape those very processes themselves.

11.4 Solms

The neuropsychoanalyst Mark Solms (1997) pointed out that REM sleep is neither necessary nor sufficient for dream generation. Therefore, one needed to identify an additional set of brain circuits that participate in dream generation and the creation of dream phenomenology. Solms used classical neurologic lesion–correlation methods to identify the set of brain lesions associated with dream generation, phenomenology, and dream recall. The cholinergic brainstem and basal forebrain mechanisms that generate REM states needed to be supplemented by mesolimbic dopaminergic mechanism. Moreover, specific forebrain structures including higher order association cortices, like the cortical areas surrounding the temporo- parieto-occipital junction (Brodmann area 40), the medial temporo-occipital cortices, as well as the deep white matter structures of the frontal lobes, were shown to be significant for the emergence of dreaming.

Solms (1997) submitted a questionnaire on recall of dreams to 332 patients with various types of cerebral lesions (and 29 non-lesioned controls). Reports of global cessation of dreaming were associated with lesions in the region of the inferior parietal lobes on either side or with lesions deep to medial frontal region in the white matter tracts connecting the frontal lobes with both cortical and subcortical sites. These lesions would presumably disconnect the anterior frontal cortex from subcortical and limbic sites, preventing generation of dreaming. The dopaminergic tracts that predict reward-related incentives (the appetitive and expectancy circuits) carry the motivational and wish-fulfillment aspects of dream content.

Solms' model of dreaming postulates a crucial role for what he calls the appetitive, expectancy, and curiosity circuits associated with ascending meso-limbic-cortical dopaminergic circuits. These dopaminergic circuits project from basal ganglia and limbic sites to mediobasal and prefrontal cortex. Independent evidence suggests that meso-cortical catecholaminergic circuits are crucial for predicting reward and are therefore involved in motivational aspects of behavior. Solms claims that activation of these dopaminergic circuits instigates the dream formation process. Since Solms agrees with Freud that protection of sleep is one of the functions of dreaming, he provides a scheme for propagation and damping of activation levels away from anterior regions to posterior regions. REM-associated activation of anterior limbic sites are hypothesized to simultaneously prevent activation of motor cortex and to facilitate a process Solms calls back-propagation. In back-propagation, the activation levels in dopaminergic circuits are kept from prefrontal, supplementary motor, and premotor areas and then rerouted posteriorly to sites in the inferior parietal lobes and occipital-temporal visual association areas. The sleeper can then safely experience a visual simulation (hallucination) that satisfies a wish and prevents awakening without becoming fully conscious or motorically active.

While Solm's model is based on well-documented clinical data, the back propagation process is ill-defined and difficult to identify as actually occurring during dreaming. If back-propagation truly occurs and functions to protect sleep, one would expect loss of dreaming to result in waking or at least poor sleep. While Solms claims that some of his patients do indeed report poor sleep, it is not clear that this is due to loss of dreaming per se, or to their medical condition. I know of no evidence that suggests that patients with frontal leucotomies or bilateral parietal lesions are rendered permanently awake. Indeed, Jus et al. (1973) demonstrated that REM occurred in leucotomized patients who nevertheless reported very few, if any, dreams.

Foulkes (1985) argued that dreams are "credible world analogs" or imaginative simulations of waking life that obey fundamental rules of waking

cognition but that to a great extent lacked reflective thought. Although he argued that dreaming plays a role in development of consciousness, he has also argued that dreams likely serve no adaptive function. Foulkes recommends that we focus more on the formal cognitive features of dreams rather than on dream content per se. Foulkes proposed that dreaming involved a diffuse activation of mnemonic material and as such, could serve no adaptive function. Yet as Foulkes himself points out, dream content is not random. The dreamer is typically represented as playing a central role in the dream narrative, which typically involves a summation of selected past experiences personal to the dreamer. The past experiences inform the dream events – the dream is not a mere replay of these past experiences. Instead it is cognitively creative.

Hartmann (1998) suggested that dreams are the product of spreading excitation between semantic nodes in a semantic network, except that the patterns of activation in dreams are guided by current emotional concerns and make connections between concepts more broadly and more inclusively than does waking cognition. Hartmann suggested that certain dream images function to contextualize intense emotions. This latter capacity allows dreams to facilitate integration of traumatic or overwhelming emotions.

11.1.4 Emotional Processing Functions of Dreams

Hartmann's position is similar to many contemporary views on functions of dreaming that emphasize potential emotional functions of dreams. All these investigators provide substantial evidence for adaptive emotional problem-solving and processing in dreams. Kuiken and Sikora (1993) argue that impactful dreams reflect activations of components of the classical orienting response and that these emotionally intense, impactful dreams have lasting effects on the mood states of waking experience.

11.5 Fear Extinction and the Affective Network Dysfunction (AND) Model

Tore Nielsen and Ross Levin Nielsen and Levin (2007) formulated a new neuro-cognitive model of nightmares that implicitly carries a basic theory of dreams as well. The AND model (Figure 11.2) suggests that normal REM dreaming involves the stripping away of contextual material from images laden with fear. This stripping process allows the fear image to be more efficiently integrated into long-term memory. REM–supported emotional memory consolidation, in this view, essentially consists in defanging, so to speak, affect-laden images so that they can be stored into long-term memory. That defanging process involves the stripping of context information from the

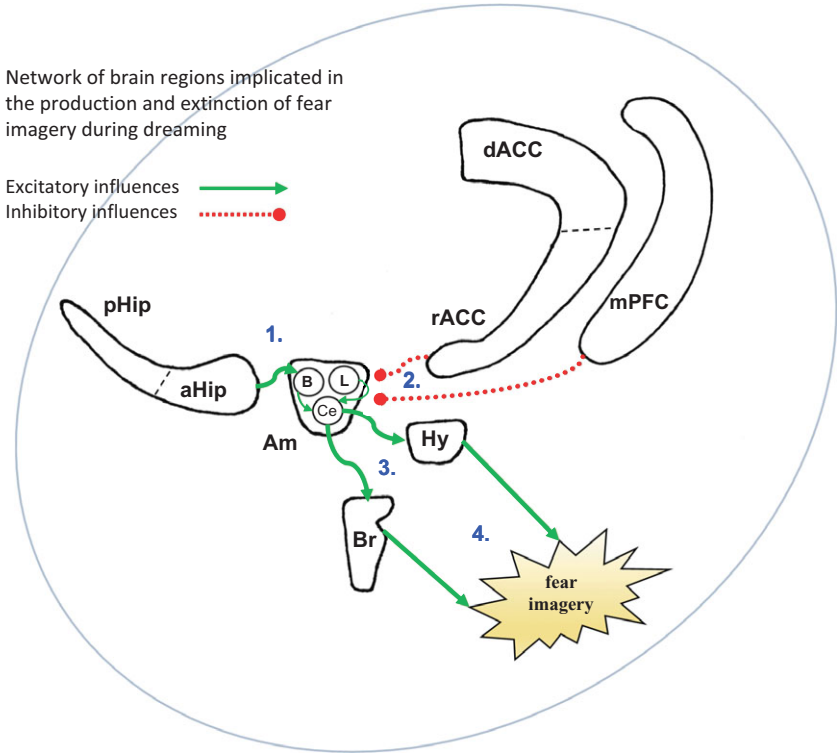

Figure 11.2 Schematic representation of one possible set of neural interactions supporting the AND model

(1) Hippocampal contextual information is relayed in realistic (virtual) form via anterior hippocampus (aHip) to basal nucleus (B) of the amygdala (Am), where it is further processed by central (Ce) nucleus; (2) medial prefrontal cortex (mPFC) and dorsal (dACC) and rostral (rACC) anterior cingulate cortex afferents to lateral (L) and Ce amygdalar nuclei regulate the output of Ce neurons to induce extinction, to signal distress, and to maintain appropriate levels of fear; (3) the Ce nucleus signals brainstem (Br) and hypothalamus (Hy) circuits, producing (4) the autonomic and behavioral correlates of fear within the dream. Excitatory connections are in solid lines; inhibitory connections are in dashed lines.

Used with permission from Nielsen and Levin (2007)

affect-laden memory fragment and then the fragment is ready for long-term storage. Image contexts are mediated by activation of the hippocampal formation. Fear extinction occurs after contextual information is stripped from the affect-laden images or memory fragments. Nightmares and recurrent dreams occur when the stripping or decontextualization process breaks down, and then the affect-laden image fragment remains in short-term memory stores where it gets reactivated periodically whenever a semantically related cues activates it.

11.6 Continuity Hypothesis (CH) on Dreams

Schredl (Schredl & Hoffman, 2003) and Domhoff (1996) have been the foremost advocates for the idea that dreams simulate the kinds of things we do and encounter in everyday life. The continuity hypothesis of dreams suggests that the content of dreams is largely continuous with waking concepts and concerns of the dreamer. Calvin Hall was the first dream researcher to argue that some content of dreams reflected the daily concerns and ideas of the dreamers rather than the hidden libidinal wishes or compensatory emotional strategies that psychodynamic theorists such as Freud and Jung advocated. Through creation of standardized dream–content scoring inventories (building on the work of Mary Calkins and others), Hall demonstrated that the most frequently appearing content items of dreams were not bizarre images at all but rather mundane social interactions between the dreamer and people he or she interacted with on a daily basis. One did not need to invoke theories concerning elaborate dreamwork to disguise latent libidinal and aggressive wishes buried in the dream.

Instead, simple counts of characters, interactions, objects, actions, and events in the dreams could yield a pretty accurate picture of what the dream was about and it wasn't dramatically different than the daily life of the dreamer. Many dream researchers since Calvin have confirmed that the bread and butter of dreams are the quotidian daily social interactions and concerns most people experience on a daily basis. Domhoff's (2003) impressive content analyses of a longitudinal dream series collected from a middle-aged woman dubbed "Barb Sanders" very convincingly shows that her pattern of aggressive and friendly interactions with key characters in her dreams matched the ups and downs of those same relationships in waking life.

Thus, the empirical support for some degree of continuity between dream content and waking life is strong. The database supporting the theory has been considerably strengthened by many dream researchers over the years, going back to Hall's pioneering efforts in the 1950s, 1960s, and 1970s. It is therefore clear that any complete theory of dreams must accommodate the data demonstrating substantial continuities between dream content and waking concepts and concerns.

But as every supporter of continuity theory acknowledges, there are also dreams that contain some significant discontinuities between dream content and waking concepts/concerns. For example, most people have had dreams that are like long adventure stories or movies. These "narrative-driven" dreams are less quotidian than everyday dreams. They contain more bizarre elements and imagery and have the dreamer engaged in actions and events that are

decidedly not like their ordinary ideas, actions, and concerns. In addition, there is a significant minority of dream reports that have few or no familiar characters, settings, or activities. Can these sorts of dreams be explained with continuity theory approaches? If attempts are made to do so, how can one avoid special pleading, circular reasoning, or ad hoc additions to the theory?

Domhoff (2003) draws on Solms' data, the recent set of findings gathered from neuroimaging studies, and his own extensive studies on dream content to propose a detailed neurocognitive model of dreaming. Domhoff suggests that dreaming depends on brain activation patterns, but the content of dreaming come from access to the conceptual system of schemata and scripts housed in the cognitive system. The convergence of these data sources indicate large-scale but selective neural networks responsible for dreaming, including the brainstem generator, structures in the hypothalamus and thalamus, amygdala, limbic system, anterior cingulate, and cortical sites. Domhoff and Fox (Fox et al., 2013) would later identify the neural networks responsible for dreaming to be essentially large portions of the default mode network (see Figure 11.3). Domhoff suggests both a "continuity principle" and a "repetition principle" in dream content, noting that there is continuity between waking life and dream life, and there is also a tendency to repeat certain themes in dreams across time. He cites repetitive nightmares, recurrent dreams of childhood and adolescence, and repeated themes found in some dream series elicited from a single individual. Domhoff links this tendency to repeat content to activation of the amgydala's fear-vigilance system. I assume he would largely agree with Nielsen and Levin's AND model of fear extinction functions for dreaming. Dreaming according to Domhoff draws on memory schemata, general knowledge, and episodic memories to produce quasi-veridical simulations of the real world in dreams. Domhoff does not believe that dreaming, per se, has an adaptive function. He therefore parts company with the next theorist of dream content: the evolutionary theorist Revonsuo.

11.7 Dreams Are Hyper-Associative

Many students of dreams have noticed that they seem to promiscuously connect things, concepts, images, ideas, meaning elements, etc. that are only distantly semantically related. Hartmann argued that this ability to think outside the box allowed dreams to operate as a kind emotional therapy for the individual, getting him or her to be a better problem solver. Problem-solving theories of the dream, in general, also rely on this hyper-associative ability of dreams. The most recent model to emphasize dream's capacity for hyper-associativity is

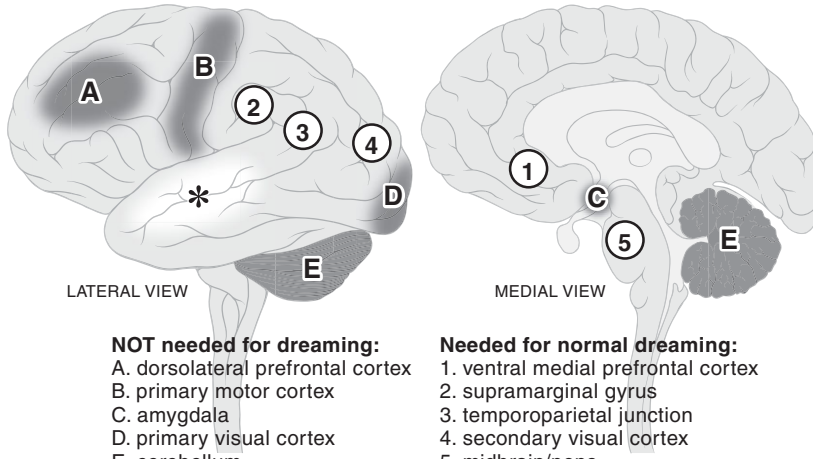

Figure 11.3 Brain areas critical to both daydreaming and dreaming
(Note. The left column shows areas critical to dreaming as determined by overlapping lesion sites based on CT scans of neurological patients. The middle column shows meta-analytic brain activations associated with waking daydreaming/mind wandering.)
The right column shows meta-analytic brain activations associated with REM sleep, which is nearly always accompanied by dreaming. All three approaches converge on three areas as being central to dreaming: the medial prefrontal cortex (panels a–c), the temporoparietal junction/anterior inferior parietal lobule (panels d–f), and the medial occipital lobes/lingual gyrus for the visual aspects of dreaming in particular (panels g–i). Panels a and d modified with permission from Solms (2000); panel g modified with permission from Solms (1997). Panels b, e, and h based on data from Fox et al. (2015). Panels c, f, and i based on data from Fox et al. (2013). X and Z values represent left–right and vertical coordinates, respectively, in stereotactic space.

Used with permission from Domhoff and Fox (2015)

Stickgold and Zadra's "Network Exploration to Understand Possibilities" (NEXTUP) model (Zadra & Stickgold 2021). NEXTUP suggests that dreams allow us to explore the brain's neural network connections in order to understand possibilities – particularly (as I read the model) uncommon possibilities. Similarly McNamara (2000) argued that most dreams are actually structured like counterfactual scenarios riffing over and over again on a central surprise or problem element. Each new counterfactual scenario explores some new realm of unexplored possibilities of the problem space. For NEXTUP, dreaming is a form of sleep-dependent memory processing that extracts new knowledge from existing information through the discovery and strengthening of previously

unexplored associations. The dream combines memories into a dream narrative that explores associations the brain would never normally consider. In doing so, dreams are endlessly creative.

Llewellyn (2021) also offers a dream theory based on dream hyper-associativity. She reviews the extensive scientific evidence for this simple fact and lays out for the reader the immense implications of this ability of REM dreams to hyper associate – dreams are essential for memory, insight, creativity, anticipation/prediction, and mental well-being.

11.8 Empathy Theory of Dream Sharing

It is a remarkable fact that most of us occasionally share dreams with others and some of us engage in dream sharing quite often. It is very likely that our paleolithic ancestors did quite of lot of dream sharing if we can take premodern and modern accounts of traditional hunter-gatherer tribes as models. Blagrove and colleagues took the act of dream sharing as the beginning of their (2020, 2021) *Empathy Theory* of *Dreaming*. They propose that dreams function, in part, to facilitate social cooperation via creation of empathy in others who hear our dreams. While the empirical base for this theory is still as yet thin, that should change fast as more studies of dream sharing motivations and effects are undertaken. It stands to reason as well that the empathy theory would sit well with social simulation theories of dreaming.

11.9 Threat Simulation Theory

Revonsuo (2000) proposes that the function of dreaming is to simulate threatening events. Threat simulations are thought to allow for rehearsal of threat perception and thus to enhance threat avoidance. Revonsuo notes that dream content is not random but instead consistently overrepresents simulations of unpleasant/threatening events. In some cases these simulations are repetitions of previous unpleasant dreams (Domhoff's repetition principle), such as a nightmare of being chased by a wild animal, etc. Repeated simulations of such threatening events in the dreaming mind of an ancestral human is thought to confer a selective advantage on that individual, relative to individuals not experiencing such simulations. The advantage might be a slightly faster response time when confronted with a threat or a slightly improved ability to detect an incipient attack and so forth. Children in particular would benefit from such a threat simulation device, and studies of dreams of modern children show that wild threatening animals are overrepresented in their dreams. Nightmares and post-traumatic repetition dreams are thought to be

instances of disinhibition of the threat simulation device. While threat simulation can nicely account for some aspects of some dream content, most dreams cannot be assimilated to threat simulation. There are too many dreams that are simply not about threats and/or not even unpleasant. An equally pressing necessity for ancestral humans had to do with interactions with other members of the group: how to find a mate, how to avoid conflict, and how to build coalitions and so forth. This "social simulation theory" of dreaming suggests that dreams also simulate social interactions, and if simulating these interactions in dreams enhanced fitness outcomes for the individuals who experienced such dreams, natural selection would have "supported" development of such dreams, as seems to be the case.

11.10 Social Simulation Theory (SST)

Many authors have remarked on the probable social functions of dreaming. Anthropologists have long treated the dream as a strategic social act; that is, dreams are used in traditional societies to facilitate negotiations in social alliances and to facilitate change in the social status of the dreamer. Dreams are known to be involuntary experiences and thus, they are hard to fake, and if they are hard to fake, then they can be considered honest signals concerning the dreamer's capacities and intentions. Freud (1900, 1950) and many authors in the psychoanalytical tradition can be read as supporting a kind of SST given that they often interpret dreams in terms of emotional conflict in families of origin or in current families, as well as between sexual partners and romantic targets. Revonsuo, Touminin, and Valli (2015) have marshaled some of the data and arguments that support the SST. According to Touminin (2022), SST postulates that dreams virtually simulate socially significant interactions for the dreamer; that is, they simulate human social reality, including the social skills, bonds, interactions, and networks that we engage in during our waking lives. These claims logically entail the following hypotheses:

1. *Sociality Bias:* Social interactions should appear more frequently in dream life than in a corresponding stretch of waking life.
2. *Exaggeration Hypothesis*: Strangers or unfamiliar people should be overrepresented in dreams as compared to waking life, to simulate and rehearse perceptual categorization (familiar vs. unfamiliar)
   Comment: This hypothesis may not be well-motivated given that the relevant literature has traditionally linked strangers in dreams to character lines inviting transformations OR to character lines whose eventual characters initiate physical aggression of one kind or another.

3 *Strengthening Hypothesis:* The function of social simulations in dreams is to maintain and strengthen the dreamer's most important social bonds from waking life.
4 *Practice and Preparation Hypothesis*: The function of social simulations in dreams is to force the dreamer to practice important social bonding skills, such as how to give social support to others.
5 *Mindreading Hypothesis*: Dreaming specifically simulates mindreading or mentalizing abilities.
6 *The Compensation Hypothesis*: When social interactions in waking are radically reduced, dreams begin to compensate by generating more social simulations to ensure group inclusion.

Touminin (2022) tested these hypotheses in a series of publications using different data sets. Study II utilized an existing dataset, collected in the 1990s at the Department of Psychiatry at Harvard Medical School in the United States. The dataset contained both wake and dream reports, with dream reports collected from both REM and NREM stages throughout the night. The full dataset consisted of hundreds of dream reports collected over two weeks from sixteen undergraduates (eight women, age range 19–26) (Stickgold et al., 2001). The author content analyzed the dreams, looking for content data either consistent or inconsistent with SST hypotheses. In study III, eighteen volunteers participated in a five-day island retreat where the participants spent three days in voluntary social isolation, without social interaction nor access to phones, television, or the Internet. They filled out dream reports and completed questionnaires daily.

In study II, dreams were found to be more social than waking life; social interactions were controlled by imposing social isolation or seclusion on some participants. The effects of this seclusion were then analyzed. While the number of social interactions did not change as a function of seclusion versus baseline, dream reports nevertheless tended to be more social than the preceding baseline reports as well as the subsequent post-seclusion reports. Specifically after seclusion, appearance of familiar versus strangers were altered relative to baseline. A key finding from this social isolation design is that in study III, REM indices were increased as a result of the isolation procedure.

In study II, the author also looked at social interactions in REM and NREM reports using the same dataset that Stickgold assembled and that McNamara et al. (2005, 2010) used. In the McNamara studies, they found that dreamer-initiated aggressive interactions were more frequent in REM dreams, while friendly interactions were more frequent in NREM dreams. The author claims not to have replicated this finding given that the dream self was not found to engage in greater amounts of positive or negative social interactions with close

others or strangers. But aggression is not the same as "negative interactions." In addition, McNamara used the validated Hall/Van de Castle scoring system, while the author used a recently devised social interaction scale.

In short, Touminin (2022) found some support for all six SST hypotheses. It is clear that there is substantial empirical support for SST.

Other authors have advanced similar accounts of dreaming as SST. Brereton (2000; see also Franklin & Zyphur, 2005) presented a similar idea in his "Social Mapping Hypothesis" that suggests that dreaming allows for rehearsal of emotional and perceptual abilities needed for relating the dreamer to emotionally significant others and social groups. Obviously the social mapping hypothesis and the SST is consistent with the argument I have been presenting in this book concerning the social functions of sleep and dreams. Indeed, I believe the SST is currently the best-supported theory of dreams that we have. Revonsuo et al. note (in support of SST) that 95 percent or more of dreams are populated by the dreamer who interacts with two to four other characters, most of whom can be recognized as familiar characters in the dreamer's immediate social network. Friendly interactions (typically verbal conversations) are found in about 40 percent of dreams, while aggressive social interactions occur in about 45 percent of dreams. In addition, mind-reading or inferring the mental states of others, particularly those characters the dreamer interacts with, occurs in more than 80 percent of dreams. Finally, people who are most important in the dreamer's waking network regularly appear in their dreams. Thus, existing data from dream-content studies is certainly consistent with SST, and I have tried to show in this book that the existing evolutionary theory as well as the data on sleep neurobiology is also consistent with SST.

11.11 Conclusions

All of the aforementioned theories of dreaming are supported by a good deal of evidence in the dreaming literature, but all of them cannot be correct as they currently stand. It is likely that elements of each contain the building blocks of an ultimate theory of dreaming. That ultimate theory of dreaming must also account for dream variation as summarized in the previous chapter as well as the facts on changing dream content with differing stages in the life cycle. Any theory of dream function must also be consistent with the available neuroscience data on the neuroanatomy of dream recall as well as the neurology of REM and NREM sleep states. Although the neuroscience of sleep and dreams has made tremendous progress in the past few decades, it still has not matured to the point where research is theory guided. Nevertheless, the social hypothesis described in these pages is evolutionarily grounded and at least provides a

heuristic theoretical framework of REM sleep and dreams that can be tested and falsified experimentally in the lab in the years to come. If it is falsified then the field can move beyond the heuristic framework to investigate other potential frameworks, and if it is supported then it will provide the field with theory-guided research questions instead of blind data–driven research programs for years to come.

11.12 Review Questions

- What are the strengths and weaknesses of the continuity hypothesis of dreaming?
- Can any current theory account for the great variety of dream experiences discussed in previous chapters?
- What theory of dreaming is most consistent with neuroscience data concerning dream recall and brain activation patterns during REM?
- Current theories of dreaming all assume that dreaming performs some function for daytime consciousness. What might be evidence for the claim that dreaming performs some function for sleep and dreaming rather than waking consciousness?

Further Reading

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