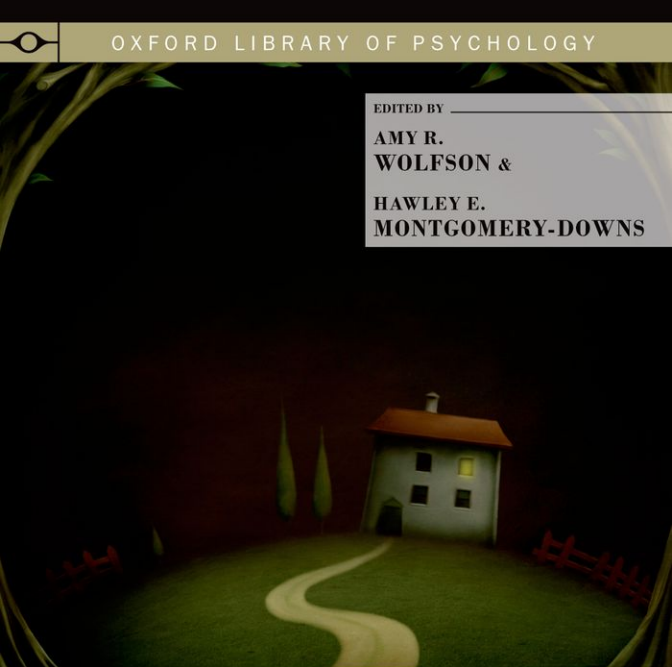


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ADOLESCENT  
SLEEP *and* BEHAVIOR

The Oxford Handbook of Infant, Child,  
and Adolescent Sleep and Behavior

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# The Oxford Handbook of Infant, Child, and Adolescent Sleep and Behavior

*Edited by*

Amy R. Wolfson

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Oxford University Press is a department of the University of Oxford.  
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Published in the United States of America by  
Oxford University Press  
198 Madison Avenue, New York, NY 10016

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Library of Congress Cataloging-in-Publication Data  
The Oxford handbook of infant, child, and adolescent sleep and behavior / edited by  
Amy R. Wolfson, Hawley Montgomery-Downs.  
pages cm.—(Oxford library of psychology)  
Includes bibliographical references.  
ISBN 978-0-19-987363-0

1. Sleep disorders in children—Handbooks, manuals, etc. 2. Sleep disorders in adolescence—Handbooks, manuals, etc.  
3. Children—Sleep—Handbooks, manuals, etc. I. Wolfson, Amy R. II. Montgomery-Downs, Hawley.  
III. Title: Handbook of infant, child, and adolescent sleep and behavior.  
RJ506.S55O947 2013  
618.92'8498—dc23  
2013003516

9 7 8 6 5 4 3 2 1  
Printed in the United States of America  
on acid-free paper

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The *Oxford Library of Psychology*, a landmark series of handbooks, is published by Oxford University Press, one of the world's oldest and most highly respected publishers, with a tradition of publishing significant books in psychology. The ambitious goal of the *Oxford Library of Psychology* is nothing less than to span a vibrant, wide-ranging field and, in so doing, to fill a clear market need.

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Dr. Montgomery-Downs is an Associate Professor of Psychology in the Behavioral Neuroscience program at West Virginia University. She studies the developmental psychobiology of pediatric sleep disordered breathing and postpartum sleep disturbance. She has published over 30 papers and her work has been supported by the United States National Institutes of Health.

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# Introduction to *The Oxford Handbook of Infant, Child, and Adolescent Sleep and Behavior*

Amy R. Wolfson

## Abstract

This introductory chapter outlines the history and the theoretical basis for studying child and adolescent sleep and development. Furthermore, this chapter celebrates the progress the field has made in the last few decades, and makes the case for the crucial role sleep plays in child development. The chapter describes the organization of the handbook, outlining the many foundation and topical areas of sleep and child development covered in the text.

**Key Words:** child, adolescent, sleep, behavior, development, psychology

Welcome to *The Oxford Handbook of Infant, Child, and Adolescent Sleep and Behavior* that has the honor of being a part of the Oxford Library of Psychology. Recognition of the significance of sleep in the cognitive and behavioral development and emotional well being of infants, children, and adolescents is relatively recent. Over the last three decades, there has been an explosion in research focused on developmental changes in sleep and circadian rhythms from birth through adolescence. Further study has led to the characterization and assessment of sleep disorders unique to different developmental stages. Links between sleep problems and overall health, as well as emotional and behavioral disorders including depression, attention deficit hyperactivity disorder (ADHD), neurodevelopmental disorders, and post-traumatic stress disorder, are now more established in children and adolescents.

Ahead of psychologists in the field, graduate programs, and psychology's professional associations, the editors of the Oxford Library of Psychology recognized the importance of including a volume on infant, child, and adolescent sleep and behavior in their Psychology Handbook series. My co-editor and I were thrilled to be a part of this significant and

historic venture. As a child clinical psychologist, I was deeply committed to inviting experimental, clinical, school, and developmental psychologists as well as other behavioral clinicians and social and behavioral science researchers to author and coauthor chapters for what would become *The Oxford Handbook of Infant, Child, and Adolescent Sleep and Behavior*. Psychologists play a crucial role in developing and contributing to what is known as pediatric and adolescent sleep medicine; however, psychology, as a discipline, has only gradually recognized the importance of sleep in child development and developmental psychopathology research and in clinical, counseling, and school psychology undergraduate and graduate education. It is our hope that this first psychology handbook on child and adolescent sleep will inform child and adolescent clinical psychology practice, raise the importance of including child and adolescent sleep as a part of graduate programs in psychology and related fields, and provide future questions and directions for research in psychology and in the behavioral sciences.

As a child clinical psychologist, I have devoted my research program to studying sleep and daytime functioning in infants, children, and adolescents,

as well as pregnant and postpartum women, since my graduate school days in the 1980s. At that time, few psychology graduate programs or individual faculty offered graduate-level courses on sleep or circadian rhythms. Psychologists, however, were beginning to lead a movement toward cognitive-behavioral approaches to treating insomnia. For example, while I was a graduate student in clinical psychology at Washington University in St. Louis (1982–1987), two faculty members, Patricia Lacks and Amy Bertelson, studied sleep in adults. Patricia Lacks developed stimulus control techniques for treating insomnia and wrote one of the first books on the treatment of insomnia, *Behavioral Treatment for Persistent Insomnia* (1987), and Amy Bertelson taught an exciting and popular undergraduate course on sleep. In contrast, scholarship on the development of sleep patterns over the course of infancy, childhood, and adolescence or on understanding and treating children's sleep problems was far less common. Observing my mentors' passion for helping adults improve their sleep and daytime lives, I was determined to study and understand how psychologists could contribute to improving children's sleep. As a graduate student, I began my ongoing, nearly 30-year inquiry into understanding how children's nighttime behaviors and sleep experiences influence not only their own daytime functioning, but also the lives of their parents and families.

Focused on the impact of healthy lifestyles as an approach to preventing mental health and behavioral difficulties in children, I started reading some of the mid-1980s publications, written by parents as well as pediatricians, on infant sleep. In particular, I focused on two parenting books: pediatrician Richard Ferber's *Solve Your Child's Sleep Problems* (2006) and parents' Joanne Cuthbertson and Susanna Schevill's *Helping Your Child Sleep Through the Night* (1985). Utilizing behavioral and social learning theories of behavior change and Cuthbertson and Schevill's insights on self-soothing and infants' developing ability to *sleep through the night*, I developed a preventive-intervention program that gave parents sleep knowledge and strategies along with a sense of self-competence to handle their infant's sleep during the first year. The empirical findings from this preventive approach were published (see Chapters 37 and 39 for summaries) in the *Journal of Consulting and Clinical Psychology* (Wolfson, Lacks, & Fetterman, 1992), launching early attention to parenting and young children's sleep in the psychology literature. It took some time, however, before the child psychology discipline recognized that

there were developmental changes in sleep and clear clinical and behavioral consequences for inadequate and poor quality sleep on developing youth. For example, when I presented my doctoral research on the effects of parent training on infants' sleep patterns at the 1988 Association for the Advancement of Behavior Therapy's (AABT) annual meeting, Jodi Mindell (coauthor of Chapter 37) and I gave the only presentations focused on behavioral approaches for improving children's sleep and at the 1995 biannual meeting of the Society for Research on Child Development (SRCD) only 0.3% of the peer-reviewed posters and presentations were related to children's sleep. In fact, in a shared van ride to that 1995 Seattle meeting of SRCD, a school psychologist asked me *if sleep was developmental?* With that story in mind, it was particularly meaningful to publish "Sleep Schedules and Daytime Functioning in Adolescents" in *Child Development* (Wolfson & Carskadon, 1998) with my coauthor and mentor, Mary Carskadon (coauthor of Chapter 8). Sleep, in striking contrast, is considered a focal area for the 2013 biannual meeting of the SRCD. The publication of this handbook underscores how far the behavioral and developmental sciences have come in recent years in the study, treatment, and public awareness of sleep and behavior in children and adolescents.

Over the last three decades, psychologists, pediatricians, and other health care providers have established an increasingly distinct understanding of children and adolescents' sleep demands, circadian timing, underlying bioregulatory processes, and environmental constraints. At the beginning of this handbook, in Chapters 2 and 3, respectively, Stephen Sheldon provides an overview of the history of child and adolescent sleep medicine and Tonya Palermo offers insights and historical background regarding psychology's historical contributions to infant, child, and adolescent sleep research. Studies have documented the clear consequences of insufficient and inconsistent sleep over the course of development, such as early behavioral and cognitive problems, poor academic performance, substance abuse, and emotion regulation difficulties. This critical area of child development and health research has begun to inspire public policy discussions and debates in areas such as school start times, regulation of preschool napping, and adolescent drivers' education laws and programs. Likewise, much of this research has practice implications and applications not only for infants, children, and adolescents with sleep disorders, but also for those who have no demonstrable

sleep disorders. In other words, school pressures and schedules, family socioeconomic status, and a range of environmental constraints and challenges compromise the sleep of increasingly greater numbers of infants, children, adolescents, and emerging adults. Furthermore, children and adolescents who present with academic and behavior problems may in fact have primary sleep difficulties. The basic research and clinical presentations that are discussed in this handbook have clear implications for the overall health, development, emotional well being, and academic performance of children and adolescents.

The scientific study of sleep is a still a relatively young discipline, but tremendous gains in knowledge have been achieved over the last two decades, including advancing our understanding of sleep's role in memory consolidation and learning processes, associations between sleep deprivation and obesity, and the sleep and the circadian systems' regulatory influence on immune functions (Besedovsky, Born, & Lange, 2012; Knutson, Zhao, Mattingly, Galli, & Cizza, 2012; Walker & Stickgold, 2006). Although the preponderance of research on sleep has been conducted in adult populations, a significant body of knowledge and current research now focuses on the sleep patterns, disruptions, and disorders in infants, children, and adolescents.

This handbook offers a unique and important contribution to the field of child and adolescent sleep patterns, difficulties, and disorders. It provides behavioral and developmental approaches to understanding sleep and circadian timing development, assessment of sleep patterns and problems, etiology of sleep disorders, and preventive/intervention approaches in working with children, adolescents, and their families. We have brought together psychologists and other behavioral and developmental clinicians and developmental scientists to discuss developmental changes in sleep and circadian rhythms; the factors that influence, mediate, and moderate sleep patterns and problems over infancy, childhood, and adolescence; and the assessment and behavioral treatment of infant, child, and adolescent sleep problems and disorders. The authors are from a range of countries and include psychologists, pediatricians, psychiatrists, nurses, child life specialists, and other health care providers and researchers with expertise on infant, child, and adolescent sleep.

Research on children's sleep can be organized into seven areas, which are reflected here. The first section focuses on *Sleep and Development*, since one of the most significant factors affecting sleep is age

or developmental stage. Following the two historical chapters mentioned earlier, Angela Staples and Jack Bates articulate what it means to consider sleep from a developmental perspective, so that developmental psychologists and other researchers might consider the role of sleep and circadian rhythms in development, and to remind sleep researchers how sleep changes from infancy through adolescence. Sleep and circadian rhythms change over the course of development from infancy into the emerging adult years, as highlighted and documented in the chapters by Kurt Lushington, Yvonne Pamula, Alfred James Martin, John Declan Kennedy and by Mary Carskadon and Leila Tarokh in Chapters 5, 6 and 8, respectively. An infant's sleep plays a particularly significant role in the lives of new parents, and Robyn Stremler's Chapter 7 examines the impact of infants' sleep and behavior on parents including marital satisfaction, coping strategies, postpartum depression, and the parents' own sleep-wake patterns. Although this section does not include a separate chapter on elementary school children's sleep, school-age children's sleep is discussed throughout the book with particular emphasis in Chapters 10, 11, 15, 21, 22, 27, and 34. Toward the end of Mary Carskadon and Leila Tarokh's chapter, they discuss an understudied developmental stage: emerging adulthood. Historically, sleep researchers, similar to researchers in other behavioral sciences, have invited college-age students to participate in studies and then generalized to wider adult populations. However, the American Psychological Association considers youth ages 10–18 as adolescents and emerging adults ages 19–29. Psychologist Jeffrey Arnett argues that emerging adulthood, or the “winding road from the late teens through the twenties,” is a distinct and normative developmental or life stage (Arnett, 2004). Recent studies point out that first-year college students exhibit weeknight bed and rise times that are over an hour later than high school-age adolescents, as well as significantly later bed and rise times than older third and fourth-year college students (i.e., Lund, Reider, Whiting, & Prichard, 2010). Similarly, Roennberg, Kuehnle, Pramstaller, and colleagues found that after age 20, sleep midpoint times became increasingly earlier again; in other words, sleep schedules seem to become increasingly delayed over the course of adolescence, yet this pattern seems to change by the third or fourth year of college—which generally corresponds to about ages 20–22 (Roennberg, Kuehnle, Pramstaller, et al., 2004). As I emphasize in my 2010 *Journal of Adolescent Health* editorial,

these remarkable cross-sectional findings suggest a developmental change; however, more research is needed to better understand and inform developmental science researchers, educators, and health care providers regarding the longitudinal trajectory of sleep and circadian timing over the late adolescence / emerging adulthood years (Wolfson, 2010).

Likewise, with each developmental period a myriad of different factors influence an infant's, child's, or adolescent's sleep patterns and, likewise, the sleep environment (i.e., college dorm room vs. family sleep environment), context, and other factors such as culture and socioeconomic status, interact with developmental changes in sleep. I was quite disturbed to learn that a 12-year-old in my National Institute of Child Health and Human Development (NICHD)-funded study of urban middle schoolers was sleeping in his neighbor's apartment until about midnight on school nights until his mother got home to awaken and escort him back to their apartment building for the remainder of the night—not an easy way to obtain the sufficient and consistent sleep needed for a developing adolescent. The second section of the book focuses on the *Complexity of Issues and Factors Influencing Sleep*. Here, authors Charles Super and Sara Harkness, Lauren Hale, Michael Gradisar and Michelle Short, Melissa Burnham, Lisa Meltzer, and James Spilsbury discuss and critique the newest research on the social, cultural, and economic determinants of children's sleep, with focus on the consequences of chronic illness and violent environments, and the rapidly changing role of technology that is interfering with children's and adolescents' ability to sustain healthy sleep-wake patterns. For example, in the National Sleep Foundation's 2011 Sleep in America Poll about in one in ten of 13–18-year-olds (9%) acknowledge that they are awakened after they go to bed every night or almost every night by a phone call, text message, or email, and nearly one in five of 13–29-year-olds note that this happens at least a few nights per week (NSF, 2011). In other words, adolescents' and emerging adults' lifestyle choices are creating a sleep disorder-like phenomenon with smart phones.

Parts three and four reflect the substantial literature on disordered sleep and sleep problems that manifest over the course of infancy, childhood, and adolescence. Section Three, *Assessment of Sleep and Sleep Problems*, articulates the range of clinical and research-based approaches for assessing and estimating sleep and circadian rhythms in infant, child, and adolescent populations. Authors Neville Blampied, Rosemary Horne and Sarah Biggs, Stephanie Crowley,

Karen Spruyt, Valerie Crabtree, Amanda Rach, and Heather Gamble; Lissy Zaremba, and Joseph Buckhalt cover behavioral analysis, use of actigraphy and diaries or logs for estimating sleep patterns, and self-report measures, as well as school psychologists' roles and clinical lab protocols. Although the field has progressed in recent years, validation of retrospective self- and parent report sleep measures, prospective sleep-wake diaries, and actigraphy are still needed. It remains difficult to study children's sleep longitudinally, since few measures have been validated across developmental stages. Moreover, although actigraphy (in corroboration with sleep diaries) is a more objective means to estimate sleep in child and adult populations, researchers and clinicians need to establish unambiguous guidelines and standards for the scoring and reporting of actigraphy findings. Pending such standards, psychologists, other behavioral scientists, and health care providers need to be conscientious when using actigraphy and clearly report scoring rules and variables. As my colleagues and I have discussed extensively, scoring rules should be established a priori, with a research or consultation group available to evaluate ambiguous nights (Acebo & LeBourgeois, 2006; Acebo, Sadeh, Seifer, et al., 1999; Meltzer, Montgomery-Downs, Insana, et al., 2012; Wolfson, Carskadon, Acebo et al., 2003).

In Part Four, *Sleep Challenges, Problems, and Disorders*, authors Brandy Roane and Daniel Taylor, Robert Auger and Stephanie Crowley, Anna Ivanenko and Kymberly Larson, Kristen Archbold, and Patricia Caldwell and Karen Waters discuss some of the common sleep problems that children, adolescents, and their families confront including insomnia, delayed sleep phase disorder, nightmares and parasomnias, sleep apnea, and nocturnal enuresis. Many such sleep problems receive the attention of pediatricians and increasingly more psychologists, and there are also numerous books available for parents and professionals (e.g., Ferber, 2006; Ivanenko, 2008; Mindell, 2005; Owens & Mindell, 2003/2010). Many problems of sleep in young children are resolved before school attendance, but these difficulties sometimes come to the attention of child clinical and school psychologists. Sleep disorders with substantial prevalence in adulthood, such as obstructive sleep apnea, insomnia, and restless legs syndrome are also prevalent in children. This section reviews the diagnostic and treatment outcome literature and provides suggestions for behavioral interventions that can be implemented by psychologists and other clinicians.

Part Five focuses on the *Consequences of Insufficient Sleep*, with chapters by Erika Bagley and Mona El-Sheikh; Victoria Molfese, Kathleen Moritz Rudasill and Dennis Molfese; Louise O'Brien; Chantelle Hart, Nicola Hawley, Elizabeth Kuhl and Elissa Jelalian; and Shelley Hershner. These chapters discuss the preponderance of evidence that insufficient sleep, inconsistent sleep-wake schedules, sleep problems, and poor sleep hygiene habits are associated with health and behavior problems including internalizing and externalizing behaviors, emotion dysregulation, hyperactivity, inattention, school adjustment, and ineffective cognitive skills in children and adolescents. Shelly Hershner emphasizes that inadequate and erratic sleep in adolescents and emerging adults can have substantial consequences—that is, “sleepy driver” and “fall asleep at the wheel” driving accidents. Some locations have regulated when young drivers can be on the road and include education materials on sleep and the consequences of sleep deprivation in drivers education programs (e.g., *Curriculum Scope and Sequence Modules for Driver Education in Virginia*, 2001; *Driver Education Program*, Massachusetts Department of Transportation, 2010). In many ways, this section is only the tip of the iceberg, as, in addition to the areas covered by these authors, research has demonstrated that insufficient, inconsistent, and disordered sleep has negative consequences for cognitive development, substance use and abuse, work-related accidents, immune system functioning, and a range of other health, behavioral, and cognitive factors.

*Sleep Difficulties Associated with Developmental and Behavioral Risks* are examined in Part Six. Authors Daphne Koinis Mitchell and Robin Everhart; Amanda Richdale, Penny Corkum and J. Aimee Coulombe; Alison Harvey, Candice Alfano, and Greg Clarke; and Richard Bootzin, Jennifer Cousins, Monica Kelly, and Sally Stevens examine children, adolescents, and emerging adults with asthma and allergies, developmental disabilities, attention deficit hyperactivity disorder, affective disorders, and struggles with substance abuse. A prevalence of comorbid sleep problems is not uncommon for children and adolescents with behavioral and emotional disorders (e.g., Quine, 2001; Redline, Tishler, Schluchter, et al., 1999; Snell, Adam, & Duncan, 2007; Stores & Wiggs, 2001; Wolfson & Armitage, 2009). Of course, psychologists regularly work with children with these difficulties, but they may be less familiar with the associated sleep problems and behavioral consequences. In some

situations, intervening and treating the sleep problems might mitigate the presenting and often challenging behavioral difficulties.

*The Oxford Handbook of Infant, Child, and Adolescent Sleep and Behavior* concludes with, perhaps, the most important area, Part Seven: *Prevention and Intervention*. These final five chapters (authors Melissa Moore and Jodi Mindell; Rhoda Au, Erica Appleman and Karina Stavitsky; Reut Gruber, Evelyn Constantin, Jamie Cassoff, and Sonia Michaelson; Pamela Thatcher; and S. Justin Thomas, Kristin Avis, and Kenneth Lichstein) focus on interventions and preventive approaches for families with young children; systemic countermeasures such as delaying school start times; prevention strategies and social learning-based programs for elementary, middle, and high school students; and approaches for working with college students and emerging adults struggling with disordered sleep. For example, my research team's Young Adolescent Sleep-Smart Pacesetter Program (funded by the National Institute of Child Health and Human Development) is evaluating the efficacy of a social learning-based, preventive intervention program designed to help early adolescents develop healthy sleep hygiene practices including decreasing caffeine use, obtaining adequate sleep, and maintaining consistent sleep schedules. Preliminary results suggest that the school-based *Sleep-Smart* program improved early adolescents' sleep patterns, hygiene practices, and sleep competence (Johnson, Harkins, Marco, Ludden, & Wolfson, 2012). Sleep-Smart participants also evidenced fewer health and behavioral difficulties and better grades following the program, whereas their comparison peers' behaviors remained the same during 7th grade.

The authors of this handbook have committed their academic and/or clinical careers to understanding the development of sleep patterns and the potential hurdles that prevent infants, children, and adolescents—along with their families—from obtaining sufficient and regular sleep. I am certain that they would agree with me and my coeditor, Hawley Montgomery-Downs, that high-quality, sound, and restorative sleep can improve our daily experiences, competence, and overall physical and emotional health. It is our hope that the research, guidance, and future directions discussed in each of the chapters in the *The Oxford Handbook of Infant, Child, and Adolescent Sleep and Behavior* will help psychologists and other behavioral scientists to understand and continue to study sleep and circadian rhythms in the context of psychological

development and children's health, and in the prevention and treatment of sleep and behavioral disorders. As I recall, at about age 9, my son exclaimed one night: "Just because you study sleep, doesn't mean that I have to have a bedtime!" In looking back, I think he really meant to say: "Duh... you do not have to be a psychologist to know that a 9-year-old like me needs to get a good night's sleep!"

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PART **1**

Sleep and Development

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# Overview of the History of Child and Adolescent Sleep Medicine

Stephen H. Sheldon

## Abstract

Development of pediatric and adolescent sleep medicine parallels the development of pediatric health care in the United States. Historical development of health care for children, as well as development of sleep medicine as a necessary and important medical discipline for adults, provides insight into the current position of pediatric and adolescent sleep medicine and future directions for clinical practice and research. An understanding of the evolution of sleep medicine into a clinical and research discipline will create important perspective. Juxtaposition of disciplines will sensitize the reader to the need for state-of-the-art evaluation of sleep and its pathologies seen in infants, children, and adolescents.

**Key Words:** pediatric sleep medicine, adolescent sleep medicine, childhood and adolescent sleep disorders, history of pediatric sleep medicine

## Introduction

The development of pediatric and adolescent sleep medicine parallels the development of pediatric health care in the United States. Historical development of health care for children, as well as development of sleep medicine for adults as a necessary and important medical discipline, provides insight into the current position of pediatric and adolescent sleep medicine and future directions for clinical practice and research.

### Development of Pediatrics as a Unique Discipline

Prior to the beginning of the twentieth century, health care for children and adolescents was virtually nonexistent. Health care for children was provided by the family. Mortality rates for infants were high. More than one-third of infants died before their fifth birthday (Holt, 1943). Despite this high incidence of infant mortality, little was done and few took particular notice. Health care for children

by the medical profession was provided using adult criteria, definition of disease/disorder, and therapeutic techniques (Cone, 1979). Medical practitioners who limited their practice to children were few and considered “baby feeders.” At the turn of the century it has been estimated that there were not more than fifty medical practitioners in the United States who were particularly interested in the health care of children, and less than a dozen limited their practice exclusively to children (Smith, 1951). Locations for clinical evaluation specifically designed for children were nonexistent (Cone, 1979). Being considered property of their parents, children were and typically remain neither a political nor economic force. Diseases were widespread. Prevention was the underlying principle. Approaches to treatment of illness during childhood included tea, barley water, and protein milk. Floating hospitals and country sanatoria were occasionally utilized for treatment of childhood illness, since sun, fresh air, and isolation were managements of choice. But, for the most part, care

of children remained in the home (Powers, 1939). Evaluation of childhood disease/disorders was based primarily on clinical signs and symptoms. Even congenital malformations were thought by many child health care practitioners to be due to maternal influences (Cone, 1979). Treatment was principally based on either adult medical interventions or was purely empiric. Climate therapy was common. Exposure to sunlight was prescribed for various illnesses including but not limited to tuberculosis, cutaneous abnormalities, anemia, and rickets. Some treatments were effective, but most were relatively ineffective. For example, treatment of pneumonia often included administration of digitalis, camphor, strychnia, and alcohol.

Child health care practitioners were thrust into the forefront of preventive medicine with the discovery and development of pasteurization of milk and immunizations for a variety of diseases. Antibiotics and the development of corticosteroids were instrumental in decreasing high childhood mortality rates existing during the first half of the twentieth century.

Subsequently, there had been rapid progress in pediatric medicine and surgery. Practice of pediatric medicine has turned from principally treatment of infectious diseases to comprehensive preventive programs, school health, community pediatrics, developmental pediatrics, and comprehensive adolescent medicine. Extensive morbidities have been identified, resulting in extensive efforts in behavioral disorders, family violence, child maltreatment, drug misuse, learning problems, school health, and developmental disabilities. Priorities have shifted, and identification of many pediatric disorders requires a multidisciplinary and interdisciplinary approach to diagnosis and management.

### **Development of Sleep Medicine as a Unique Discipline**

Although there has been a fascination with sleep since antiquity, the scientific investigation of sleep and its disorders can be traced back to 1930 when Berger first described spontaneous EEG activity in the brains of sleeping subjects (Berger, 1930); differentiation of sleep into specific and distinct states by Harvey, Loomis, and Hobart in 1937 (Harvey, Loomis, & Hobart, 1937); and the first description of rapid eye movement (REM) sleep by Aserensky and Kleitman at the University of Chicago in 1953 (Aserensky & Kleitman, 1953). In 1955, Aserensky and Kleitman observed sleeping infants and described a “rest–activity” cycle during sleep. These

periods were characterized by quiet periods, where there were no body or eye movements, and active periods of body movements and rapid eye movements under closed lids (Aserensky & Kleitman, 1955). Two years later, Dement and Kleitman reported the cycling of REM sleep and non-rapid eye movement (NREM) sleep throughout the sleep period, proposed a classification system of NREM sleep into four distinct stages, and hypothesized the association of eye movements in REM sleep with dream mentation (Dement & Kleitman, 1957a, 1957b).

It had become clear that these discoveries ushered in the realization that it was not enough to evaluate health and disease during only waking hours but throughout the 24-hour continuum. A new era of medical and scientific research emerged focusing on physiological, pharmacological, pathophysiological, and even anatomical that are different during sleep than during the waking state (Orem & Barnes, 1980). Sleep research provided the groundwork and basis for the realization that clinical evaluation and management of patients might differ during sleep when compared to wake, resulting in the emergence of clinical sleep medicine (Carskadon & Roth, 1989).

At first, clinical sleep medicine evolved from patient self-referrals. Most sleep complaints were related to problem insomnia. However, it became clear that the common belief that the majority of etiologies of insomnia were purely psychiatric in origin was false (Carskadon & Roth, 1989). Obstructive sleep apnea had been identified in Europe, but there had been little notice of the condition in the United States. In 1970, Lugaresi and colleagues published remarkable success of tracheostomy in the treatment of obstructive sleep apnea (Lugaresi, Coccagna, Mantovani, & Brignani, 1970). Nonetheless, similar evaluation and management of obstructive sleep apnea was not yet accepted. In 1972, Guilleminault demonstrated remarkable results in managing uncontrollable hypertension in a 10<sup>1/2</sup>-year-old boy with tracheostomy (Dement, 1994). *It is stunning that the demonstration of the first successful treatment of sequelae of obstructive sleep apnea in the United States was in a child.*

Physiological evaluation of sleep had also progressed with adaptation of polygraphy, used in monitoring EEG, to evaluate other physiological variables during sleep. Termed *polysomnography*, its use by Holland (Holland, Dement, & Raynal, 1974) changed the face of clinical assessment of sleep in adult patients. Now there were methods for both basic evaluation by history and physical

examination, as well as physiological assessment of sleep-related complaints in a clinical laboratory setting.

By the end of the 1970s, clinical sleep disorders medicine became an accepted area of medical inquiry, although practice of sleep disorders medicine was still couched in other disciplines of pulmonology, psychiatry, neurology, and internal medicine. In 1968, the *Manual of Standardized Terminology, Techniques, and Scoring System for Sleep Stages of Human Subjects* was published (Rechtschaffen & Kales, 1968). This was a significant step forward in standardizing sleep stage scoring in adults and eliminating unreliability and inconsistencies in laboratory evaluation of sleep, both between laboratories and within laboratories. It was clear at that time that this standardization was not appropriate for identification of stages of sleep and evaluation of sleep in newborns, infants, and children. Anatomical and physiological variables differed markedly from those of the adult. Similar standardization of sleep stage identification was a daunting task due to the rapid and constantly changing biology of the maturing and developing child. Therefore, the newborn infant became a starting point for a similar process that was started by Drs. Rechtschaffen and Kales in 1968. Drs. Thomas Anders, Robert Emde, and Arthur Parmelee were pioneers in bringing the science of sleep in infants and children to child health care practitioners. Because of their seminal work related to sleep in infants and children, Drs. Anders, Emde, and Parmelee co-chaired an ad hoc committee to provide similar standards, and the result was the publication in 1971 of *A Manual for Standardized Techniques and Criteria for Scoring of States of Sleep and Wakefulness in Newborn Infants* (Anders, Emde, & Parmelee, 1971). Strikingly, since publication of this manual, 42 years later there has been no similar effort for infants older than 2 months of age and children older than the beginning of puberty. Many problems precluded this task. Standardization in the pediatric age group is a formidable endeavor. First, there are rapid and dynamic changes that occur during the first two decades of life. The nervous system is constantly changing, structurally and functionally, during this period of life. Attempting to define cross-sectional criteria for evaluation of children both within same-age subjects and between subjects is extraordinarily difficult because of normal internal and external variability. Normal ranges can be extensive. Limitations include number of evaluations required for appropriate power. External reliability and validity can also be quite difficult

to establish. Several longitudinal points are often required for appropriate comparison of polysomnographic variables. This has been suggested to be termed *developmental polysomnography* (Sheldon, 1996). This would then take into account normal progression of maturation, rather than evaluating a single polygraphic study at a single point in time. Because of these immense difficulties, little evidence-based standardized information has been available to provide accurate and reproducible normative data, despite evidence that sleep and its normal structure and maturation has far-reaching implications for growth, development, and learning (Karni, Tanne, Rubenstein, Askenasy, & Sagi, 1994; Wilson & McNaughton, 1994).

Identification of effective noninvasive treatments for many sleep-related disorders developed (for example, treatment of obstructive sleep apnea in adults with nasal CPAP), resulting in rapid development of therapeutic protocols and widespread use. The combination of high prevalence of obstructive sleep apnea in the adult population, management of the obstructive sleep-disordered breathing with nasal continuous positive airway pressure (CPAP), a relatively innocuous procedure, and effective management of sequelae led to the rapid expansion of sleep medicine into a unique medical discipline. Sleep disorders medicine has become an accepted and distinct specialty within the medical community.

Beginning in 1978, the American Board of Sleep Medicine (ABSM) provided an examination in clinical polysomnography to assure quality of practitioners practicing sleep disorders medicine and interpreting polysomnograms. The first examination certified 21 candidates. During the next 28 years, the ABSM certified more than 3400 individuals (Quan, Berry, Buysse, Collop, Grigg-Damberger, Harding et al., 2008). This examination was not specialty-specific and was taken by internists, psychiatrists, psychologists, neurologists, family practitioners, and pediatricians. Successful applicants became diplomats of the ABSM. Indeed, sleep disorders medicine as a new and unique discipline became the focus of more clinical practitioners.

Pediatric and adolescent sleep medicine has become an outgrowth of this sleep disorders medicine practice. Inspiration has come from several directions: scientific and clinical interest in sudden infant death syndrome (SIDS); identification of obstructive sleep apnea and other sleep-related breathing disorders occurring with significant prevalence in the pediatric population; identification of the importance of sleep in the origin of daytime

behavioral difficulties; and the influence of sleep disorders and insufficient sleep on children's daytime performance and learning.

In the early 1980s the practice of pediatrics was a highly respected medical discipline. One of the principal textbooks utilized by most students and practitioners of health care for children was entitled *Nelson's Textbook of Pediatrics* (Behrman, 1992). Nevertheless, the 14th edition of this text published in 1992 had a total of only *eleven paragraphs* uniquely devoted to sleep disorders in children.

Between 1978 and 1987, several seminal works were published. Publication of three books for parents, entitled *Solve Your Child's Sleep Problems* (Ferber, 1985), *Helping Your Child Sleep Through the Night* (Cuthbertson & Schevill, 1985), and *Healthy Sleep Habits, Happy Child* (Weissbluth, 1987), focused on different techniques parents might employ to affect their child's sleep. Interestingly, these books tended to employ different approaches, creating considerable controversy in the child health care community and parental confusion. A compilation of scientific articles was published in 1978 and was entitled *Sleep and Its Disorders in Children*, edited by Dr. Christian Guilleminault (Guilleminault, 1987). This book gathered groundbreaking scientific papers on normative data providing a basis for future direction in the scientific study of sleep and sleep-wake cycles during infancy, childhood, and adolescence.

More changes occur in anatomy, physiology, and sleep-wake patterns during the first 15 years of life than over the next four decades. Nonetheless, while the work in this volume attests to the high-quality empirical work conducted, comparatively little information has been published regarding this transformation. Prevalence and impact of dysfunctional sleep on the developing child requires large population-based studies. It is imperative to determine how sleep and its organization develop in infancy and early childhood, since disruption of normal progression of development during these vastly important stages in human maturation may have lifelong consequences.

Clinical pediatric sleep medicine has had to rely on nosology developed for adults (Thorpy, 1990). Adaptations have been attempted (Sheldon, Spire, & Levy, 1992), but it is clearly apparent that adapting adult criteria to infants and children can lead to many false starts and wrong turns. Most sleep-related problems in children might carry similar nomenclature, but children and adolescents are clearly different. It would be inappropriate, improper, and incongruous to apply adult sleep

medicine anatomical, physiological, and pathological criteria to children.

Yet, the general pediatric community has been very slow to grasp the significance of the entirety of pediatric sleep disorders. Child health care practitioners have been resistant to absorb the importance of sleep physiology and sleep structure to human development and behavior. However, over the past 5 to 10 years, pediatric pulmonologists, otolaryngologists, neurologists, psychiatrists, and psychologists have increasingly recognized the importance of sleep and its disorders and have incorporated this large portion of the child's life into clinical and academic endeavors, with particular focus on sleep-related breathing abnormalities. With an apparent "epidemic" of obstructive sleep apnea in the pediatric population, this again seems to be an outgrowth of adult sleep medicine. To this end, child health care professionals began in 2005 to meet to discuss priorities for research, patient care, policy, and education. This first conference was sponsored by Brown University, Alpert Medical School, and was attended by more than 100 participants. There has been continued and ongoing excitement about this meeting, along with continued and rapid growth. In 2010, the first international conference was held in Rome, Italy, with more than 400 attendees. These conferences are now held biannually in alternate years.

In 2002, the American Academy of Sleep Medicine (AASM) applied to the Accreditation Council on Graduate Medical Education (ACGME) for establishment of sleep medicine training programs under the auspices of the ACGME as part of a comprehensive plan, along with the American Board of Medical Specialists, to accept sleep medicine as an independent medical specialty. In 2003 this was approved, and a consensus plan was developed for establishment of a new multidisciplinary specialty examination in sleep medicine to be jointly offered by the American Board of Internal Medicine, American Board of Psychiatry and Neurology, American Board of Pediatrics, American Board of Family Medicine, and the American Board of Otolaryngology, Head and Neck Surgery (Quan et al., 2008). The first examination was administered in 2007. Considerations and disorders unique to childhood comprised 2% of the first examination. Although pediatrics is a required portion of a sleep medicine fellowship curriculum, it is unclear how much pediatric medicine and sleep disorders in children is afforded to internists, otolaryngologists, psychiatrists, and neurologists studying general

sleep medicine in these programs. It is also unclear whether training in developmental medicine and children's health care can be translated into practice of sleep medicine without a comprehensive underpinning of pediatric medicine.

Success in incorporating pediatric sleep medicine objectives into undergraduate, graduate, and postgraduate training curricula will depend upon outcome and cost-effectiveness. Integrating disciplines (including but not limited to medicine and psychology) is crucial in development of successful and effective sleep medicine services for children. Interdisciplinary and multidisciplinary approaches to diagnosis and management of sleep-related disorders in infancy, childhood, and adolescence requires this model. Many questions remain. First, can the provision of comprehensive sleep medicine services to children by pediatricians specializing and devoting full time to the practice of pediatric sleep medicine have a significant impact on comorbid medical illnesses such as sickle cell anemia, cystic fibrosis, neuromuscular disorders, craniofacial malformations, or congenital/acquired cardiovascular disease? Second, what effect does early disruption of sleep and/or sleep-wake cycling have on learning, memory, and cognitive development? Finally, can understanding sleep and its disorders in childhood contribute to a better understanding of behavioral disorders, problems of attention, and learning disabilities? The work described in this book illustrates the exciting, ongoing work that is being done. The authors of these chapters also highlight the most important future directions in each of their areas. The mystery of establishing and integrating neural networks required for early brain development and later executive functioning may be locked within the sleeping brain.

As was true of the development of pediatrics as a unique medical discipline, further appreciation of the development of sleep and its structure, as well as the effects of disruption of its normal maturation, might lead to improved diagnosis, treatment, and prevention of a wide variety of disorders unique to both children and adults. It is evident that the present is only the beginning of the understanding of pediatric sleep and pediatric sleep medicine.

## Summary

Pediatric and adolescent sleep medicine has followed a similar path in its maturation to the development of pediatrics as a recognized and unique medical discipline. There has been a very significant increase in evidence-based knowledge regarding

sleep and its disorders in infants, children, and adolescents over the past decade. Nonetheless, what is known now about the importance of sleep in normal human development and sleep in health and disease is likely only the "tip of the iceberg." The future of pediatric and Adolescent Sleep Medicine is truly before us. Many questions remain:

1. How important is the basic rest-activity cycle during gestation in growth and maturation of the central nervous system, neuronal migration, and neural network development?
2. What impact does disruption of normal sleep and/or its continuity during the first few years of life have on future human development and performance?
3. What effect does sleep deprivation during adolescence have on health and well-being as an adult? How might this contribute to chronic illness affecting these individuals as adults?

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# A Brief History of Child and Adolescent Sleep Research: Key Contributions in Psychology

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## Abstract

The scientific understanding of sleep in infants, children, and adolescents has expanded significantly over the past few decades. Within psychology, key discoveries have been made in several important areas including (1) the understanding of prevalence and impact of childhood sleep problems and disorders; (2) the development and evaluation of behavioral treatments for childhood sleep problems; (3) the development of conceptual models of children's sleep and sleep dysfunction; (4) the measurement of sleep patterns, behaviors, and disorders in children; and (5) the identification of sleep-related concerns in pediatric medical, neurodevelopmental, and psychiatric populations. Sleep is now recognized as a cross-cutting issue in child and pediatric psychology. Expanding opportunities within psychology for involvement in pediatric sleep research and sleep clinical training are part of this evolving history. A research agenda building from these key discoveries will move the field of pediatric sleep medicine forward over the next several decades.

**Key Words:** history, psychology, pediatric, sleep research

## Introduction

Within the field of psychology, important contributions have been made to the scientific understanding of sleep in infants, children, and adolescents. Over the past three decades, scholarly work in psychology has expanded the evidence base of pediatric sleep medicine, led to practice changes in treatment of childhood sleep problems, produced policy changes (e.g., school start times), and led to increased emphasis on training in behavioral sleep medicine (e.g., certification in Behavioral Sleep Medicine, postdoctoral training). This has all been accomplished in a relatively short time, as the focus on infants, children, and adolescents within sleep research is recent.

The aim of this chapter is to highlight key areas of research contribution in psychology to the understanding of children's sleep. Specifically, five areas of key discoveries in child sleep research are reviewed, including: (1) prevalence and impact of sleep

problems and disorders; (2) evaluation of behavioral treatments for pediatric sleep problems; (3) conceptual models of children's sleep and sleep dysfunction; (4) measurement of sleep behaviors, sleep patterns, and sleep disorders in children; and (5) sleep in special populations of children with medical, neurodevelopmental, and psychiatric conditions. Each of these areas is considered from a historical perspective in this chapter, highlighting key findings that moved the field forward. This chapter is not intended to provide a comprehensive historical overview of sleep research in psychology; rather, it is meant to draw attention to key discoveries and provide examples to demonstrate the progression of research.

## Prevalence and Impact of Sleep Problems and Disorders

Three decades ago, relatively little was known about normative patterns and variation in children's

sleep, and most advice about proper sleep patterns was based on personal observation and opinion. Thus, an important focus of psychology research aimed to describe infant, child, and adolescent sleep patterns and to identify the frequency of problematic sleep patterns and behaviors in childhood in order to establish scientific data on this topic. A number of cross-sectional survey studies emerged to fill in this gap in knowledge. Developmental changes in patterns of sleep including average bedtimes, wake times, and sleep duration of children across the pediatric age span were described (e.g., Anders & Keener, 1985; Carskadon, 1990; Levy, Gray-Donald, Leech, Zvagulis, & Pless, 1986; Sadeh, Lavie, Scher, Tirosh, & Epstein, 1991). Moreover, studies were conducted to identify parental concerns about problematic child sleep behaviors (e.g., Blader, Koplewicz, Abikoff, & Foley, 1997; Johnson, 1991; Richman, 1981). Screening measures for sleep disorders were developed in community samples, identifying the prevalence of a range of behaviorally based and physiologically based sleep disorders to understand potential treatment needs. Progress was made in the investigation of behaviorally based sleep disorders; for example, prevalence data emerged showing that 12% to 16% of adolescents have clinically significant insomnia (Morrison, McGee, & Stanton, 1992; Ohayon, Caulet, & Lemoine, 1998; Roberts, Lee, Hernandez, & Solari, 2004). Researchers also recognized the importance of studying the course of sleep problems longitudinally, finding, for example, that a significant portion of children who experience sleep disturbances at earlier ages continue to do so over time (e.g., Kataria, Swanson, & Trevathan, 1987; Pollock, 1992) and that sleep disturbances may be chronic or persistent (e.g., Roberts, Roberts, & Duong, 2008). Together, data on the prevalence and course of sleep behaviors and sleep disorders were compelling in demonstrating that childhood sleep problems were an important pediatric health problem worthy of study.

Beyond prevalence and epidemiology, the impact of sleep problems became an important research topic in psychology. There was interest in understanding how sleep problems may affect children and families in a variety of domains. In infants and young children, researchers asked questions about the impact of the child's problematic sleep on adult caregivers in regard to their own sleep, mood and affect, and parenting stress (e.g., Hiscock & Wake, 2002). It was recognized that the child's sleep could impact the entire family. This followed from similar theoretical movements in the fields of pediatric

and child psychology, where family systems theories were developing in the conceptualization of child illness (e.g., Kazak, 1989). The study of childhood sleep in the context of the family has continued to date; in particular, research has focused on the complexity for sleep in the family when a child has a chronic illness or developmental disorder (Meltzer & Montgomery-Downs, 2011).

Efforts were also directed toward describing the functional impact of disrupted sleep on children. Research in psychology showed clearly that children were affected by sleep loss and sleep disruption in a multitude of domains. For example, key findings emerged demonstrating that children and adolescents who experience excessive daytime sleepiness due to insufficient or interrupted sleep are at greater risk for mood disturbances, behavioral disruption, impaired cognitive functioning, reduced academic performance, attentional difficulties, and increased school absences (e.g., Fallone, Owens, & Deane, 2002; Gozal, 1998; Wolfson & Carskadon, 1998). Poor sleep was also linked with socioemotional problems, particularly symptoms of anxiety and depression, behavior problems, and substance abuse in youth (Johnson & Breslau, 2001; Smedje, Broman, & Hetta, 2001).

In child neuropsychology research, these functional indicators were further explored. Comprehensive studies were undertaken to examine neurocognitive function and sleep in children, finding that disturbed sleep is associated with problems with cognitive functioning, learning, and attention (Sadeh, Gruber, & Raviv, 2002, 2003). Neurocognitive function was studied in children with known sleep disorders such as sleep disordered breathing (SDB), as well as in children who are otherwise healthy. These data extended the study of consequences that had been defined in the adult literature related to SDB where neurocognitive deficits, which may be a consequence of sleep fragmentation or hypoxemia, had been reported. For example, investigations within neuropsychology of children with SDB showed similar deficits (as in adults) in neurocognitive performance, behavioral impairments, and reduced school performance (e.g., Blunden, Lushington, & Kennedy, 2001; Gozal, 1998; Lewin, Rosen, England, & Dahl, 2002; Owens, Spirito, McGuinn, & Nobile, 2000). Further research attempted to describe whether these deficits may be completely or partially reversible with treatment of SDB (Gozal, 1998; Lewin et al., 2002; Owens, Spirito, McGuinn et al., 2000). Major research discoveries have continued over

the last decade providing new data on the consequences of SDB on children. This continues to be an important topic of study. For example, several recent investigations have examined whether behavioral and neurocognitive functions of children with obstructive sleep apnea syndrome (OSAS) improve after adenotonsillectomy (e.g., Landau et al., 2012; Redline et al., 2011). Findings to date suggest that the impact of OSAS on behavioral and cognitive functions begins in early childhood.

Contemporary research in neuropsychology is focusing on sleep restriction and neurocognitive function in otherwise healthy children (e.g., Beebe, Difrancesco, Tlustos, McNally, & Holland, 2009), using brain imaging methods. This innovative work continues to increase understanding of the broad impact of sleep disruption on children. Part Five of this book provides a contemporary focus on consequences of insufficient sleep.

In addition to neurocognitive effects, elegant work has emerged on the impact of OSAS on children's health care use (Tarasiuk et al., 2007). In this study, findings demonstrated that children with OSAS have higher health care use starting from the first year of life, and the majority of the health service use was due to higher occurrence of respiratory tract morbidity (Tarasiuk et al., 2007). Further research is needed to demonstrate the potential health economic impact of other sleep disturbances in infants, children, and adolescents.

Research discoveries in psychology in the area of prevalence and impact of childhood sleep problems have also been translated into public policy changes. One of the best exemplars of this is the topic of sleep in adolescents. In the 1980s, extending descriptive research on sleep to adolescents, Carskadon conducted pioneering studies that contributed knowledge of normal sleep-related changes in adolescence, identifying the optimal sleep needs of adolescents and serving to highlight the significant deficit in sleep obtained by most youth (Carskadon et al., 1980; see Carskadon and Tarokh, Chapter 8). This line of research on sleep regulation in adolescents has now spanned several decades and has led to increased understanding of sleep problems and impact on behavior in adolescents (e.g., Carskadon, Acebo, & Jenni, 2004; Carskadon, Wolfson, Acebo, Tzischinsky, & Seifer, 1998). These findings fueled a tremendous interest in the study of sleep in adolescents as well as advocacy efforts, particularly around school start times and consideration of how changes in policy may help to prevent sleep problems (see Au, Appleman, and Stavitsky, Chapter 38).

An influential study was published in 1998 by Wolfson, Carskadon, and colleagues specifically on the relationship between inadequate sleep quantity and quality and adolescent school performance. Using high school records of academic performance, this study clearly linked inadequate sleep to problems with attendance and reduced academic performance (Wolfson & Carskadon, 1998). This work became critical in a national debate about school start times. Over the subsequent years, a number of school districts have responded to these research findings regarding the prevalence of inadequate sleep among high school students with a policy change of delaying school start times (e.g., from starting at 7:30 a.m. to starting at 8:30 a.m.) to allow adolescents to sleep longer in the morning. Many school districts continue to debate the issue, and psychology research is active in extending these data to the study of sleep in middle school students (Wolfson, Spaulding, Dandrow, & Baroni, 2007).

The conclusions from this body of work in psychology are that sleep problems are common across the pediatric age span and, importantly, that they are also associated with major sources of child and family functional impact, highlighting the significance of directing efforts toward prevention and treatment of sleep problems in children.

### **Evaluating Behavioral Treatments for Sleep Problems in Children**

In the 1980s and 1990s, pediatric sleep medicine was emerging as a legitimate clinical specialty. Psychologists began consulting with and collaborating alongside sleep medicine physicians to provide behavioral treatment to children with a range of sleep disorders. A number of books for health professionals on children's sleep emerged during this time (e.g., Ferber & Kryger, 1995; Guilleminault, 1987), focused broadly on describing sleep disorders in children and adolescents. Moreover, key books about children's sleep were published during this time specifically for a parent audience (e.g., Cuthbertson & Schevill, 1985; Ferber, 1986; Mindell, 1997; Weissbluth, 1987). Importantly, behavioral treatment strategies were introduced in these books, such as the application of behavioral techniques to treat bedtime resistance in young children. Clearly moving the field forward, many of the treatment and intervention strategies presented in these early professional and lay books continue to be used today.

Given that the most frequent childhood sleep problems involve bedtime resistance and night

wakings in infants and young children (Meltzer & Mindell, 2006), the thrust of the treatment research in psychology has been in this area. Published reports of behavioral treatments for pediatric sleep problems emerged in the 1980s and 1990s with mostly case series and small treatment studies (e.g., France & Hudson, 1990; Rapoff, Christophersen, & Rapoff, 1982). Important review articles were written by psychologists describing treatment of sleep disorders in children (e.g., Mindell, 1993) and summarizing priorities for research. For example, Mindell (1993) highlighted a number of methodological problems in studies on sleep disorders in children including the large number of case studies and uncontrolled trials, the lack of long-term follow-up, subjectivity of the outcome data on sleep, and large gaps in research. Since Mindell's review was published, it has been exciting to see research in psychology address many of these research gaps and limitations, due to the concerted efforts to study sleep interventions in controlled trials in children and to improve on the measurement of sleep and related outcomes.

A solid body of literature now exists supporting the use of evidence-based behavioral management strategies to treat bedtime problems and night wakings in infants, toddlers, and preschoolers. An influential program of research on behavioral interventions is the work conducted by Mindell and colleagues, including randomized controlled trials of behavioral interventions for bedtime settling and night waking problems, as well as systematic reviews synthesizing this treatment literature (Mindell, 1999; Mindell, Kuhn, Lewin, Meltzer, & Sadeh, 2006). A recent systematic review was used in practice parameters developed by the American Academy of Sleep Medicine (AASM) presenting recommendations for the use of behavioral treatments for bedtime problems and night wakings in young children (Morgenthaler et al., 2006) based on this scientific evidence. Several specific behavioral treatments had enough evidence to be considered "standards" for treatment of bedtime problems and night wakings. These specific treatments include unmodified extinction, extinction with parental presence (see Blampied, Chapter 15), and preventive parent education. These were all rated as individually effective therapies in the treatment of bedtime problems and night wakings, producing reliable and significant clinical improvement in children's sleep.

Treatment directed toward the sleep problems of older children and adolescents has been relatively slow to develop in contrast to the tremendous

surge of treatment research conducted over the past two decades in younger children. However, there are recent examples within psychology of research focused on the development and evaluation of interventions with older children and adolescents with insomnia (Bootzin & Stevens, 2005; Paine & Gradisar, 2011) and delayed sleep phase (Gradisar et al., 2011), and this is an area ripe for future investigation. Part Seven of this book highlights contemporary work on prevention and intervention of childhood sleep problems.

## **Conceptual Models of Sleep and Sleep Dysfunction**

Within psychology, a number of conceptual models of childhood sleep and sleep dysfunction have been put forth over the years. Many of these models are intended to establish links between child and family risk of perpetuating factors for sleep problems, and consequences of sleep dysfunction, with the intent of identifying areas to intervene. Similar to conceptual models proposed in the child psychology literature more broadly (e.g., biopsychosocial models) to describe factors that influence children's adjustment to medical conditions, most models of childhood sleep are integrated in developmental models of sleep within the larger context of the child's biology, psychological factors, and social systems.

For example, one of the early models proposed by Sadeh and Anders (1993) was a transactional model of infant sleep which identified multiple etiological sources for infant sleep problems from a systems perspective. This model linked etiology to methods of assessment of sleep disturbances and application of specific intervention methods for the specific system involved. Similarly, in understanding adolescent sleep, Carskadon (2011) has described the convergence of biologic, psychological, and sociocultural influences on sleep. Other models have expanded understanding of family and culture to incorporate that children's sleep is shaped and interpreted by cultural values and beliefs (e.g., Boergers & Koinis-Mitchell, 2010).

Within childhood sleep research, conceptual models provide useful organizing frameworks helping to shape and focus research efforts in psychology. One example of how a conceptual model influenced subsequent research in psychology is in the area of children's pain and sleep. In 1999, Lewin and Dahl published a theoretical paper describing a model to explain the links between the regulation of sleep and pediatric pain. The primary tenet of the framework is that there are bidirectional effects between pain and

sleep. Pain can directly affect sleep by prolonging sleep onset and interfering with the depth and continuity of sleep states, and the psychological and physiological sequelae of insufficient sleep (e.g., worry, negative thoughts, and decrements in behavioral control) may have deleterious effects on pain management. After the publication of this model, descriptions of sleep in children and adolescents with chronic pain became a topic of growing interest in psychology and pediatrics (e.g., Bruni, Russo, Violani, & Guidetti, 2004; Bruusgaard, Smedbraten, & Natvig, 2000; Haim et al., 2004; Palermo & Kiska, 2005). Researchers began to examine both directions of the pain–sleep relationship and focused attention on describing the impact of sleep disturbances on children’s functional outcomes and health-related quality of life (LaPlant, Adams, Haftel, & Chervin, 2007).

Conceptual and theoretical models of childhood sleep are a major contribution within psychology, guiding subsequent research efforts. The continued focus on sleep within the child’s broader systems of influence will remain a critical guide to further intervention development. Current research priorities on factors influencing sleep are described in Part Two of this book.

### **Measurement of Sleep Behaviors, Sleep Patterns, and Sleep Disorders**

Because sleep is a multidimensional construct, measurement approaches have also been multimodal. In psychology, many contributions have been made over the past few decades to the measurement of sleep patterns, behaviors, and disorders in children. Research to describe patterns of children’s sleep has encompassed both self-report of sleep and the measurement of activity patterns to derive sleep times using actigraphy (see Horne and Biggs, Chapter 16). For example, in the 1980s and 1990s, in validating actigraphy in child populations, the important work of Acebo, Sadeh, and colleagues (e.g., Carskadon, Acebo, Richardson, Tate, & Seifer, 1997; Sadeh & Acebo, 2002; Sadeh, Hauri, Kripke, & Lavie, 1995; Sadeh et al., 1991) supported this measurement technique as a viable option in child sleep research. Early publications focused on actigraphy procedures, scoring, and validity, guiding the approach to the use of actigraphy in child sleep research. Actigraphy validation and scoring in children remains an important contemporary area of scientific inquiry (e.g., Meltzer, Montgomery-Downs, Insana, & Walsh, 2012).

During the last 20 years, the number of pediatric subjective sleep measures (see Spruyt, Chapter 18)

used in psychology research has increased significantly. Many specific measures have been developed to provide developmentally appropriate assessment of sleep across the entire pediatric age span from infancy to adolescence. There are now many available well-validated subjective measures to describe sleep habits, sleep quality, sleep beliefs, daytime sleepiness, and sleep disorders in children and adolescents; for example, see review by Lewandowski et al. (2011). Many of these instruments were developed with the contributions of psychologists.

One of the most widely used multidimensional measures of children’s sleep is the *Children’s Sleep Habits Questionnaire* (Owens, Spirito, & McGuinn, 2000). The publication of this measure was important in child sleep research, as it provided a tool to screen broad pediatric populations for sleep dysfunction and sleep disorders. It was widely available and quickly translated into many languages. More recent research on sleep measures has also involved validating subjective questionnaires against other forms of sleep assessment. For example, validation studies for both the *Brief Infant Sleep Questionnaire* (Sadeh, 2004) and the *Sleep Habits Survey* have demonstrated associations with actigraphy (Wolfson et al., 2003). Similarly, the sleep-related breathing disorder subscale of both the *Pediatric Sleep Questionnaire* (Chervin, Hedger, Dillon, & Pituch, 2000) and the *Sleep Disturbance Scale for Children* (Ferreira et al., 2009) have been validated with polysomnography. Important progress in measurement has greatly expanded available options in the study of children’s sleep.

Accurate assessment of sleep disturbance and associated behaviors has practical applications in research and clinical care. As one example, the Ped-IMPACT consensus group, charged with developing recommendations for relevant outcome domains in clinical trials for pediatric pain management, identified sleep as a core outcome domain (McGrath et al., 2008) in therapeutic trials for youth with chronic pain, recommending several validated subjective measures of sleep for outcome assessment.

### **Sleep in Special Populations: Children with Medical, Neurodevelopmental, and Psychiatric Conditions**

The study of sleep in special populations of children with medical, neurodevelopmental, and psychiatric conditions emerged in the 1980s and 1990s as an important clinical topic in psychological or behavioral treatment of individuals with sleep problems and comorbidities. Published applications of behavioral strategies (e.g., positive routines,

graduated extinction, and scheduled wakings; see Blampied, Chapter 15) to treat sleep problems in special pediatric populations are a significant part of this early history (e.g., Bramble, 1996, 1997; Durand, Gerner-Dott, & Mapstone, 1996; Milan, Mitchell, Berger, & Pierson, 1981). Because children with special health needs represent a large number of visits to child and pediatric psychologists, the treatment of sleep problems represented an important clinical challenge. For example, the majority of adolescents presenting for treatment of insomnia in a sleep clinic have been found to have a diagnosis of a psychiatric disorder, often a mood disorder (Ivanenko, Barnes, Crabtree, & Gozal, 2004; see Harvey, Alfano, and Clarke, Chapter 35).

Case series of behavioral interventions (e.g., positive routines, faded bedtimes, response cost) demonstrated that interventions used in physically healthy populations could be modified to successfully treat bedtime resistance in children with neurodevelopmental disorders (see Richdale Chapter 33.). For example, Piazza conducted a series of intervention studies in the 1990s with children with developmental disabilities (Piazza & Fisher, 1991a, 1991b; Piazza, Fisher, & Moser, 1991; Piazza, Fisher, & Sherer, 1997); finding improvements from behavioral interventions in children's sleep behaviors. Later that decade, Durand (1997) published a book appropriate to both health professionals and parents on treatment of childhood sleep problems, with particular emphasis on children with neurodevelopmental disorders. This collection of treatment studies on children with special needs propelled a much broader research focus on sleep in children with autism (see Richdale, Chapter 33, this volume), attention deficit disorder (see Corkum and Coulombe, Chapter 34), and other developmental and psychiatric conditions. Part Six of this handbook is devoted to sleep difficulties associated with developmental and behavioral risks and showcases the significant ongoing work being done in this area.

The clinical observations made by psychologists spurred additional research on the prevalence and impact of sleep disturbances in children with developmental disorders, chronic medical conditions (see Meltzer and Walsh, Chapter 13), and psychiatric conditions. A robust body of literature developed over the past decade demonstrated that the presence of chronic medical or developmental conditions in children is associated with increased prevalence of sleep problems (e.g., Hysing, Sivertsen, Stormark, Elgen, & Lundervold, 2009; Long, Krishnamurthy, & Palermo, 2008; Sivertsen, Posserud, Gillberg, Lundervold, &

Hysing, 2012). Multiple factors including both acute and chronic pain, underlying disease processes, concurrent medications, the impact of hospitalization, and comorbid psychiatric conditions such as depression and anxiety have been identified as important to consider in assessing the bidirectional relationship of sleep problems and acute and chronic illness in children (Lewandowski, Ward, & Palermo, 2011). In recognition of the emerging focus on these populations within psychology, two special issues were published in the *Journal of Pediatric Psychology* in 2008 (Owens & Palermo, 2008; Palermo & Owens, 2008) on the topics of sleep in children with medical conditions and neurodevelopmental disorders. Current priorities in this area are dedicated to developing and evaluating behavioral interventions for special populations of children with sleep problems (e.g., Vriend, Corkum, Moon, & Smith, 2011).

My own interest in sleep was sparked by a desire to treat sleep problems in child medical populations. As a pediatric psychologist engaged in clinical services and research related to children with chronic pain, I had observed that sleep was an important issue interwoven with children's experiences with chronic pain and in their daily functioning. However, I stumbled upon sleep medicine in my early career in the late 1990s as a junior faculty member at Rainbow Babies and Children's Hospital, where I was asked to provide behavioral services within a children's sleep program. At that time, I was unaware of the emerging literature underlying the connection between sleep and health outcomes. I found the provision of behavioral sleep services to children and families to be highly satisfying and was drawn to merging sleep with my research interest in chronic pain. The theoretical model proposed by Lewin and Dahl (1999) was extremely influential in how I approached the study of pain and sleep in children. Thus, my work included description of sleep characteristics, patterns, and behaviors in youth with chronic pain (e.g., Long et al., 2008; Palermo & Kiska, 2005; Palermo, Toliver-Sokol, Fonareva, & Koh, 2007), and the bidirectional relationship between sleep, pain, and functional outcomes (e.g., Lewandowski, Palermo, De la Motte, & Fu, 2010; Palermo, Fonareva, & Janosy, 2008; Palermo, Wilson, Lewandowski, Toliver-Sokol, & Murray, 2011). My current research aims to develop and evaluate behavioral interventions for insomnia in youth with chronic pain. In my own career, I have straddled the fields of pediatric psychology, pain management, and sleep medicine; at the nexus is exactly where my passion lies.

## Conclusion

Sleep is now recognized as a cross-cutting issue in child and pediatric psychology, one that inextricably connects health, mental health, and infant, child, and adolescent development. As such, investigators with diverse backgrounds may work toward addressing similar research questions. Conclusions from the last few decades of research on childhood sleep in psychology are that sleep problems are not only common across the pediatric age span in healthy as well as medical and psychiatric populations, but that they are also associated with significant child and family functional impact. Research findings have reinforced the importance of directing efforts toward prevention and treatment of sleep problems across the pediatric age span.

From the work in psychology, a solid body of literature now exists supporting the use of behavioral interventions to treat bedtime problems and night wakings in infants, toddlers, and preschoolers. Conceptual and theoretical models have guided subsequent research efforts in these areas, and are an important contribution within psychology. Strides have been made in the measurement of multiple domains of sleep, sleep behavior, and sleep disorders in children, and valid methods are available for outcome assessment in sleep intervention research.

## Future Directions

Many topics in pediatric sleep research remain to be addressed within psychology. Based on the five areas of key research contribution covered in this chapter, the following questions, if solved, would provide important future direction for the field.

- What factors predict success of behavioral treatment for specific infants, children, adolescents, and families?
- Which sleep disturbances in childhood are associated with elevated health care utilization and costs?
- What behavioral treatments are effective for managing the sleep problems of older children and adolescents?
- How do we best measure insomnia symptoms in children and adolescents?
- Are existing sleep measures valid in culturally and racially diverse pediatric populations?
- Which interventions are effective for sleep problems in special populations of children with acute and chronic medical conditions and mental health and psychiatric disturbances?

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# Developmental Science in the Study of Sleep

Angela D. Staples and John E. Bates

## Abstract

Developmental research has typically focused on daytime behaviors. However, the burgeoning interest in the role of sleep in the development of cognitive, social, emotional, academic, and health outcomes (Beebe, 2006; Beebe & Gozal, 2002; Carskadon, Acebo, & Jenni, 2004; Ednick et al., 2009; 2009; El-Sheikh, 2011; Ohayon, Carskadon, Guilleminault, & Vitiello, 2004; Sadeh, Raviv, & Gruber, 2000) calls for a consideration of sleep as a developmental process that co-occurs, co-regulates, and is causally linked with other developmental processes. This chapter discusses what it means to consider sleep from a developmental perspective. We hope this chapter will be useful for both developmental researchers who want to consider sleep as a variable of interest and for sleep researchers who want to take a more developmental approach to understanding sleep as a behavior that undergoes dramatic change from infancy through adolescence. The chapter is organized around three questions. First, what does it mean to have a developmental perspective? Second, how does one approach the study of sleep from a developmental perspective? Finally, how do we incorporate the study of sleep into the study of other areas of development?

**Key Words:** developmental science, sleep, developmental systems

## Introduction

“Sleep, in its ubiquity, seeming nonsociality, apparent universality, and presumed biologically driven uniformity, has been overlooked [by anthropology] as a background variable” (Worthman & Melby, 2002, p. 69).

Although sleep is universal and certainly biologically driven to some extent, research on sleep and sleep-related behaviors has begun to define the ways in which sleep is social, is influenced by forces other than biology, and varies between people as well as across the lifespan. This chapter discusses what it means to consider sleep from a developmental perspective. We hope this chapter will be useful for both developmental researchers who want to consider sleep as a variable of interest and for sleep researchers who want to take a more developmental

approach to understanding sleep. The chapter is organized around three questions. First, what does it mean to have a developmental perspective? Second, how does one approach the study of sleep from a developmental perspective? Finally, how do we incorporate the study of sleep into the study of other areas of development?

## Developmental Perspective

“Major concerns of the developmental orientation are understanding when and how behavioral changes occur and the potential in development for optimal adaptations” (Cairns, Costello, & Elder, 2001, p. 228).

The central question that separates developmental research from other areas of research is the quest to understand how both change and continuity

occur. Broadly speaking, developmental science is concerned with understanding the process of human development over the course of the lifespan. How is it that an infant transitions from crawling to walking? How do infants' vowel and vowel consonant sounds play a role in the development of language? How do children develop skills for regulating their attention, emotion, and behavior? How do adolescents form their identities? It is not enough to merely list a sequence of milestones, for example to say that 6-month-olds do not walk and 12-month-olds walk. Developmental science strives to understand and quantify the conditions that promote or hinder qualitative and quantitative shifts in development across the lifespan. In other words, it seeks to understand *process* in both change and continuity.

Developmental science, like other areas of science, has become increasingly multidisciplinary in its approach to understanding the process of development. Today, researchers from the areas of developmental psychology, pediatrics, clinical and educational psychology, psychiatry, biology, neuroscience, sociology, and epidemiology work together on a wide variety of research topics. The broad, multidisciplinary field of developmental psychopathology (Cicchetti, 2006), which seeks to integrate multiple levels of organization from biology to social systems, as well as to consider both typical and atypical development, is a dominant example of how developmental science has been growing. Along with the increased emphasis on interdisciplinary research, theories of development are evolving to link changes that occur on time scales from the millisecond (e.g., neuronal activity), hourly (e.g., cellular activity), daily (e.g., cortisol fluctuation), yearly (e.g., mastering addition or calculus), or lifespan (e.g., identity, interpersonal relationships) with other facets of life such as family stress, health problems, school settings, and neighborhood quality.

To impose some structure on the vast diversity of developmental phenomena, we turn to the systems theories of Ford and Lerner (1992), Bronfenbrenner and Morris (2006), Fogel (1993), and Thelen and Smith (1994) among others. Each of these theorists provides a slightly different emphasis on how to approach development from a systems perspective. For example, Ford and Lerner emphasize the lifespan perspective and use systems terminology to target qualitative changes in development (Keller, 2005). In contrast, the systems approaches advocated by Thelen and Smith (1994) and colleagues (see Adolph & Robinson, 2008; Simmering & Spencer, 2008; Spencer, Perone, & Buss, 2011) as well as van

Geert and colleagues (Steenbeek, 2007; van Geert, 2011) emphasize rigorous mathematical quantification of change processes that are biologically plausible. Adding to the complexity, Bronfenbrenner urges consideration of the nested, interconnected systems in which development occurs through the framework of studying process, person, context, and time (Bronfenbrenner & Morris, 2006). Together, these systems perspectives challenge developmental researchers to consider continuity and change processes that occur within, between, and among individuals.

Further complexity, beyond multiple time scales and multiple, nested systems, comes from considering individual differences in how people respond to similar situations at similar points in development (Rothbart & Bates, 2006). To any given situation, different people bring different emotional or motivational tendencies, as well as different self-regulatory tendencies. These differences derive at least in part from early-appearing, biologically based, relatively stable traits that we call *temperament* or *basic personality* (Rothbart & Bates, 2006). However, individuals also change due a variety of factors including learning, biological development, and changes in environments (Patterson, Reid, & Dishion, 1992).

We think processes involving sleep can and should be considered in relation to the various components of the general systems models mentioned. The level at which one tackles various components of the system (e.g., neurological, behavioral, cultural) will be largely determined by the phenomena under study and the particular research question. For example, several studies have linked biological fluctuations in cortisol and napping in preschool children (Ward, Gay, Alkon, Anders, & Lee, 2008; Watamura, Donzella, Kertes, & Gunnar, 2004; Watamura, Kryzer, & Robertson, 2008). El-Sheikh and colleagues have extended the biological-sleep link to also include measures of physiological responding as well as family processes (El-Sheikh & Buckhalt, 2005; El-Sheikh & Erath, 2011; El-Sheikh, Buckhalt, Mize, & Acebo, 2006). There have even been studies of sleep in adulthood using computational models of the regulation of sleep-wake cycles, sleep deprivation and sustained attention, sleep and neurobehavioral performance, and the effects of fatigue on learning (Gonzalez, Best, Healy, & Kole, 2011; Gunzelmann, Gross, Gluck, & Dinges, 2009; Klerman & Hiltaire, 2007; Postnova, Voigt, & Braun, 2009). Although these studies share the commonality of considering multiple system levels, much more research is needed to

link multiple systems to intra- and inter-individual development.

### Studying Sleep Developmentally

To study sleep developmentally requires a shift in theoretical perspective, different methods of data collection, and the addition of statistical methods designed to detect and explain processes of change. Sleep is a complex developmental phenomenon (van Geert, 2011) that undergoes periods of rapid change (e.g., shifting the longest sleep period to occur at night in the first weeks of life) and periods of stability. Much of the work done on changes in sleep across the lifespan has been through comparing age-group averages on sleep measures. From this research a clear picture is emerging of normative changes in sleep with consolidation of sleep into a single nighttime period by age five, a reduction in the number, frequency, and duration of nighttime awakenings, and considerable stability in the average amounts of nighttime sleep from toddlerhood through childhood (Staples & Bates, 2011). For example, in a prospective longitudinal study beginning at age one and continuing annually through age ten, parents reported child bedtimes, rise times, and amount of sleep during the day (Jenni, Molinari, Caffisch, & Largo, 2007). Similar to the charts used by pediatricians to track height and weight, there was a corresponding pattern of long-term rank order stability, in which children who slept less at one year of age tended to also sleep less through ten years of age. Although this type of research is a good example of a developmental approach to documenting normative patterns of sleep, there remain substantial questions—developmental questions—about *how* these changes occur, *why* these changes occur, and the consequences of these changes for other areas of development.

One area where there is progress in the study of normative changes of sleep and its possible consequences is in the area of slow wave sleep. Ringli and Huber (2011) provide an account of this area of research, the details of which are beyond the scope of this chapter. In brief, changes in slow wave sleep coincide with changes in synaptic density to such a strong degree that changes in slow wave sleep are hypothesized to occur because of changes in synaptic density. Furthermore, brain regions that show changes in slow wave sleep progress from the posterior to the anterior areas, which is consistent with increasing cognitive functioning from childhood through adolescence (Luna & Sweeney, 2004). It is also plausible that increases in synaptic density

in early childhood followed by synaptic pruning in adolescence are influenced by the child's environment (e.g., nutrition, learning, family stress). It appears likely that there is a reciprocal relation between neurological development, developmental changes in sleep architecture, and contextual factors such as familial, social, and educational environments. Ultimately, developmental research that considers changes in slow wave sleep along with other measures such as cognition, language, and attention, may provide information about *why* we sleep and how sleep is related to and/or plays a causal role in development more broadly.

Another area that benefits from a developmental perspective is the study of sleep problems. Along with normative changes in sleep from infancy through adolescence, there is a sizeable minority of children who develop and maintain behavioral sleep problems, including difficulty falling asleep, difficulty staying asleep, and experiencing daytime fatigue (Mindell & Meltzer, 2008). For example, a recent study from a large national sample reported 25% of children between the ages of 6 and 11 and 39% of children between the ages of 12 and 17 did not get adequate sleep one or more nights in the preceding week according to parental reports (Smaldone, Honig, & Byrne, 2007). In addition, Smaldone and colleagues (2007) reported that children and adolescents who were not getting adequate sleep were also more likely to have trouble in school, emotional problems, and health problems. However, the cross-sectional nature of this and other studies (for a review, see Staples & Bates, 2011) makes it hard to establish the causal directions of associations between sleep and daytime functioning. In addition, the definition of poor sleep was based on the previous week, which does not address questions about possible differences between the consequences of short- and long-term sleep problems.

It is likely that short, infrequent periods of poor sleep like those during a brief illness, will have little impact on longer-term development. However, chronic or long-lasting periods of poor sleep during childhood and adolescence may have developmental consequences. Evidence from a variety of studies with children from infancy through adolescence have shown that sleep problems are associated with slower academic gains, increased negative affect, and higher rates of problem behavior (Staples & Bates, 2011). This is particularly evident in studies linking sleep disordered breathing with poorer academic outcomes, increased inattentiveness, difficulty regulating emotions, and increased daytime

fatigue (Beebe, 2011; Beebe, Ris, Kramer, Long, & Amin, 2010). These findings of association between sleep problems and academic and behavioral outcomes point to the need to understand the contexts in which sleep problems occur, are maintained, and change. However, to date, much of the longitudinal research linking sleep with later developmental outcomes leaves questions about the directionality, timing, and magnitude of the association between sleep and development. Although we might prefer to attribute the outcomes to sleep based on sleep restriction and extension experiments, a few of which have been done with children (Carskadon, Harvey, & Dement, 1981; Fallone, Acebo, Arnedt, & Seifer, & Carskadon, 2001; Randazzo, Muehlbach, Schweitzer, & Walsh, 1998, Sadeh, Gruber, & Raviv, 2003), without a developmental analysis we lack a firm basis for this. We think of most common behavior problems as representing a social system's failure to solve smaller conflicts, and we think that chronic sleep deficits could impair children's and parents' efforts to effectively resolve these more minor, "building-block" conflicts, for example through efficiently stopping a child's coercive behavior (Patterson, Reid, & Dishion, 1992). Sleep restriction studies suggest that sleep deficits impair executive functioning (Walker & Stickgold, 2006), which would make it harder to regulate impulses and solve parent-child conflicts.

To understand sleep from a developmental perspective, one must consider questions of stability and change *within* an individual alongside questions of differences *between* individuals. For example, children show relative stability in being short or long sleepers from ages one to ten; however, children who were born in 1974 slept longer on average compared to children born in 1986 (Iglowstein, Jenni, Molinari, & Largo, 2003). The relation between intra-individual stability in conjunction with cohort differences in the amount of sleep highlight the need to study sleep as an "event in a context" (Kagan, 2007). Children are developing at particular rates and in particular families and in eras that could be socioculturally distinct.

Sleep is often considered as a solitary, nonsocial event, but how children transition from wakefulness to sleep, where that transition happens, and what is done if the child awakens at night is decidedly social and shows cultural variation (see Super and Harkness, Chapter 9). For example, parents of infants in the United States place less importance on both regular bedtimes and regular bedtime routines when compared to parents in Italy

(Wolf, Lozoff, Latz, & Paludetto, 1996). Parents of infants in Japan place greater importance on a regular bedtime routine, but not a regular bedtime, compared to parents in the United States (Wolf et al., 1996). Understanding the importance parents place on regular bedtimes as well as regular bedtime routines may shed light on the relation between bedtime schedules and the development and/or maintenance of sleep problems. There are also wide variations in where the transition from wakefulness to sleep occurs, with both bed-sharing and room-sharing depending on the child's age and cultural context (Owens, 2008). One implication is that sleeping behaviors that may be problematic for one family may not be a problem for another, partially due to cultural differences (e.g., bedtime resistance in the United States, nighttime awakenings in Japan; Wolfson, 1996). In terms of development, it is important to understand how cultural factors may influence parental expectations and practices surrounding sleep-related behaviors, which may in turn have consequences for the development of both typical and atypical sleep behavior as well as typical and atypical daytime behavior.

Another difference in sleep that varies both with age and culture are naps. The transition to a single period of nighttime sleep is viewed as a developmental milestone in the United States, with most children no longer napping by age five (Staples & Bates, 2011). However, in Iceland, most children are no longer napping by age three (Owens, 2004). This difference suggests that the transition to a single period of sleep is likely to be a product of both biological forces and cultural practices. For example, in the United States there are large differences in napping between European-American and African-American children, with European-American children ceasing naps at an earlier age than African-American children. If researchers only account for nighttime sleep between the ages of three and five, it would appear that African-American children are getting much less sleep than European-American children (Owens, 2004). However, if the entire 24-hour period is taken into consideration, there is no difference in the amount of sleep between African- and European-American preschoolers. Thus, it appears that sleep development takes place in a larger context that considers familial and sociocultural factors (El-Sheikh, 2011). However, follow-on questions concern how familial and cultural differences arise and what impact they might have on later development. For example, how does earlier or later consolidation into a single period of sleep affect the development

of sleep behavior and/or the development of other behaviors such as self-regulation, attention, or memory? One could ask if children who stop napping earlier also show corresponding advances in other regulatory areas such as motor development, emotion regulation, attentional flexibility, or learning. And if such consequences are found, are they common across all children or do they depend on the child's age (e.g., large differences between ages three and five, but not later) as well as their larger socio-cultural environments (e.g., association of continued naps with lagging regulation occurs only in contexts where napping violates age norms)?

Variations in sleep that are associated with particular times in history, locations, and cultures typify the notion of an "event in context"; studying sleep developmentally also raises the issue of "response in context" (Kagan, 2007), where the same apparent behavior may mask different underlying causes. For example, the reasons why a child takes a long time to fall asleep are likely to differ if the child is three (perhaps they had a late or longer nap than usual) or thirteen (perhaps they are ruminating about an upcoming test or an earlier social interaction). Sleep disturbances, such as nightmares, may have different consequences for daytime functioning in young children (and their parents) compared to adolescents. Additionally, the same apparent behavior may evoke different responses. Not all parents view nighttime awakening as problematic and therefore their responses may differ, which could result in different outcomes. For example, parents who viewed their infant's nighttime crying as a sign of distress were more likely to intervene compared to parents who viewed the behavior as age-typical and not problematic (Erath & Tu, 2011). Intervening during nighttime awakenings during infancy has been associated with maintenance of sleep problems into toddlerhood and early childhood (Mindell & Meltzer, 2008). The interrelatedness between parent beliefs, child behavior, and parental response highlights the need for a developmental systems approach that considers not only the behavior, but also the behavior and response within a larger context.

In order to study sleep in a developmental-systems way, ideally one would measure sleep on more than one occasion. This asks a lot from both researchers and participating families. Choices must be made about the type of sleep measurement (questionnaire, diary, actigraphy, polysomnography, etc.), the number of measurements (daily, monthly, annually), and the breadth and depth of sleep behaviors (e.g., physiological, behavioral; see Spruyt, Chapter 18,

and Horne and Biggs, Chapter 16). Several of these issues are discussed in depth in Part Three of this volume. From a developmental perspective, one of the central issues concerns the number of observations and their spacing.

An inescapable fact of research employing multiple measures is that the number of measurement occasions and the time between occasions limits the scope of the type of change that can be observed (Adolph, Robinson, Young, & Gill-Alvarez, 2008). In the simplest case of two measurement occasions, the only type of change that could be observed is that of linear change. At the other extreme, say, daily measurements for a year, larger trends may be masked by short-term variability. Achieving the balance between capturing the shape of change, maintaining a reasonably accurate measurement of the behavior, and not subjecting participants to undue burden, is not a trivial task. A more complete treatment of the issues concerning developmental research can be found in Laursen, Little, and Card (2012). One method that has been successfully used to balance within-person and between-person variability is the use of the burst measurement design. In this method, data are sampled in short bursts (e.g., daily for several weeks) separated by longer periods (e.g., 6-month intervals). This approach provides, among other things, a more stable estimate of within-person level at each burst occasion, which can then be linked to a longer-term trajectory of change (or stability). For example, in a recent study we measured sleep behaviors over seven consecutive nights followed by a laboratory assessment, which is repeated three times over the course of a year. These burst measurement occasions allow us to answer questions about whether the quality/quantity of sleep the night before, or the average of these variables over the preceding week, is more strongly related to the laboratory measures. In addition, we are able to answer questions about the relation between persistent or intermittent sleep problems and development over the course of a year.

A related issue concerns change versus development (Raeff, 2011). Imagine a situation in which a child who is sleeping through the night experiences several nights of poor sleep due to an illness and then returns to the previous pattern of sleeping. In this example, the child's sleep certainly *changed*, but there was no *development* per se. In general, developmental change occurs when there is a movement toward a more advanced form of development. This is not to say that there are not periods of growth followed by apparent periods of regression. Indeed,

motor, language, cognitive, and emotional development in early childhood all show evidence of high variability in behavior during developmental transitions from earlier to later phases (Adolph et al., 2008; Hollenstein, Granic, Stoolmiller, & Snyder, 2004; Lewis, Koroshegyi, Douglas, & Kampe, 1997; van der Maas & Molenaar, 1992). Our expectation is that if sleep were studied on a daily basis for a sufficient period of time, it would show similar patterns of high variability in children's sleep during the transition from two daily sleep periods to a single period of sleep occurring at night. Developmentally, then, measurements must be sufficiently dense to separate meaningful change from normal variations in behavior. Of course, the time scale of observation would depend on the particular question of interest.

It is also possible that sleep may show increased variability during development in other domains (e.g., motor, cognitive, language). For example, a child who is beginning to crawl may experience a period of sleep disruption due to increased motor activity after falling asleep. In this example, changes in sleep during the later part of an infant's first year may be normative but not necessarily developmental. To date, there have been a handful of studies demonstrating greater nighttime awakenings in infants who are crawling/walking compared to infants who are not crawling/walking (Scher, 1996; 2005; Scher & Cohen, 2005). However, since each infant was assessed at only one time point, the relation between motor development and changes in sleep patterns may merely coincide due to some other factor. For example, it may be that children who are more active during the daytime crawl earlier and are also more active at nighttime compared to their less active peers. When the needed longitudinal data are collected, causal developmental-process associations between motor development and changes in sleep may be discovered.

### **Integrating Sleep and Developmental Research**

Part of framing the integration of sleep and developmental research begins with the types of questions that require a developmental approach in research design, measurement, and analysis. For example, are the age-related changes in slow wave sleep that have been observed in studies with between-person designs also observed longitudinally within person? What person, context, and time variables are associated with or play a causal role in the reduction of slow wave sleep? Are variations in

slow wave sleep preceded or followed by periods of learning? For example, do children show changes in slow wave sleep relative to the academic calendar? Is the relation between learning and sleep architecture the same in childhood and adolescence? Is there a longitudinal association between motor development and sleep development? Is there a longitudinal association between self-regulation development and regulation of the sleep-wake cycle? Is the association between regulation and sleep bidirectional, reciprocal, constant during development, or changing in sync with other developmental processes (e.g., synaptic development, motor development, puberty)? Each of these questions calls for research that considers process, person, context, and time to some degree. Preliminary answers to some of the questions can come from cross-sectional, multi-age samples, but we see the need ultimately for longitudinal designs that untangle within-person variability from between-person variability.

Another aspect of blending of sleep and developmental research areas is to ground the work in a larger theoretical milieu. While developmental systems theories provide guidance on *what* to consider, other theories will need to be utilized to explain *how* sleep and daytime behaviors mutually or directionally influence each other. The specific mechanistic hypothesis that one chooses will depend largely on the level at which the behaviors are observed. To date, sleep has been considered as part of a larger regulatory system that operates at the biological (Carskadon et al., 2004), neurological (Ringli & Huber, 2011), cognitive (Walker, 2009), and sociocultural levels (El-Sheikh, 2011). Each of these system levels are inherently interrelated, simultaneously active, going through periods of change and stability, thus providing a complex picture of the ways in which sleep and daytime behavior are inexorably linked.

In our research on the development of self-regulation in toddlerhood, we have posited three possible mechanisms for the role of naturally occurring sleep deficits. First, sleep deficits can be viewed as stressors (McEwen, 2007; Weissbluth, 1989) that produce dynamics in cortisol and other stress hormone levels that, in turn, can influence cognitive processing of information (Blair, Granger, & Peters Razza, 2005). Chronic sleep deficits, like chronic stress in general, would produce abnormal stress hormone dynamics with negative implications for cognitive processing and thus for self-regulation. Second, sleep deficits can more directly produce difficulties in cognition that impair behavioral and learning processes. In Dahl's (1996) terms, sleep deficits

produce desynchronization of neuronal communication between brain centers, which, in complex high-challenge situations such as preschool, might lead to dysregulated affect and behavior. And third, sleep deficits diminish consolidation of learning, except for negative affect material (Walker, 2009). Thus, in addition to experiencing cognitive processing deficits due to hormonal and neural-network disruptions, the sleep-deprived child may fail to acquire sufficient knowledge from daily experience, especially in relation to amounts retained from negative emotional experiences. Each of these mechanisms require measurement of somewhat different systems and it is likely that until developmental data are available on all of these levels, we will not have a complete picture of how sleep plays a role in daytime functioning.

Framing questions of sleep developmentally and having a theory of how they are likely to relate then leads to questions of what to measure. The issues regarding measurement of sleep mentioned previously also apply to the selection of developmentally sensitive measures—what, how often, in what detail. As is always true of measurement, the answers depend upon the question. If one is interested in the relation between bedtime routines and the development of behavioral sleep problems during the transition from crawling to walking, then a burst measurement design that includes a 7- to 10-day assessment of sleep with daily diaries and actigraphy repeated at 4- to 6-weeks intervals seems appropriate. If one is interested in the relation between sleep and the development of behavior problems, choosing the instrument to measure behavior problems will depend upon how often the assessments are repeated. For example, the widely used the *Child Behavior Checklist* (Achenbach, 1991) will be useful for studies examining changes that occur at semiannual or longer intervals, whereas the *Eyeberg Child Behavior Inventory* (Eyeberg & Pincus, 1999) may be more useful for assessments happening at more frequent intervals. Ultimately, the selection of measures will play a large role in whether a particular study is able to detect developmental change. If the measure selected is insensitive to change during the assessment period, then researchers may incorrectly conclude that there is no relation between sleep and the outcome variable. In contrast, if the measure shows wide variability with repeated assessments, then researchers may incorrectly conclude there is a relation between sleep and the outcome variable if other relevant factors such as parenting or cognitive development are not also assessed.

Similar issues arise when developmental researchers assess sleep. Some measures of sleep may be less sensitive to important sleep behaviors than others. For example, parents tend to underreport the number of times their child awakens at night (Tikotzky & Sadeh, 2001). Also, parents may underreport the presence of a sleep problem if they consider their child's sleep behavior (e.g., night awakening) to be age-normative and transitory (Lam, Hiscock, & Wake, 2003). Sleep problems in childhood often occur in conjunction with other issues such as medical problems, developmental disorders, and psychological and social relationship problems. Primary sleep disorders, such as sleep disordered breathing are associated with attention, emotion regulation, and academic performance problems (Beebe, 2006). Several recent reviews demonstrate sleep problems are often higher in children with a wide variety of conditions such as ADHD (Cortese, Faraone, Konofal, & Lecendreux, 2009), autistic spectrum disorder (Cortesi, Giannotti, Ivanenko, & Johnson, 2010), asthma (Bandla & Splaingard, 2004), anxiety, and depression (Dahl & Harvey, 2007). We would encourage developmental researchers, particularly those using normative community samples, to include health-related measures to better account for sleep problems that may be primarily physiological (e.g., sleep disordered breathing) and distinct from those that are primarily psychological and behavioral (e.g., nightmares or irregular parent management of bedtime).

## Summary

In general, we have approached this chapter by asking what would it mean to view sleep from the perspective of developmental science. This was initially challenging for us because of our difficulty in imagining how sleep could be viewed from any other larger theoretical context. At its core, developmental science is the study of change throughout the lifespan. Why and by what mechanisms sleep is critical for human survival remains an open question. What is clear is that variations in sleep are related to learning, memory, information processing, motor coordination, decision making, emotion regulation, neural development, and other biological processes. How these variations in sleep come to be, the conditions under which variations in sleep are maintained or changed, and the consequences of variations in sleep for development are but a few ways that a developmental systems approach to understanding sleep can be informative.

Exciting advances will happen when sleep and human development are simultaneously considered.

Sleep research has made enormous strides toward a truly developmental perspective since 1978, when most our knowledge about the role of sleep in daytime functioning was based on studies of young, healthy men (Horne, 1978). Since then, studies have focused on sleep loss in adolescence (Wolfson & Carskadon, 1998; Acebo & Carskadon, 2002; Carskadon et al., 2004; Labege, Petit, Simard, & Vitaro, 2001), normative changes in sleep patterns (Carno, Hoffman, Carcillo, & Sanders, 2003; S. Ottaviano, Giannotti, Cortesi, Bruni, & Ottaviano, 1996; Palmstierna, Sepa, & Ludvigsson, 2008), and the continuity and change in sleep problems from infancy into childhood and adolescence (Anders & Keener, 1985; Ednick et al., 2009; Gaylor, Burnham, Goodlin-Jones, & Anders, 2005; Iglowstein et al., 2003; Jenni et al., 2007). In the next decade there will likely be significant strides in our understanding of why we sleep and how sleep is necessary for human development.

How we approach the challenges of measuring individuals over time, and how often the assessments are made, will determine much of what is learned about sleep and development. The suggestion to measure behaviors in more than one domain is to explicitly direct researchers to consider that development does not happen uniformly across all areas of behavior, that development is shaped by biological, psychological, and social factors, and that understanding development in one domain will not be complete until it is placed within a larger developmental context.

### Future Directions

From the perspective of developmental systems theories, future research needs to:

- Measure multiple aspects of sleep (e.g., behavioral, physiological, health-related)
- Follow individuals over time
- Take repeated measurements sufficiently close in time before, during, and after a period of developmental change
  - Measure variables in more than one domain (e.g., sleep, cognition)
  - Measure person and situation variables that are hypothesized not to change during the period under study (e.g., temperament, personality, housing)

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# Developmental Changes in Sleep: Infancy and Preschool Years

Kurt Lushington, Yvonne Pamula, James Martin, and John Declan Kennedy

## Abstract

The early years of childhood are characterized by rapid advances in growth, cognition and behavior. It is also the period of the human lifespan most occupied by sleep with correspondingly rapid changes in the structure, organization and regulation of sleep. The development and maturation of sleep during childhood is a dynamic process influenced by a range of physiological, genetic, biological, and psychosocial factors. Marked transformations are observed in sleep neurophysiology during the first few months of life, reflecting underlying central nervous system (CNS) growth and maturation while the emergence of circadian, homeostatic and ultradian regulatory processes modulate the timing and distribution of sleep-wake states. It is now well recognized that adequate sleep is essential for the health and well-being of children, with increasing evidence that sleep plays a crucial role in physical, psychological, and cognitive development. An improved understanding of normal sleep across the entire pediatric age range is therefore essential in understanding the role of sleep in child development and for the timely identification and treatment of sleep problems.

**Key Words:** sleep ontogeny, sleep EEG, circadian, infant, development

## Ontogeny of Sleep EEG

*“By age 2 years, the average child has spent 10,000 hours asleep and about 7,500 hours in all waking activities combined ... by early school age, a typical child has spent more time asleep than in all social interactions, environmental explorations, eating, playing or any other waking activities” (Dahl, 1996, pp. 44–45).*

Despite the fact that we spend approximately one-third of our lives asleep, the functions of sleep remain largely unknown. Nevertheless, it is well recognized that adequate sleep is an essential requirement for normal physiological and psychological functioning and that sleep loss or disruption can result in a range of adverse health outcomes. The early years of childhood are characterized by rapid advances in growth, cognition and behavior. It is also the period

of the human life span most occupied by sleep, with correspondingly rapid changes in sleep structure and organization. That early childhood cognitive and behavioral advancement parallels sleep development is not surprising given the substantial degree of central nervous system (CNS) growth and differentiation that occurs during this time (Casey, Tottenham, Liston, & Durston, 2005; Levitt, 2003; Segawa, 2006). However, sleep ontogenesis may be more than just a correlate of CNS development, as mounting evidence intimates reciprocal interactions between sleep and brain plasticity. In particular, sleep may play a role in two aspects of brain plasticity that underlie neurocognitive development: (1) brain growth and maturation and (2) memory consolidation (Graven, 2006; Maquet, Smith, & Stickgold, 2003; Miyamoto & Hensch, 2006; Peirano & Algarin, 2007; Sejnowski & Destexhe, 2000; Tarullo, Balsam, & Fifer, 2011).

Newborn infants spend approximately 60% of the 24-hour period sleeping, and while this percentage declines over childhood, children aged 4 to 5 years still sleep almost 12 hours a day (Galland, Taylor, Elder, & Herbison, 2012). Consequently, it is likely that a significant amount of early human development occurs during sleep (Sheldon, 1996). Conjecture of a more direct relationship between sleep and brain development was first advanced by Roffwarg, Muzio, and Dement (1966) following the unexpected finding that preschool children had more REM sleep than adults and the discovery of REM sleep in infants, suggesting that REM sleep had a functional role (see Sheldon, Chapter 2). Since this time, a growing body of evidence based on animal and human studies supports the view that sleep may play an important role in neural and cognitive development, with accumulating data suggesting that there may even be periods of brain development that are sensitive to sleep (Arditi-Babchuk, Feldman, & Eidelman, 2009; Aton et al., 2009; Bernier, Carlson, Bordeleau, & Carrier, 2010; Dionne et al., 2011; Frank, Issa, & Stryker, 2001; Holditch-Davis, Belyea & Edwards, 2005; Mirmiran & Ariagno, 2003; Mirmiran, Uylings & Corner, 1983; Ringli & Huber, 2011; Scher, 2005; Thomas et al., 2000; Touchette et al., 2007; Weisman, Magori-Cohen, Louzoun, Eidelman, & Feldman, 2011).

One of the putative mechanisms by which sleep may facilitate neural development is via endogenous stimulation arising from electroencephalographic (EEG) oscillations generated during sleep (particularly ponto-geniculo-occipital [PGO] waves, sleep spindles, and delta waves) or other sleep-related activities such as limb twitching (Blumberg, 2010; Denenberg & Thoman, 1981; Frank & Stryker, 2003; Jenni, Borbely, & Achermann, 2004; Marks, Shaffery, Oksenberg, Speciale, & Roffwarg, 1995; Roffwarg et al., 1966). The importance of endogenous neural activation in stimulating brain development is now well recognized, particularly with respect to neurogenesis, cell differentiation, neuronal migration, dendritic branching, apoptosis, and the formation of neural networks (Corner & Ramakers, 1992; Graven, 2006; Kilb, Kirischuk, & Luhmann, 2011; Penn & Shatz, 1999).

Given the above findings, there has been renewed interest in the development of sleep during infancy and childhood. A better understanding of the neural processes occurring during sleep, particularly during early human development, may elucidate and clarify the role of sleep in brain and cognitive development. The EEG provides a unique opportunity

to study brain function and neural processes during sleep. Moreover, many aspects of the sleep EEG show predictable age-related changes, some of which are related to underlying brain reorganization and maturation, and thus the trajectory of sleep development can be used as a surrogate marker of CNS development and integrity (Feinberg & Campbell, 2010; Kohyama, 1998; Parmelee & Stern, 1972; Scher, 2011; Sheldon, 1996). The following section presents a brief outline of the general neurophysiological characteristics of sleep. This is followed by an overview of the development of the sleep EEG during infancy and early childhood.

### ***The Electrophysiological Characteristics of Sleep***

The human brain is distinguished by three main behavioral states: wake, non-rapid eye movement (NREM) sleep, and rapid eye movement (REM) sleep. These behavioral states are characterized by a recurring and relatively stable pattern of physiological and behavioral parameters that represent distinct modes of brain organization and function (Curzi-Dascalova, 2003; Parmelee & Stern, 1972; Prechtl, 1974). Conventionally, sleep-wake states are defined by the features of three major electrophysiological signals: the electroencephalograph (EEG), eye movements (electrooculogram, EOG) and chin muscle tone (submental electromyogram, EMG). In normal healthy children and adults these three parameters display a distinctive and concordant pattern of behavior that differs markedly between NREM and REM sleep, enabling highly reproducible sleep state identification. In the neonate and young infant, however, sleep structure and organization is comparatively immature and disorganized in the first few months of life as underlying brain structures and sleep regulatory systems are not yet fully developed. As a result, coherence between electrophysiological and behavioral parameters is quite variable in the neonatal period, making state identification more difficult. Additional parameters including behavioral observations and cardio-respiratory patterns are therefore used to assist in sleep-wake state identification. As the sleep EEG of newborns is also less differentiated than that of older children and adults, different terminology (*active sleep*, *quiet sleep*, and *indeterminate sleep*) is used to describe sleep states in early infancy. Over the first 3 to 6 months of life the characteristic features of NREM sleep gradually appear in the infant EEG, reflecting underlying CNS maturation and reorganization.

## EEG Patterns

The EEG, when recorded from electrodes applied to the scalp, measures the summed electrical activity of numerous neurons, providing good temporal but poor spatial resolution. EEG activity is described by a number of standardized parameters including amplitude (or voltage, in microvolts), frequency (in cycles per second, hertz), and spatial distribution and patterning. EEG frequencies are conventionally grouped into four commonly used band widths: delta (<4 Hz), theta (4–7 Hz), alpha (8–13 Hz) and beta (>13 Hz). Distinct EEG waveforms and rhythms can also be seen over the background EEG activity and are described by their morphology and topography (the EEG electrode derivation in which they appear).

Additional topographical descriptors include whether EEG waveforms appear in homologous head regions (symmetrical versus asymmetrical) and, if symmetrical, whether their appearance is temporally synchronized. While at any given point in time the EEG comprises a mixture of frequencies, specific frequencies dominate particular sleep stages, and some EEG waveforms (e.g., sawtooth waves) are sleep-stage specific.

One of the most striking differences between wake and sleep is EEG activity. During wakefulness the EEG is characterized by relatively low-voltage, mixed-frequency activity, with alpha and beta (typically 18–25 Hz) frequencies dominating (Avidan, 2005). During relaxed wakefulness with eye closure, alpha rhythm (8–13 Hz) is very prominent, particularly in the occipital EEG derivations; this pattern is known as the *dominant posterior alpha rhythm*. In adults and older children, sleep onset begins with NREM sleep. Non-rapid eye movement sleep is further divided into four stages (1 to 4)<sup>1</sup> which approximate a continuum of sleep depth, with stage 1 being the lightest and stage 4 the deepest sleep and with arousal thresholds being correspondingly lowest in stage 1 and highest in stage 4 NREM sleep (Carskadon & Dement, 2011).

Stage 1 NREM sleep is characterized by a low-voltage mixed-frequency EEG background pattern with theta activity dominating. The EEG is generally slower than that observed during wake. Early in stage 1 sleep slow rolling eye movements may be observed, while vertex sharp waves may be seen in the latter stages. Stage 1 NREM sleep

is a transitional sleep stage often following wake or gross body movements (Rechtschaffen & Kales, 1968). Stage 2 NREM sleep is characterized by two landmark EEG waveforms: sleep spindles and K-complexes. Sleep spindles and K-complexes show marked developmental changes during infancy and will be discussed in more detail later. In adults, sleep spindles appear as transient clusters of rhythmic activity with a mean frequency range of 12–14 Hz and often having a waxing and waning appearance. A K-complex is a transient biphasic EEG waveform comprising a large sharp negative EEG wave (upward deflection on the EEG trace) followed immediately by a slower positive (downward) component. K-complexes may appear spontaneously, in response to external stimuli, and may have sleep spindles as part of the complex (Rechtschaffen & Kales, 1968). Stages 3 and 4 NREM sleep are easily recognized by the appearance and predominance of slow, high-voltage delta waves (0.5–2 Hz). When combined, stages 3 and 4 NREM sleep are termed *slow wave sleep* (SWS). The very-high-voltage delta waves seen in SWS arise because the electrical activity of neuronal populations is very highly synchronized. Eye movements are generally absent in stage 2 and slow wave sleep, and chin EMG activity is reduced compared to wakefulness.

The EEG of REM sleep is very different from that of NREM sleep and is characterized by low-voltage, mixed-frequency (mostly theta) activity similar to that of wakefulness. Alpha activity is prominent in REM sleep but is typically 1–2 Hz slower than seen during wake (Rechtschaffen & Kales, 1968), and trains of sawtooth waves in the theta frequency range, having a characteristic triangular/serrated appearance, are commonly seen. Thus, during REM sleep the brain is significantly activated due to a high level of cortical activity. Two patterns of physiological behavior are seen in REM sleep: tonic and phasic. The tonic phase dominates REM sleep and is relatively quiescent, characterized by skeletal muscle atonia and the absence of eye movements. Phasic REM sleep is characterized by bursts of rapid, conjugate eye movements (REMS), transient muscle twitching, and irregularities in respiration and heart rate (Rama, Cho, & Kushida, 2005), while sleeping NREM and REM sleep periods alternate in a predictable cycle. The sequence, distribution and duration of sleep states across the night can be displayed visually as a sleep hypnogram. As with other aspects of sleep, sleep cycling shows marked developmental changes across the human lifespan.

The human sleep EEG undergoes significant ontogenetic changes, particularly in the first few years of life. In newborn infants the sleep EEG does not show the degree of differentiation and organization as described above but gradually develops into this pattern over the first 6 to 12 months. The following section broadly outlines sleep development in normal, healthy, term children from the time of birth to the early preschool years.

### ***Development of the Sleep EEG***

The structure and regulation of sleep in infants and children is significantly different from that of adults and in addition to showing a greater variety of EEG waveforms, infants and children often have higher EEG amplitudes (Scholle & Schafer, 1999). Furthermore, while sleep development in normal children follows an organized and predictable pattern of change, beginning during fetal development and continuing into adolescence, there is significant interindividual variation in the trajectory of sleep maturation (Hoppenbrouwers, Hodgman, Arakawa, Geidel, & Serman, 1988; Sheldon, 1996). Thus variation is the rule rather than the exception. Sleep-wake states first emerge during fetal development. Cyclic periods of activity and rest become established after 24–28 weeks of gestation, with distinct cycling between REM-like and NREM-like sleep usually evident by 32 weeks of gestation (Curzi-Dascalova, 2003; Koos, 2008). Sleep-wake states in the premature infant will not be discussed here (see Andre et al., 2010; Vecchierini, André, & d’Allest, 2007), but have been well studied due to the extended period these infants spend in the neonatal intensive care unit where comprehensive and serial EEG monitoring is often performed.

The coordination and integration of physiological and behavioral activities by the CNS, including the generation of sleep, requires complex interactions between multiple interconnected neuronal networks. In the newborn infant neuronal networks are comparatively less developed, resulting in reduced concordance between the physiological and behavioral parameters that define behavioral states. With increasing postconceptional age physiological and behavioral variables become more coupled, leading to the emergence of more organized and sustained sleep-wake patterns. These developmental changes are believed to reflect maturation of the CNS, in particular the development of inhibitory and feedback control mechanisms in the brain (Parmelee & Stern, 1972). Maturation of sleep in the infant is reflected by the following features (Anders &

Keener, 1985; Curzi-Dascalova & Challamel, 2000; Kohyama, 1998; Parmelee & Stern, 1972):

- increasing concordance among physiological and behavioral state parameters
- increasing quiescence and stability of several physiological processes (e.g., motor activity, respiration)
- the emergence of organized and stable between-state transitions
- increasing differentiation of the NREM sleep EEG including the disappearance and appearance of specific EEG waveforms and rhythms
- modification of the ultradian sleep cycle
- the emergence of a sleep circadian rhythm.

### ***Sleep Organization and EEG Patterns in the Neonatal Period***

The brain in developing infants is immature, and a degree of maturation is necessary before the classical behavioral states of NREM and REM sleep can be clearly differentiated. The sleep states in newborns, therefore, have some but not all the features of NREM and REM sleep. Three sleep states can be identified in the normal term infant: active sleep (AS), quiet sleep (QS) and indeterminate sleep (IS). While the precise mechanisms underlying the developmental progression of sleep are not yet fully understood, AS is believed to be the precursor of REM sleep while quiet sleep is believed to differentiate into the four NREM sleep stages (Anders, Emde, & Parmelee, 1971). Indeterminate sleep displays characteristics of both AS and QS and therefore does not unequivocally meet the criteria for either of these sleep stages. Within these behavioral states four major EEG patterns can be distinguished (Anders et al., 1971; Scher, 2006; Sheldon, 1996):

- (1) A high-voltage, slow-frequency EEG pattern (HVS) dominated by continuous, moderately rhythmic delta activity and seen predominantly in QS.
- (2) *Tracé alternant* (TA), a discontinuous EEG pattern seen only in QS. *Tracé alternant* typically comprises 3–8 second bilateral bursts of high-voltage, 0.5–3 Hz EEG waves that may have fast, low-voltage sharp waves superimposed. These high-voltage bursts are interspersed with low-voltage, mixed-frequency EEG activity of similar duration.
- (3) A low-voltage irregular EEG pattern (LVI) seen in AS or when awake. The LVI pattern is characterized by a continuous, low-voltage, mixed-frequency EEG that is generally dominated by theta rhythms (especially when following QS) but

also contains significant amounts of delta activity (particularly if occurring at sleep onset).

(4) A mixed-frequency EEG pattern (M) that can be seen in wake and AS but is infrequently seen in QS. The mixed pattern consists of slow, moderate- to high-voltage delta activity and low-voltage mixed-frequency (theta, alpha, and beta) EEG components. The amplitude is usually lower than that seen in the HVS pattern.

Due to overlap of some of these EEG patterns across sleep-wake states and reduced concordance among state parameters (as discussed earlier) behavioral observations and cardiorespiratory patterns are important adjuncts to electrophysiological signals (EEG, EOG, submental chin EMG) in identifying behavioral states. Relevant behavioral observations include the type, pattern and frequency of eye movements, facial expressions, motor activity, and vocalizations (Thoman, 1990). With respect to cardiorespiratory behavior, irregularity in heart and respiratory rate and the presence of respiratory events such as sighs, apneas, and periodic breathing aid in sleep state identification (Anders et al., 1971; Sheldon, 1996).

Sleep in infants typically begins with AS, which can make sleep onset difficult to establish as the EEG of wakefulness and AS are not always easily differentiated. Sleep onset is therefore often behaviorally determined by the presence of sustained eye closure. The mixed (M) and LVI EEG pattern is seen in AS or, rarely, the HVS pattern, but TA does not occur (Anders et al., 1971). The background EEG voltage of AS is somewhat higher than seen in older children and adults (Hoban, 2005). Rapid eye movements (REMs) are usually observed, but slow rolling eye movements may also be present. Considerable motor activity occurs during AS (phasic periods), which is interspersed with periods of quiescence (tonic intervals). Facial movements commonly observed include sucking motions, twitches, smiles, frowns, and grimaces, and vocalizations (cries, whimpers and grunts) may be heard. Irregular and brief movement of digits and limbs is interspersed with more prolonged gross body movements. Body movements may be slow and writhing or sudden and jerky in nature and are more frequent and of longer duration than seen in older children and adults (Anders et al., 1971; Kahn, Dan, Groswasser, Franco, & Sottiaux, 1996; Sheldon, 1996). While muscle tone is reduced during AS there is frequent phasic activity and the pattern of the chin EMG can be quite variable and difficult to interpret, particularly if the infant is using a pacifier. During AS heart rate is quite variable and respiration is irregular,

particularly during phasic periods. Episodes of tachypnea and bradypnea may be observed in addition to central apneas. Periodic breathing may also be present, particularly in preterm infants, although persistent periodic breathing in the neonatal period, or once the infant reaches conceptional age, suggests an underlying respiratory control problem (Sheldon, 1996). The generalized skeletal atonia which occurs during AS and a highly compliant chest wall seen in infants and young children can result in paradoxical breathing during AS.

Quiet sleep by contrast is characterized by an absence of body movements (except for occasional startles or twitches), regular and stable cardiorespiratory patterns, and an absence of REMs. The EEG shows the HSV, TA, or M pattern but not the LVI pattern (Anders et al., 1971). Indeterminate sleep does not unequivocally meet the criteria for AS or QS and can be seen interrupting an ongoing sleep state when the infant is aroused or occurring as a between-state transition (Anders et al., 1971; Curzi-Dascalova, 2003). Indeterminate sleep is more likely to occur when transitioning from AS to QS than when transitioning from QS to AS (Anders et al., 1971). There has been a tendency to disregard IS as little more than a transitional sleep state; however, IS represents a distinct and independent mode of CNS functioning (Curzi Dascalova, 2001) and excessive amounts or persistence of this sleep state in infancy may reflect a delay in CNS maturation (Anders et al., 1971; Gould, 1983; Ornitz, 1972).

Newborns spend approximately 15 hours of the 24-hour period sleeping (Galland et al., 2012), half of which is spent in AS. As stated previously the first sleep state typically seen in infants on entering sleep is AS, which is followed by a period of QS and then alternating episodes of AS and QS. The first episode of AS at sleep onset is often shorter than subsequent episodes (Roffwarg et al., 1966). Indeterminate sleep may or may not occur between episodes of AS and QS. Sleep is also punctuated by periods of wakefulness, particularly related to feeding. In newborns the transition between behavioral states is often quite rapid and the AS-QS cycle ranges between 30–70 minutes in duration (Scher, 2006; Sheldon, 1996).

### ***Developmental Changes in the Sleep EEG During the First Twelve Months***

During the first 3 months post-term, the sleep EEG gradually changes from the neonatal pattern to the infant pattern (de Weerd & van den Bossche, 2003). Two major changes in particular occur during

this period: (1) the EEG begins to differentiate into the four NREM sleep stages and (2) the proportion of AS decreases. In normal term infants the *tracé alternatif* pattern disappears between 3–4 weeks of age and is gradually replaced by high-voltage, slow-frequency EEG activity (Sheldon, 1996). Rudimentary sleep spindles emerge at around 4 weeks post-term but are often fragmentary and may be difficult to distinguish (Shibagaki, Kiyono, & Watanabe, 1982). Infant sleep spindles exhibit interhemispheric asymmetry and asynchrony (Sheldon, 1996) and have a different morphology (a spiky negative component with a rounded positive component), which gradually changes during the 9 months following birth (Jenni et al., 2004). By 8 to 12 weeks post-term, spindles are usually seen in most infants and are generally well formed, and this coincides with the emergence of a prominent peak in sigma activity in the EEG spectrum during QS (Ellingson, 1982; Jenni et al., 2004; Sankupellay et al., 2011; Sheldon, 1996). Spindle density and duration appears to be maximal at around 3 months of age with major developmental changes seen in topography, morphology, amplitude, and frequency over the first 12 months following birth (Dan & Boyd, 2006; Ellingson, 1982; Grigg-Damberger et al., 2007; Louis, Zhang, Revol, Debilly, & Challamel, 1992; Scholle & Schafer, 1999; Shibagaki, Kiyono, & Watanabe, 1982; Tanguay, Ornitz, Kaplan, & Bozzo, 1975). However, there is significant interindividual variation in development, and spindles may also show interhemispheric asynchrony up to 2 years of age (Dan & Boyd, 2006; Ellingson, 1982; Kahn et al., 1996). Sleep spindles are generated by synchronized activity of thalamocortical and thalamic reticular neurons (Destexhe, 2009), and the maturational changes seen in the first few months of life are believed to reflect developmental changes occurring in thalamocortical structures and in myelination and dendrite growth (Gould, 1983; Jenni et al., 2004; Louis et al., 1992). A reduction in spindle frequency and sigma activity has been reported in near-miss SIDS infants and infants at increased risk of SIDS (Gould, 1983; Guilleminault & Coons, 1983), while spindle abnormalities have also been found in children with CNS or neurodevelopmental disorders such as Down's syndrome (Sheldon, 1996; Shibagaki, Kiyono, & Watanabe, 1982; Shibagaki, Kiyono, Watanabe, & Hakamada, 1982). These findings may reflect delayed CNS maturation or more specific structural or functional abnormalities in the cerebral cortex or thalamus (Guilleminault & Coons, 1983; Gould 1983; Shibagaki, Kiyono, & Watanabe, 1982).

One of the most striking changes seen in sleep during human development is the marked decrease in AS/REM sleep. From birth through to adolescence there is an 80% reduction in REM sleep, most of which occurs during the first 5 years of life. In contrast, NREM sleep only declines by 25% (Roffwarg et al., 1966). The functional significance of this ontogenetic change has been the subject of much discussion (Garcia-Rill et al., 2003; Horne, 2000; Roffwarg et al., 1966). At conceptional term, AS comprises 50% of total sleep time which declines to between 35%–40% by 1 year of age (Anders & Keener 1985; Sheldon, 1996). This reduction in AS is paralleled by a concomitant increase in QS (Anders & Keener 1985; Hoppenbrouwers et al., 1988). In addition to the reduction in AS, sleep onset begins to gradually shift from AS to QS between 10–12 weeks post-term (Sheldon, 1996). The developmental changes in the distribution of AS and the proportion of AS to QS is sometimes viewed as an indicator of CNS maturation (Hoppenbrouwers et al., 1988; Ornitz, 1972). At both 6 weeks and 3 months of age, near-miss SIDS infants were found to have a significantly higher proportion of sleep onset via AS compared to control infants, which the authors concluded may reflect maturational delay or abnormalities in brain functioning (Guilleminault & Coons, 1983).

Three months of age appears to be a significant juncture for infant neurodevelopment, and the degree of sleep maturation at this time can be used as a surrogate benchmark for CNS organization (Sheldon, 1996). At 3 months post-term the EEG of QS has started to differentiate, and concordance between physiological and behavioral sleep–wake parameters is high (Kohyama, 1998). Coincident with these neurophysiological developments is the emergence during wake periods of attentive behaviors and social interactions. These maturational attainments are thought to reflect the development of inhibitory and feedback control mechanisms, particularly with respect to interactions between the brainstem and higher brain centers (Parmelee & Stern, 1972).

Significant changes in sleep organization and in the sleep EEG continue between 3–12 months post-term. Sleep gradually becomes consolidated into fewer but longer periods that occur mostly at night (Coons, 1987). Sleep onset is more common through QS after 3 months post-term, although episodes of AS onset can still be seen in normal infants up to 6–8 months of age (Sheldon, 1996). By 6 months of age the three major NREM sleep states (stage 1, stage 2 and SWS) can usually be identified in most infants. Over the course of the next 6–12 months the NREM EEG continues

to become better differentiated, with changes in EEG amplitude and frequency enabling clearer delineation between stage 2 and SWS (Sheldon, 1996; Metcalf et al., 1971). Rudimentary vertex sharp waves can be seen in the neonatal EEG, and by 6 months of age they are generally well established and continue to undergo maturational changes in morphology, amplitude, frequency and duration throughout childhood, attaining the adult form by early adolescence (Grigg-Damberger et al., 2007; Sheldon, 1996). Clearly identifiable spontaneous K-complexes first appear in the infant EEG at around 6 months of age and undergo changes in morphology between the ages of 6 months and 2 years and again between the ages of 6 to 12 years (Metcalf et al., 1971). Age-related changes are also seen in the frequency with which spontaneous K complexes are generated (Grigg-Damberger et al., 2007). The dominant posterior alpha rhythm emerges between 3–4 months of age and shows the following age-related increases in EEG frequency: 3–4 months: 3.5–4.5 Hz; 5–6 months: 5–6 Hz; 3 years: 8 Hz; 9–10 years: 9 Hz and 15 years: 10 Hz (Grigg-Damberger et al., 2007).

In summary, as the infant brain matures the relative amount of time spent in active sleep decreases while quiet sleep increases, and by the first year of life sleep has evolved into well-defined REM and NREM sleep (Louis, Cannard, Bastuji, & Challamel, 1997). In conjunction with the changes in quiet and active sleep, the amount of time spent asleep also decreases. Data from a large US survey suggests that sleep length decreases from up to 18 hours in newborns, to 14–18 hours in the first year of life, to 12–15 hours by the third year of life, and 11–13 hours by the fifth year of life (National Sleep Foundation, 2012). Much of this decrease is due to the reduction in active sleep. Sleep length and the distribution of active–quiet sleep demonstrate large interindividual differences and especially in the first year of life, with some newborns sleeping as few as 10 hours and others up to 18 hours per day, and active sleep accounting for as little as 30% and up to 70% of sleep time (e.g. Anders, Keener, & Kraemer, 1985; Iglowstein, Jenni, Molinari, & Largo, 2003). The preference for sleep length appears to remain consistent across childhood (Touchette et al., 2007) suggesting a genetic contribution to sleep length (He et al., 2009; Hor & Tafti, 2009).

### ***Sleep EEG Development: One to Five years***

Comparatively few studies have been undertaken to investigate sleep EEG development in normal children aged between 1 and 5 years. By 1 year of age sleep has attained a more mature pattern and changes in sleep architecture occur more slowly than

seen in the first 12 months of life (Kahn et al., 1996; Sheldon, 1996). By 1 year of age the proportion of NREM sleep is greater than REM sleep, which is a reversal of the relationship seen at birth (Anders & Keener, 1985). The proportion of REM sleep continues to decline during early childhood, reaching the adult level of 20%–25% of total sleep time by 5 years of age (Sheldon, 1996). The distribution of NREM and REM sleep across the night changes, resulting in a preponderance of NREM sleep in the first third of the night and REM sleep toward the end of the night (Kahn et al., 1996). Sleep cycle length is approximately 60 minutes in duration at 2 to 3 years of age, which gradually increases to 90 minutes in duration by age five (Sheldon, 1996).

Changes in the sleep EEG are less prominent between the ages of 1 to 5 years. In general, background EEG frequencies are a little slower and EEG amplitudes are significantly higher than seen in adults (Scholle & Schafer, 1999; Sheldon, 1996). Prominent features of sleep–wake characteristics in early childhood are the EEG patterns of wakefulness and drowsiness, which are quite different compared to adults (Grigg-Damberger et al., 2007; Sheldon, 1996). Furthermore, a number of normal EEG patterns or variants can be seen during sleep in the pediatric age group that are not normally present in adults. These EEG patterns include anterior slow wave activity, hypnagogic hypersynchrony, hypersynchronous theta, post-arousal hypersynchrony, rhythmic anterior theta activity of drowsiness, and the frontal arousal rhythm. Discussion of these EEG patterns is beyond the scope of this chapter and the reader is referred to Westmoreland and Klass, (1990), Sheldon, (1996) and Grigg-Damberger et al. (2007) for a more comprehensive discussion.

### **Overview of Sleep Organization**

The induction and maintenance of sleep is facilitated by multiple physiological and psychosocial factors. The most well accepted model for describing the regulation of sleep is Borbely's two-process model, where it is proposed that sleep propensity is dependent on the interaction between the (a) time spent awake (Process-S) and (b) time of day or circadian phase at which sleep is initiated (Process-C; Borbely, 1982). Process-S models the homeostatic drive that increases sleep need as a function of prior wakefulness, while Process-C models the cortically controlled circadian component that facilitates sleep at night and counteracts sleep during the day. The two-process model has been successfully used to predict sleep duration, sleep depth, and alertness

based on timing and amount of prior wakefulness (Dijk & Lockley, 2002; Duffy, Kronauer, & Czeisler, 1996). It is thought that neuromodulating sleep-promoting substances that accumulate with increased wakefulness, such as adenosine, may underlie the homeostatic drive for sleep (Porkka-Heiskanen et al., 1997). Both the homeostatic drive for sleep and the dissipation of sleep pressure with sleep are greater in infants than adults. By contrast, the circadian influence on sleep is weaker in infants until relevant neural circuits reach maturity around 3 months of life (Rivkees, 2003).

### ***Sleep and Process-S***

The increased homeostatic drive for sleep and the dissipation of sleep pressure with sleep may partly account for the frequent cycling of sleep–wake bouts in infants and need for regular naps. Studies undertaken in older children and adolescents have revealed that sleep deprivation results in higher sleep pressure than that typically reported in adults (Jenni, Achermann, & Carskadon, 2005). The latter findings suggest that the homeostatic drive for sleep is higher in older children, and it is likely but untested that the response to sleep deprivation may be higher again in infancy. Indirect evidence for increased homeostatic drive for sleep in younger children is the high frequency of napping in that age group. A typical infant at 6 months of age may take two to three naps in a day of about 3–4 hours in duration, whereas by 12 months of age the typical infant may take two naps a day of 2–3 hours duration and by 18 months of age one nap during the day of about 1–2 hours duration (Acebo et al., 2005; Iglowstein et al., 2003; Sadeh et al., 1991).

### ***Sleep and Process-C***

The timing of sleep is regulated by the circadian system consisting of a biological clock and input and output pathways (see Crowley, Chapter 17). The “clock” component consists of specialized pacemaker cells located in the suprachiasmatic nucleus (SCN) of the anterior hypothalamus that oscillates with a periodicity slightly longer than 24 hours. This endogenous pacemaker facilitates the synchronization of internal body rhythms so that they are optimally timed with respect to each other. It also facilitates the synchronization of internal systems with the external environment such that an organism is optimally entrained to the day–night cycle. Animal studies suggest that the SCN regulates the timing and duration of sleep and wakefulness by facilitating the initiation and maintenance of

wakefulness at night and by opposing a tendency to sleep that increases during the day (Gillberg, 1997). This “opponent model” of sleep/wake regulation is generally accepted for humans.

The phase of the circadian clock is synchronized to the day–night cycle by external time cues or “zeitgebers” (time givers). Apart from one exception, the circadian clock in humans is refractory to environmental, behavioral, or social cues, such as the timing of meals and rest/activity. The one exception is bright light, with the pacemaker cells differentially sensitive to light exposure at dawn and dusk (Duffy et al., 1996; Lowrey & Takahashi, 2000; Middleton, Arendt, & Stone, 1996). Light falling on the retinas is transduced via the retinohypothalamic tract to the SCN and other nonvisual brain regions (Edgar, Dement, & Fuller, 1993). As well as responsiveness to daylight, the SCN is sensitive to appropriately timed artificial bright light (Boivin, Duffy, Kronauer, & Czeisler, 1996; Czeisler et al., 1986; Duffy et al., 1996), thus making bright light exposure a useful tool for shifting phase in circadian-related sleep disorders (Czeisler et al., 1989). A high light intensity is thought necessary to shift phase, but exposure to normal room light has been reported to shift phase in young adults (Boivin & Czeisler, 1998), raising concerns as to the impact of excessive screen time and extended light exposure on sleep quality in children (Kohyama, 2011). When the phase-shifting response to light is plotted against the timing of light exposure, a characteristic phase-response curve (PRC) is produced (Honma & Honma, 1988; Minors, Waterhouse, & Wirz-Justice, 1991). The PRC to light has been plotted in adults but not in children. In adults who are normally entrained to the day–night cycle, exposure to bright light at dusk delays (i.e., shifts phase to a later time in subsequent cycles), while exposure at dawn advances (i.e., shifts phase to an earlier time in subsequent cycles), the circadian system. Between dusk and dawn is an inflection point separating the delay (dusk) and advance (dawn) portion of the PRC. The timing of the inflection point corresponds to the timing of the core body temperature minimum, approximately 0300–0500 h in entrained adults, and the closer the timing of the light exposure to the minimum, the greater the magnitude of the phase shift (Czeisler et al., 1989).

The effect of the circadian system on sleep–wake can also be studied by examining the response of an individual to the absence of entraining light, such as may occur naturally in the Antarctic winter and experimentally in specially constructed

temporal isolation laboratories. Such studies have revealed that the circadian clock “free runs” with an endogenous rhythmic period ( $\tau$ ) greater than 24 hours (Czeisler et al., 1999). The slight discrepancy between the endogenous  $\tau$  and the 24-hour terrestrial day permits for flexibility in clock timing but necessitates the daily resynchronization of the body clock with the external environment. As  $\tau$  is longer than the length of a terrestrial day, the clock must be continually reset by light to maintain entrainment with the external environment; otherwise, with successive days it will drift progressively later in time or “free run.”

The circadian system is not synchronized with the terrestrial day at birth. Indirect evidence from primate studies suggest that while the circadian system in human neonates may be sensitive to light (Hao & Rivkees, 1999), there is little evidence of a circadian rhythmicity in sleep–wake before 3 months of age (Meier-Koll, Hall, Hellwig, Kott, & Meier-Koll, 1978; Rivkees & Hao, 2000; Stern, Parmelee, Akiyama, Schultz, & Wenner, 1969). However, with neuronal development and exposure to the light–dark cycle, by 1 month of age wakefulness is greater during the day and sleep greater at night, and by at least 3 months of age, hormonal and sleep–wake cycles have begun to consolidate and show a regular 24-hour rhythm with characteristic peaks and troughs (de Weerd & van den Bossche, 2003; McMillen, Kok, Adamson, Deayton, & Nowak, 1991; Nishihara, Horiuchi, Eto, & Uchida, 2002) which remain stable until puberty (Kahn, Dan, Groswasser, Franco, & Sottiaux, 1996). The immaturity of the circadian system may explain the free-running rhythms observed in neonates with wake–activity plots revealing a progressive daily delay in the timing of the main sleep period, sometimes resulting in a reversal of normal sleep–wake timing with infants sleeping through the day but staying awake during the night (Kleitman & Engelmann, 1953; McGraw, Hoffmann, Harker, & Herman, 1999; Shimada et al., 1999; Weissbluth & Weissbluth, 1992). In addition to light–dark exposure, mother–infant interaction may further reinforce daily rhythm patterns and hence circadian rhythmicity (Ferber, Laudon, Kuint, Weller, & Zisapel, 2002).

In adults, sleepiness and hence the propensity for sleep also demonstrates a predictable circadian rhythmicity (Lack & Lushington, 1996; Lavie, 2001). Sleepiness gradually rises in the morning with a minor peak in the early afternoon (12:00–14:00 h), corresponding to the traditional siesta time, followed by a decline to a minimum in the early evening (19:00–22:00 h) (Carskadon

& Dement, 1992; Monk, Buysse, Reynolds, & Kupfer, 1996). Sleepiness then increase throughout the evening to a major peak in the early morning (03:00–05:00 h), typically at the midpoint of the major nocturnal sleep period (Lack & Lushington, 1996). This pattern can be seen in the evolution of nap behavior from birth to early childhood with the establishment of a bimodal sleep pattern during daytime consisting of midafternoon and late evening sleeps, the midafternoon component of which, depending on culture practices, may be maintained until schooling is commenced (Iglowstein et al., 2003).

### *Sleep and Thermoregulation*

The circadian rhythms of body temperature and sleep propensity are closely related. In adults, the two rhythms demonstrate an inverse reciprocal relationship (Lack, Gradisar, Van Someren, Wright, & Lushington, 2008), and of special note is that the magnitude of the pre-sleep increase in peripheral temperature and concomitant nocturnal decline in core body temperature is highly predictive of sleep onset and maintenance (Campbell & Broughton, 1994; Krauchi, Cajochen, Werth, & Wirz-Justice, 2000; Lushington, Dawson, & Lack, 2000; Van Someren, 2000). There is also extensive evidence that stimuli which raise core body temperature inhibit, while those that lower core body temperature facilitate, sleep (Bach et al., 2011; Krauchi & Deboer, 2010).

The relationship between the circadian rhythms of sleep and temperature are not well explored in infants. Nonetheless, available evidence suggests that by 3 months of age the circadian variation in body temperature coincides with the circadian variation in active sleep (Abe & Fukui, 1979; Glotzbach, Edgar, & Ariagno, 1995), with the core body temperature reaching a minimum about the midpoint of the nocturnal sleep period (Lodmore, Petersen, & Wailoo, 1992) and shifting to the last third of night, as in the adult, by 1 year of age (Glotzbach et al., 1995). It is likely that the emergence in infants of a robust core body temperature rhythm may facilitate sleep onset and maintenance. In support of this suggestion, there is preliminary evidence that lower core body temperature minimums are associated with longer mean sleep durations in 4-month-old infants (Lodmore, Petersen, & Wailoo, 1991).

### *Ultradian Rhythms*

A third mechanism is also involved in the control sleep–wake behavior. Together with circadian variation, an ultradian rhythm (i.e., frequency < 24-hour)

is also evident in many sleep parameters. Ultradian rhythms are evident at all levels of biological organization and are thought to provide the timekeeping for intracellular processes (Lloyd, 2008). An ultradian rhythm is evident in the cycling of NREM–REM sleep, which can be recognized from 4 to 6 weeks of age with cycles progressively lengthening from about 60 minutes at 1 month of age to 90 minutes by 5 years of age (Sheldon, 1996), with some groups reporting lengthening by 1 year of age (Hoppenbrouwers, 1992; Pace-Schott & Hobson, 2002). The sleep–wake rhythm itself also demonstrates ultradian variation. At birth, the sleep–wake rhythm cycles with a mean period of between 3–4 hours, but by 3 months of age a clear circadian sleep–wake rhythm has emerged (Meier-Koll et al., 1978; Peirano, Algarin, & Uauy, 2003).

## Conclusion

During infancy and early childhood more time is spent sleeping than on any other activity. Congruent with all the other remarkable physical and behavioral changes that occur as a child grows and develops, sleep also undergoes striking developmental changes that are unparalleled in later adult life. The neurophysiological maturation of sleep is underpinned by structural and functional changes in CNS anatomy and physiology, while a complex interplay between physiological, biological, and psychosocial factors modulate sleep behavior. However, despite the central role that sleep occupies in early life much is still unknown about either the precise mechanisms underlying the development and regulation of sleep or of the role that sleep plays in infant and child development. This high proportion of time infants and young children spend sleeping corresponds to high levels of brain plasticity and sensitive periods of brain development, with mounting evidence suggesting that sleep plays a crucial and active role in the neurocognitive development of children.

Elucidating the role that sleep plays in brain development and, as a corollary, understanding the effects of sleep disturbance on neurocognitive development requires an improved understanding of normal sleep across the entire pediatric age range including the establishment of better normative data. Current normative data are limited largely to pioneering studies performed several decades ago. While these pioneering studies were seminal in establishing pediatric sleep medicine as a field in its own right, they have the usual shortcomings of early investigations including small biased samples that were restricted to certain age

groups and a lack of standardization in methodology and data analysis. The advent of digital polysomnography (PSG), which has the ability to capture significantly more physiological data with relative ease, will extend the groundbreaking studies performed in the latter part of the last century and can provide a more comprehensive overview of normal sleep development.

## Future Directions

There are many unanswered questions in relation to the development and regulation of sleep during infancy and childhood. Addressing the following questions may provide insights into the functions of sleep and may assist in identifying children who require clinical or behavioral interventions for sleep disorders:

- How does sleep architecture change with age? There is a need for longitudinal PSG data derived from normal, healthy children sampled at regular developmental periods.
- What is the relationship between sleep and brain development? That is, how does PSG data correlate with other measures of development such as motor skill, language, memory, social development, etc.?
- What are the genetic and epigenetic underpinning of sleep development?
- How do biopsychosocial factors influence sleep development?
- Does sleep intervention improve neurocognitive outcomes? For example, in children with neurodevelopmental disorders such as Down's or Asperger's Syndrome, which are often associated with significant sleep disturbance.

## Notes

1. The traditional sleep stage terminology of Rechtschaffen and Kales (1968) is used throughout this chapter. See Carskadon and Dement (2011), with whom we concur, for a discussion on sleep nomenclature.

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