Sleep Problems and Early Developmental Delay: Implications for Early Intervention Programs

Karen Bonuck and Roy Grant

Abstract
Sleep disorders negatively impact behavior, cognition, and growth—the same areas targeted by early intervention. Conversely, developmental delays and disabilities may themselves precipitate sleep disorders. Young children with developmental delays experience sleep disorders at a higher rate than do typically developing children; the most common types are difficulties initiating or maintaining sleep and sleep disordered breathing. To date, attention has been focused on sleep problems in children with specific conditions (e.g., autism, genetic syndromes, prematurity, and seizure disorder). The authors review evidence of sleep problems’ broader impact across the range of children screened for early intervention. Eligibility evaluations for early intervention address the five developmental domains: adaptive, motor, cognitive, communication, and socioemotional. Disordered sleep may be symptomatic of socioemotional and adaptive problems. Assessing sleep problems within the evaluation may help establish eligibility for early intervention services and would maximize developmental potential by ensuring timely identification, referral, and treatment.

Key Words: breathing problems; early intervention; eligibility for early intervention; Individuals with Disabilities Education Act, Part C; sleep disorders

Pediatric sleep disorders impact a child’s behavioral, cognitive, and physical development (Bonuck, Parikh, & Bassila, 2006; Montgomery & Dunne, 2007; Owens, 2009; Schechter, 2002), with long term consequences (Jan et al., 2010). As pediatric sleep specialist Owens stated that nearly every neuro-behavioral and neuro-cognitive condition of childhood is potentially linked to sleep problems, and, furthermore, the link is likely to be a bidirectional one (Owens & Palermo, 2008). The most frequently occurring pediatric sleep disorders among all children are (a) difficulties initiating or maintaining sleep and (b) sleep disordered breathing. Difficulties initiating or maintaining sleep affect 40% to 75% of children with developmental delays—two to three times the rate among typically developing children (Cotton & Richdale, 2006; Krakowiak, Goodlin-Jones, Hertz-Picciotto, Croen, & Hansen, 2008; Richdale & Schreck, 2009). Sleep disordered breathing rates in children with developmental delays are unknown but are high in certain congenital conditions (e.g., 50% in Down syndrome) associated with developmental delay (Ng et al., 2006). This higher prevalence of sleep problems in children with developmental disabilities and delays may be related to intrinsic abnormalities in sleep regulation and circadian rhythms, medications used to treat associated symptoms, cognitive delays, sensory deficits, or increased parental stress (Owens & Palermo, 2008).

Sleep Problems: Definition and Description
The term sleep problems as used here refers to both difficulties initiating or maintaining sleep and sleep disordered breathing. Regarding difficulties initiating or maintaining sleep, the International Classification of Sleep Disorders (American Academy of Sleep Medicine, 2005) identifies several diagnoses, including behavioral insomnias of childhood, circadian rhythm disorders, and inadequate sleep hygiene diagnosis. Difficulties initiating or maintaining sleep are more prevalent in children with atypical development and are associated with inadequate and fragmented sleep, which can have serious behavioral and cognitive consequences and contribute to family stress.
The etiology of difficulties with initiating or maintaining sleep in infants and toddlers are primarily behavioral, such as poor parental limit setting, inconsistent bedtime routines, or dependence upon excessive sensory stimuli. Social and environmental factors, such as crowding, noise, and shared sleeping arrangements also contribute to difficulties initiating or maintaining sleep. In fact, sleep ecology (e.g., where child sleeps) and parental behaviors (e.g., night feeding upon waking) explains much of the variance in young children’s sleep patterns (Sadeh, Mindell, Luedtke, & Wiegand, 2009). In addition, medical conditions (e.g., colic, middle ear disease, gastroesophageal reflux, or fetal alcohol spectrum disorders) may interfere with initiation or maintenance of sleep.

In contrast, sleep disordered breathing is a manifestation of anatomical processes (e.g., airway narrowing), local tissue compliance, and neurophysiological contributions. This condition, which spans a continuum from primary snoring to obstructive sleep apnea, adversely affects behavior, cognition, and growth (Schechter, 2002). Approximately 4% to 11% of children in the general population have sleep disordered breathing per varying constellations of parent-reported symptoms on a questionnaire (Lumeng & Chervin, 2008). Adenotonsillar hypertrophy is the primary cause of sleep disordered breathing, and tonsillectomy and/or adenoidectomy is curative in up to 80% of cases (Chan, Edman, & Koltai, 2004; G. Rosen, 2003). Sleep disordered breathing peaks between the ages of 2 and 6 years, given the relative hypertrophy found at this time (Halbower & Marcus, 2003), but it is also found in younger children (American Academy of Pediatrics, 2002).

**How Do Sleep Problems Affect Young Children?**

Despite differing etiologies, sleep disordered breathing and difficulties initiating or maintaining sleep both cause disrupted and/or inefficient sleep, resulting in mood changes, excessive daytime sleepiness, fatigue and somatic complaints, and cognitive impairment and poor school performance related to excessive sleepiness (American Academy of Sleep Medicine, 2005). Cognitive deficits linked to sleep disordered breathing are believed to occur via intermittent hypoxia (reduced oxygen), repeated arousal, or hypercarbia (excess CO₂), resulting in damage to the prefrontal cortex (Beebe & Gozal, 2002). In a review of 61 studies, Beebe (2006) concluded that independent of potential confounders, sleep disordered breathing increases a child’s risk of school failure, attention deficit disorder, behavior problems, and difficulty with emotional regulation. Even when symptoms resolve, a prior history of sleep disordered breathing (e.g., snoring) increases a child’s risk of developmental problems (Beebe, 2006).

The high degree of brain plasticity in the first 3 years of life and effects of early experience upon later developmental, emotional, and behavioral outcomes are the rationale for early intervention programs. Physical, sensory, and motor stimulation during this period has the greatest impact upon promoting the interaction among neurons that develop connections among the sense organs and the brain (axonal growth and targeting) (Bonnier, 2008). Sleep disturbances cause widespread deterioration of neuronal functions, memory and learning, gene expression, and neurogenesis, which, in turn, cause decline in cognition, behavior, and health. Although sleep deprivation may occur at any age, adverse consequences are more likely to occur and persist in younger children, whose brains are rapidly developing, compared with adults (Jan et al., 2010). The opportunity for neurological insults to occur is also related to the amount of sleep young children require (e.g., more than half of an infant’s first year is spent in a sleep state. Infants and toddlers with neurodevelopmental deficits have different sleep patterns than do typically developing young children. Furthermore, early sleep patterns have been associated with temperamental differences. Sleep disrupted by frequent awakening is associated with lower sensory thresholds and temperaments subjectively considered to be “difficult” (Ednick et al., 2009).

Population-based studies strongly support an association between parent-reported sleep problems and behavioral and cognitive measures. In a Canadian longitudinal study of 2- to 3-year-olds (approximately 9,000 children during three waves of investigation), Reid, Hong, and Wade (2009) found that difficulties initiating or maintaining sleep-type problems were as strong a correlate of internalizing (i.e., anxiety) and externalizing (i.e., hyperactivity/aggression) problems as well-established risk factors, such as maternal depression. Among Australian 4- to 5-year-olds (n = 4,983), the prevalence of mild and moderate/severe sleep problems was 14% and 20%, respectively, with an approximate doubling of effect size for quality of life.
and conduct outcomes, from no to mild and from mild to moderate/severe sleep problems. Children with moderate/severe sleep problems were 12 times as likely to be diagnosed with attention deficit hyperactivity disorder (ADHD)/attention deficit disorder (ADD) compared with children with no sleep problems (Hiscock, Canterford, Ukoumunne, & Wake, 2007).

**What Is the Bidirectional Link Between Sleep Problems and Developmental Disorders?**

It is difficult to disentangle mechanisms and directionality, given the heterogeneity of children in early intervention and special needs programs. Further, absent collection of behavioral and cognitive data before report of sleep problems, a temporal association cannot be confirmed. Still, it is instructive to briefly highlight the reciprocal relationship between sleep problems and selected developmental disorders.

**Autism Spectrum Disorders**

Difficulties initiating or maintaining sleep are particularly well known as comorbidities of children with autism spectrum disorders (Myers & Johnson, 2007; O’Connell & Vannan, 2008), who experience difficulties initiating or maintaining sleep at a high rate and severity (Cotton & Richdale, 2006). Children with autism spectrum disorders have disruptions in GABA and melatonin, both which regulate sleep. Conversely, their psychiatric symptoms may interfere with sleep initiation (Johnson & Malow, 2008). Thus, autism spectrum disorder, in particular, is an example of the bidirectional association noted earlier.

**Growth Delays**

Evidence supports a causal relationship between sleep disordered breathing and impaired growth in children (American Academy of Pediatrics, 2002). Impaired growth hormone secretion in children with sleep disordered breathing may result from interruptions in slow-wave sleep, when a large proportion of growth hormone is secreted (Bonuck et al., 2006). In a recent systematic review and meta-analysis, Bonuck, Freeman, and Henderson (2009) found that standardized height and weight and insulin-like growth factor (IGF)-1 and insulin-like growth factor binding protein-3 (IGFBP-3) increase significantly after adenotonsillectomy.

**Congenital Conditions**

Congenital syndromes and malformations (e.g., Pierre Robin sequence, Treacher Collins syndrome, achondroplasia) involve anatomical narrowing of the upper airways, predisposing to obstructive sleep apnea syndrome. For example, it is estimated that 50% of children with Down syndrome have sleep apnea (Ng et al., 2006). In neuromuscular disorders (e.g., muscular dystrophy), decreased upper airway muscle tone is the dominant factor in obstructive sleep apnea syndrome (Greenfeld, Tauman, DeRowe, & Sivan, 2003), and most such children develop sleep disordered breathing (Panitch, 2009). For example, cleft palate, a relatively common congenital condition, is associated with increased sleep disordered breathing risk. Among children with cleft palate referred for polysomnography (overnight hospital-based studies of sleep respiration), 87% were positive for sleep disordered breathing and 28% had severe sleep disordered breathing (MacLean, Fitzsimons, Hayward, Walters, & Fitzgerald., 2008).

**Speech and Language**

Articulation difficulties linked to sleep disordered breathing can occur due to the effect of adenotonsillar hypertrophy, the primary cause of pediatric sleep disordered breathing, upon oral motor function. Compared with control children, children undergoing tonsillectomy for sleep disordered breathing were more likely to have phonological impairment presurgery (62.7% vs. 34%, respectively; p < .001), and to have more severe impairment (p < .001). Phonology improved postsurgery (Lundeborg, McAllister, Samuelsson, Ericsson, & Hultcrants, 2009). Prefrontal cortex damage as a result of sleep disordered breathing, in turn, is associated with impaired verbal fluency and retrieval and dissociation between language behaviors and ability to use verbal cues to direct or organize behavior (Andreou & Agapitou, 2007). Prefrontal cortex activity is reduced in children with autism spectrum disorder (Wang, Lee, Sigman, & Dapretto, 2007) and pervasive developmental disorders (Kuwabara et al., 2006).

Speech and language issues may precipitate behavior problems. Expressive language delays are linked to socioemotional problems, whereas receptive language delays are more common in children with pervasive developmental disorders (Tervo, 2007).
(NEILS), poor communication skills reported by parents were associated with specific behavioral problems (e.g., difficult behavior, lack of persistence, withdrawn, and distractible). It is unclear whether communication problems cause these behavioral difficulties, vice versa, or both are caused by another underlying mechanism (Scarborough, Hebbeler, Spiker, & Simeonsson, 2007).

**Socioemotional**

The antecedents of ADHD are seen in preschoolers (Daley, Jones, Hutchings, & Thompson, 2009) but may be difficult to diagnose in children under 3 (Mahone, 2005), notwithstanding a small retrospective study in which Magallon, Crespo-Eguizal, Ecy, Poch-Olive, and Narbona (2009) found differences in behavioral profiles in toddlers by ADHD status at school-age. Adenotonsillectomy performed for sleep disordered breathing yielded improvements in behavior, cognition, or quality of life in at least one behavioral (e.g., hyperactivity, aggressiveness) or neurocognitive outcome (e.g., memory, attention, or school performance) among the 25 studies evaluated. Chervin, Ruzicka, Archbold, and Dillon (2005) estimated that 15% to 25% of children with hyperactivity could experience improved behavior if underlying sleep disordered breathing were diagnosed and treated.

How Might Assessment of Sleep Disorders Affect Early Intervention and Special Education Program Eligibility?

The Early Intervention Program, Part C, of the U.S. Individuals with Disabilities Education Act (IDEA) was designed to evaluate and serve 0- to 36-month-olds. Eligibility is based upon documentation of a diagnosed condition with a high probability of developmental delay (e.g., Down syndrome, cerebral palsy) or identification of developmental delay(s) per a multidisciplinary evaluation. States establish their own eligibility thresholds, resulting in considerable variation in eligibility nationwide. The federal goal for participation (i.e., eligible and having a service plan) is 2% of the age-eligible population (Shackelford, 2006).

Data suggest that the early intervention participation rates are low, relative to children who need services. In 2007, early intervention served approximately 315,000 children, 2.5% of 0- to 36-month-olds (IDEA Data Table 8-1, 2007) Yet, results of nationally representative survey data from direct assessment, caregiver interviews, and birth data suggest that the prevalence of developmental delays in children aged 0 to 2 years is 13%, with just 10% actually receiving any early intervention services (Rosenberg, Zhang, & Robinson, 2008). The preschool special needs program served 5.74% of 3- to 5-year-olds (IDEA Data: Part B IDEA 618 Tables, 2007).

From preschool to school-age, there is a marked increase in conditions frequently associated with difficulties initiating or maintaining sleep and sleep disordered breathing, namely, learning disability, developmental delay, and autism (see Table 1). Notably, 8.6% of parents reported that their school-age child was diagnosed with ADD/ADHD (Merikangas et al., 2010) and 11.5% reported a learning disability (Nolin, Montaquila, Nicchitta, Hagedorn, & Chapman, 2004). Combined, just over one million children aged 6 to 11 years received IDEA school age special education services for learning disability, autism, and developmental delay. Though ADD/ADHD is generally not a qualifying diagnosis for special education, children with this disorder are at increased risk of learning disabilities (Barry, Clarke, McCarthy, & Selikowitz, 2009; Biederman et al., 2004; Capana, Minden, Chen, Schacher, & Ickowicz, 2008). Likewise, anxiety, although not a qualifying diagnosis for early intervention either, is elevated among children with global developmental disorders (de Bruin, Ferdinand, Meester, de Nijs, & Verheij, 2007) and ADD/ADHD (Rommelse et al., 2009). Further, there is significant comorbidity among these symptoms. In a large national survey of children's health, Larson, Russ, Kahn, and Halfon (2011) found that children with ADHD compared with their peers had increased adjusted risks of comorbid learning disability (8-fold), anxiety (8-fold), and low social competence (3-fold).

Turning to the early intervention program (see Table 2), we find it striking that among the five domains evaluated for eligibility, the preponderance of identified delay is in communication; whereas adaptive and socioemotional problems are sparsely reported. These data on the developmental characteristics of children receiving early intervention services are from the NEILS noted earlier, which is a nationally representative sample of more than 3,000 children and families recruited from 1997–1998 (Hebbeler et al., 2001). Although these
data are based upon parent and provider report, they are probably more reflective of a child's functional characteristics than is a medical diagnosis. In contrast, relatively few children are identified by their providers as having socioemotional problems.

Sleep disorders could bear on early intervention eligibility in several ways. First, and most concretely, sleep disordered breathing, and perhaps difficulties initiating or maintaining sleep as well, might qualify as a medical risk in early intervention. For example, New York State’s regulations define medical/biological risk as early developmental and health events suggestive of medical needs or biological insults to the developing central nervous system which, either singly or collectively, increase the probability of later disability” (New York State Final Regulations, 2005, state: 69-4.1 S [ab]).

Second, sleep problems, now part of the adaptive domain, could be incorporated into eligibility determinations if appropriately valid measures existed. The New York State Department of Health (2005) operates one of the largest early intervention programs in the country. Its guidance on eligibility determination defines the adaptive domain as follows:

Adaptive development refers to the development of behaviors and self-help skills that assist children in coping with the natural and social demands of the environment, including sleeping, feeding, mobility, toileting, dressing, and higher-level social interactions. A child who is experiencing delays in adaptive development has difficulty in learning and acquiring these behaviors and skills. Delays in adaptive development may be associated with delays or impairments in other areas of development, including fine and gross motor skills, oral-motor functioning, cognitive development, communication development, and social-emotional development. (p. 33)

Third, appropriately identified sleep problems could bear on early intervention eligibility in that delay in this functional aspect of the adaptive domain affects socioemotional status (temperament for infants, attention and behavior for toddlers) and parent–child relationships. Technically, both the adaptive and socioemotional domains are assessed during eligibility determinations, although the NEILS data from Table 2 suggest that this is neither routine nor thorough.

What Are the Barriers to Assessing Sleep Problems in Early Intervention?

The low prevalence of children in the NEILS identified as having socioemotional problems may partially reflect the lack of a requirement for a socioemotional development specialist to participate in early intervention eligibility evaluations. Further, states offer only a limited range of services for socioemotional problems (Cooper & Vick, 2009). That is, sleep problems may be underidentified because they do not correspond to missed developmental milestones or a quantified developmental delay.

Whether and how sleep problems contribute to adaptive and socioemotional delay could, theoretically, be ascertained by informed clinical opinion rather than a standardized instrument (Shakelford, 2002). However, this is problematic in practice for a variety of reasons. Early intervention professionals generally consider for sleep issues as background; this information is unlikely to inform eligibility or service determination. Second, instruments used in early intervention eligibility are validated for outcomes other than for sleep disorders. There has been no systematic review of the sleep items in

Table 1

<table>
<thead>
<tr>
<th>Diagnostic category</th>
<th>Birth–5 years</th>
<th>6–17 years</th>
</tr>
</thead>
<tbody>
<tr>
<td>Learning disability</td>
<td>2.9</td>
<td>11.5</td>
</tr>
<tr>
<td>Developmental delay or physical impairment</td>
<td>3.2</td>
<td>3.6</td>
</tr>
<tr>
<td>Speech problems</td>
<td>5.8</td>
<td>3.0</td>
</tr>
<tr>
<td>Autism</td>
<td>0.2</td>
<td>0.6</td>
</tr>
<tr>
<td>ADD/ADHD</td>
<td>1.0</td>
<td>8.6</td>
</tr>
<tr>
<td>Depression/anxiety</td>
<td>0.6</td>
<td>5.4</td>
</tr>
<tr>
<td>Behavior/conduct</td>
<td>2.4</td>
<td>6.3</td>
</tr>
</tbody>
</table>

Note. N = 3,000. Data from Blanchard, Gurka, and Blackman (2006).
ADD/ADHD = attention deficit disorder/attention deficit hyperactivity disorder.
developmental screening and assessment instruments to know whether they have been validated against objective measures (i.e., polysomnography) or, as is recommended by the American Academy of Sleep Medicine (2005), clinical interview with a sleep medicine specialist. In fact, in an October 2010 review, Spruyt and Gozal found that of 183 pediatric sleep questionnaires identified, only 57 reported psychometric properties and just followed all of the fundamental operational principles of instrument development. Further, aside from the one exception (Goodlin-Jones, Sitnick, Tang, Liu, & Anders, 2008), sleep questionnaires have not been validated with children who have developmental disability and delay. Finally, and of critical importance, is our preliminary assessment that none of the tools used in early intervention assess sleep disordered breathing.

Although not one of the standard instruments used by early intervention evaluators assesses sleep disordered breathing, about half contain at least one behavioral sleep problem (i.e., difficulties initiating or maintaining sleep). According to a systematic review (Bonuck, Hyden, Ury, & Briggs, 2011), the most extensive are the Infant–Toddler Social and Emotional Assessment version, with eight items pertaining to sleep ecology, night wakings, and night terrors (Carter, 2001); the Child Behavior Checklist for Ages 1.5–5 (Achenbach & Ruffle, 2000), with seven sleep-related items; and the Temperament and Atypical Behavior Scale, with five items (Bagnato, Neisworth, Salvia, & Hunt, 1999). Still, these groupings of sleep have not been validated against either objective sleep measures or a sleep professional’s clinical assessment. Conversely, of the sleep questionnaires validated for difficulties initiating or maintaining sleep in the general population (Bruni et al., 1996; Owens, 2001; Sadeh, 2004), none have been validated in a heterogeneous sample of young children with developmental delay representative of early intervention or preschool special need program participants.

If early interventionists are able to meaningfully assess the presence of a sleep disorder in the context of adaptive and/or socioemotional development, this might prove decisive for determining eligibility. This is because, despite variation among state early intervention eligibility thresholds, one commonality is a higher threshold with a delay in

Table 2
Early Intervention Providers’ Disability Descriptors for Eligibility by Age Group (in %)

<table>
<thead>
<tr>
<th>Descriptor</th>
<th>0–12 months</th>
<th>12–24 months</th>
<th>25–36 months</th>
</tr>
</thead>
<tbody>
<tr>
<td>Delayed development in multiple domains</td>
<td>11.0</td>
<td>14.8</td>
<td>11.6</td>
</tr>
<tr>
<td>Sensory system impairment</td>
<td>3.6</td>
<td>4.0</td>
<td>2.3</td>
</tr>
<tr>
<td>Motor delay</td>
<td>20.4</td>
<td>21.9</td>
<td>10.6</td>
</tr>
<tr>
<td>Physiologic/neurologic system impairment</td>
<td>3.9</td>
<td>1.9</td>
<td>0.7</td>
</tr>
<tr>
<td>Cognitive delay</td>
<td>2.9</td>
<td>7.3</td>
<td>11.9</td>
</tr>
<tr>
<td>Socioemotional problem</td>
<td>1.9</td>
<td>3.3</td>
<td>6.2</td>
</tr>
<tr>
<td>Communication delaya</td>
<td>5.0</td>
<td>48.5</td>
<td>75.4</td>
</tr>
<tr>
<td>Adaptive delayb</td>
<td>1.7</td>
<td>2.5</td>
<td>3.5</td>
</tr>
<tr>
<td>Congenital disorders</td>
<td>18.0</td>
<td>4.9</td>
<td>2.0</td>
</tr>
<tr>
<td>Prenatal/perinatalc</td>
<td>40.0</td>
<td>9.4</td>
<td>3.1</td>
</tr>
<tr>
<td>Illness or chronic disease</td>
<td>2.8</td>
<td>1.2</td>
<td>1.4</td>
</tr>
<tr>
<td>Musculoskeletal</td>
<td>3.8</td>
<td>1.4</td>
<td>0.4</td>
</tr>
<tr>
<td>Central nervous system</td>
<td>3.8</td>
<td>8.6</td>
<td>2.0</td>
</tr>
<tr>
<td>Medical condition, unspecified</td>
<td>2.7</td>
<td>1.1</td>
<td>0.2</td>
</tr>
<tr>
<td>Socioenvironmental risk factors</td>
<td>4.4</td>
<td>4.6</td>
<td>2.3</td>
</tr>
</tbody>
</table>

Note. Data from Hebbeler et al. (2001).

aFor example, speech–language.
bSelf-help skills.
cFor example, low birthweight, prenatal exposure.
just one domain. The most common standards are 33% or greater delay in one domain or 25% or greater delay in two or more domains. Thus, hypothetically, a child with a 25% speech delay and a 25% sleep-associated adaptive delay would qualify for early intervention, but he or she would not have qualified based on their speech delay alone.

**What Medical or Socioenvironmental Risk Factors Place Children in Early Intervention at Greater Risk of Sleep Problems?**

Sleep disorder assessments are indicated in the early intervention program not only because of the shared symptom profile between sleep problems and developmental delays in general, but also because specific early intervention populations experience increased rates of sleep problems. Both low birthweight and prematurity, which in most states render an infant eligible for early intervention, are closely associated with adverse neurocognitive and behavioral outcomes (Aarnoudse-Moens, Weisglas-Kuperus, van Goudoever, & Oosterlaan, 2009). Prematurity and low birthweight increase the risk of sleep disordered breathing by two- (Paavonen et al., 2007) and three-fold (C. Rosen et al., 2003), respectively. Close to 32% of children in the NEILS weighed less than 2500 g (Scarborough et al., 2004). Further, early intervention participation rates are inversely correlated with gestational age. Although 16% of term infants participated in early intervention, 82% of very preterm (24 to 31 weeks) and three-fold (32 to 36 weeks) infants do so (Clements, Barfield, Ayadi, & Wilber, 2007).

Children with socioenvironmental risk factors placing them at risk for developmental delays, correspondingly, are at increased risk of sleep disorders. Nearly half of children under 3 years of age in foster care (47%) have developmental delays commensurate with early intervention eligibility (Rosenberg & Smith, 2008). Since 2003, states have been required to formulate policies for referring all infants and toddlers with substantiated child maltreatment cases to their local early intervention program. This legislation led to an average 44% increase in children in foster care receiving early intervention services (Derrington & Lippitt, 2008). Among children 3 and under in foster care (mean age = 23 months), 13% met the Achenbach Child Behavior Checklist criteria for sleep problems, and 25% showed symptoms consistent with an externalizing behavior disorder (Reams, 1999). Compared with control children, 3- to 7-year olds in foster care had greater difficulties initiating sleep per actigraphic measures. This sleep problem may reflect stress-related hypervigilence at bedtime and/or earlier difficulties with state regulation and wake–sleep transitions (Tininenko, Fisher, Bruce, & Pears, 2010).

**Implications for Prevention**

Preventive interventions to ameliorate sleep problems in young children may avert later developmental problems and morbidity associated with both difficulties initiating or maintaining sleep and sleep disordered breathing. Yet, state early intervention programs do not formally address sleep problems. One known exception is New Jersey’s early intervention program, which identifies “sleep problems, including snoring, frequent waking” as among the frequently identified behaviors/symptoms that may need referral to an otolaryngologist (New Jersey Early Intervention System, Birth to Three, 2007). Developers of early intervention programs should begin to assess sleep disorders, given the growing evidence-base of data supporting their effects upon later development. Early intervention providers and service coordinators, as well as preschool special education teams, can and should elicit information on sleep problems as part of a multidisciplinary evaluation. (Given the obstacles cited earlier—few relevant items in current assessment instruments and lack of validation of sleep problem questionnaires in children with developmental delays—additional research in this area is needed.) Early intervention service coordinators could then initiate referrals to the appropriate medical or behavioral health care providers, to assist with parent-based education or medical treatment options, respectively. There has, in fact, been a call for early intervention to increase access to and linkages with supportive services (Turnbull et al., 2007). Doing so would also further the program’s mission of supporting families, given the considerable negative effect that sleep problems of children with developmental disabilities and delays have upon family quality of life (Gallagher, Phillips, & Carrol, 2010).

Preventive interventions for young children ameliorate sleep problems and may avert associated morbidity. When respiratory-related sleep disorders
are diagnosed, treatment such as adenotonsillectomy for obstructive sleep apnea is often indicated. Regarding difficulties initiating or maintaining sleep, early intervention providers can reinforce advice parents may have received from their child’s physician about good sleep hygiene practices (i.e., consistent comforting bedtime routines, allowing the child to fall asleep on his or her own), or offer this information if it has not been introduced to the parent. Cognitive–behavioral interventions for difficulties initiating or maintaining sleep include a range of extinction programs (e.g., unmodified, graduated, with parent presence) or a bedtime pass program for children older than 3 years (Galland & Mitchell, 2010). A review of 52 treatment studies showed that in 94%, behavioral treatments were efficacious, with 80% of children retaining improvements 3 to 6 months later. Compared to drug treatments, behavioral interventions are more effective in the short- and long-term (Keenan, Wild, McArthur, & Espi, 2007; Ramchandani, Wiggs, Webb, & Stores, 2000) and address behavioral aspects of the problem (Wiggs, 2009).

Timely access to medical or behavioral services can mitigate the effects of sleep problems, thereby maximizing developmental potential and reducing stress within the family. In this paper we have shown that there is considerable shared comorbidity between sleep problems and both the conditions that qualify children for early intervention and special needs services as well as the at-risk populations in which they occur. Although ideally sleep disorders would be identified in the child’s medical home, in practice sleep problems are often unrecognized and untreated in primary care (Cherin, Archbold, Panahi, & Pituch, 2001; Mindell & Owens, 2003; Owens, 2001). For this reason, the screening and evaluation processes for IDEA programs could be a key portal for identifying sleep problems.

References


Individuals with Disabilities and Education Act (IDEA). Data: Part B IDEA 618 Tables. Available at http://www.idea-data.org/IDEA618DataTables.asp


Received 1/12/11, first decision 3/4/11, accepted 3/16/11.

Editor-in-charge: Michael Wehmeyer

Authors:
Karen Bonuck, PhD (e-mail: karen.bonuck@einstein.yu.edu), Professor, Family and Social Medicine, Albert Einstein College of Medicine, Jack and Pearl Resnick Campus, 1300 Morris Park Ave., Mazer Building., Rm. 418, Bronx, NY 10461.