Behavioural Sleep Disorders across the Developmental Age Span: An Overview of Causes, Consequences and Treatment Modalities

Sarah Lee Blunden
Appleton Institute, Central Queensland University, Adelaide, Australia
Email: s.blunden@cqu.edu.au

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Behavioural sleep problems, that is, sleep problems that do not have a physiological aetiology, but rather a behavioural or psychological aetiology, are reported in between 20% - 40% of children and adolescents. These sleep disorders are categorised as Behavioural Insomnia of Childhood (BIC) in the International Classification of Sleep Disorders. BIC can result in short sleep duration and poor quality sleep and can have wide ranging effects on mental and physical health, cognitive and social functioning and development in infants, pre-schoolers, school aged children and adolescents. Each age group have a particular set of behaviourally based sleep disorders. This paper presents a broad overview of BIC and covers essential information about these sleep disorders, their aetiologies, effects on development and non medical treatment modalities.

Keywords: Behavioural Sleep Disorders; Behavioural Insomnia; Behavioural Sleep Treatments; Delayed Phase

Introduction

There is an abundant literature on the consequences of short sleep duration and poor quality sleep on a wide range of negative outcomes in mental and physical health, cognitive and social functioning in young people (Fallone et al., 2001) Seque-lae include motor skill deficits (Laureys, 2002); greater emotional liability, increased impulsivity, aggression and hyperactivity (Pearl et al., 2002; Sadeh et al., 2002; Blunden, 2010); increased potential for alcohol and drug abuse in adulthood (Wong et al., 2004); and suicide risk in adulthood (Wojnar, 2009). When left untreated, sleep problems developing during the primary school years can become a persistent problem and equate to poorer health in general (Moore et al., 2002) and sleep difficulties as adults (Chaput, 2008; Buckhault, 2011). Poor sleep in these cases perpetuate poor overall physical health in adults, perhaps mediated by compromised immune function (Vgontzas, 2008), and is more likely among those of lower socioeconomic position (Sekine, 2006). Importantly, short sleep duration is associated with increased risk of overweight and obesity (Carter 2011) due to changes in appetite regulation.

Sleep disorders in the early years can be largely divided into those that have a physiological aetiology (such as sleep related respiratory disorders, parasomnias and periodic limb movement disorder) and those without. The most abundant information regarding the effects of sleep disturbance on daytime function, particularly among children, comes from studies of sleep disordered breathing (SDB). These sleep disorders are prevalent in between 2% - 15% of children (Blunden et al., 2003).

However, the most common sleep problems in the early years are overwhelmingly those of a non-physiological aetiology. Most children will experience some degree of transitory and developmentally normal behavioural sleep problem (Stores, 1999; Sadeh et al., 2000), but some children will develop more chronic and persistent sleep problems. In fact, the large majority of sleep problems in young children do not have a physiological aetiology but are behaviourally based (Hiscock et al. 2007).

Defining Behavioural Sleep Disorders

Behavioural sleep disorders in children are described as Behavioural Insomnia of Childhood (BIC) by American Academy of Sleep Medicine in (AASM 2005), in their International Classification of Sleep Disorders—Second Edition (ICSD-2; 2005; see Table 1). The hallmark feature of BIC is difficulty falling asleep or staying asleep.

The ICSD has documented BIC as dysomnias. Categorisation of sleep disorders in the ICSD follows the model established by the American Psychiatric Association’s Diagnostic and Statistical Manual, (DSMIII-R), to assist in the classification, diagnosis and treatment of sleep problems. The dysomnias described in the second edition of the ICSD are the disorders that produce either difficulty initiating or maintaining sleep or excessive sleepiness and have been further divided into three groups of disorders: intrinsic sleep disorders, extrinsic sleep disorders, and circadian rhythm sleep disorders. The behaviour sleep disorders of interest in this paper are categorised in the ICSD as extrinsic sleep disorders and include those disorders that originate or develop from causes outside of the body. External factors are integral in producing these sleep disorders, and removal of the external factors leads to resolution of the sleep disorder (AASM, 2005). This is not to say that internal factors are not important in the development or maintenance of these sleep disorders. However, the internal factors are unlikely to themselves to produce the sleep disorder without presence of...
an external factor. 

The extrinsic sleep disorders that are considered behavioural for inclusion in this chapter are summarised in Table 2.

Prevalence

Behavioural sleep disorders are generally a diagnosis of observation or description by third party report and complemented by a complete sleep history (Mindell et al., 1999). Epidemiological estimates of these reported sleep problems in children, as classified by the ICSD, are largely based on age specific cross sectional data and are rarely separated into classifications as listed above. In general however, behavioural sleep problems are common with 20% - 30% of infants and toddlers, (Morgenthaler 2006), in 30% - 40% of pre-school children (Blader et al., 1997; Owens et al., 1998; Blunden et al., 2005) between 10% - 45% of pre-pubescent children (Wolfson et al., 2007) and 11% - 30% of adolescents (Wolfson, 2003; Carskadon et al., 2004). Despite the wide prevalence range, it is clear that behavioural sleep disorders are common in the paediatric life span. Whether these sleep problems become chronic or significantly problematic is less clear.

Aetiology of Behavioural Sleep Disorders across the Ages

Evidence would suggest that while certain behavioural sleep disorders are more common in certain age groups (such as a high prevalence of Sleep-onset Association Disorder in young children), all of the behavioural sleep disorders presented above occur across the paediatric spectrum.

Infants and Pre-Schoolers

In infants and pre-schoolers, the vast majority of sleep problems fall under the classification of either Sleep Association Type or Limit-Setting Type or a combination. In Australia, the sheer prevalence of infant sleep problems poses a substantial population and health burden in the first year of life (Armstrong, 1994). In Sleep Association Type, the child is unable to initiate sleep without the presence of an object or person (e.g., bottle, rocking, feeding, parental presence), thus creating a dependency at sleep onset, during day, evening and overnight sleep initiation or re-initiation. In general, night wakeings, viewed as problematic by caregivers because of a child’s inability to ‘self soothe’ (that is, the ability to calm themselves enough to fall asleep alone), fall within the diagnostic category of Behavioural Insomnia of Childhood (BIC), Sleep Onset Association Type. In BIC Limit Setting Type, parents demonstrate difficulties in adequately enforcing bedtime limits resulting often in delayed bedtime and subsequent reduced sleep duration. This is often associated with difficulties adjusting to parenting and can be associated with Adjustment Sleep Disorders (AASM, 2005).

It has been suggested that bedtime resistance and night wakeings in childhood are a “regression” in behaviours associated with the neurodevelopmental processes of sleep consolidation and sleep regulation that evolve over the first years of life (Morgenthaler, 2006). However, this ‘regression’ must take into account psychological factors that may be contributing to sleep problems, notably a potentially normal developmental resistance or adaptation to sleeping alone by children as they become old enough to express their preferences (McKenna, 2000).

Other contributors to these bedtime behavioural problems in young children include temperament—children with more “needy” temperaments require more active parental participation (Weissbluth, 1984; Owens-Stivel et al., 1997)—and culture—cultures with minimal nighttime separation report less overnight wakeings and sleep disruption for caregivers (McKenna 2000). Clearly, these factors have a bi-directional relationship with the caregiver and the child’s environmental and behavioural variables (Morgenthaler, 2006). Maladjustment to the disruption to caregiver sleep can lead to significant caregiver stress, particularly maternal depression and anxiety, which have been consistently associated with infant sleep problems, even after controlling for known depression risk factors (Hiscock et al., 2007). This is evidenced in the common occurrence of Adjustment Sleep Disorder experienced by parents of young children, when parents adjust to the changes in sleep and daytime routines that are evident with the birth of a new baby.

School Aged Children

Although children generally achieve sleep consolidation (that is, the ability to achieve a stretch of uninterrupted sleep and to self soothe) by the time they attend school (Mindell et al., 1999), behavioural sleep disorders such as Sleep-onset Association Disorder and Limit Setting Disorder are still commonly re-
Many contributing factors during the teenage years make increasing demands on an adolescent’s evening activities that compete for sleep time. These demands include social activities, sports, part-time employment, and increased academic workloads (Wolfson, 2003). Similar to the younger age groups, there is considerable evidence that increased computer or mobile telephone activity at night, (Carskadon et al., 1998) and the presence of televisions and computers in the bedrooms have a significant delaying effect on sleep onset in the age group (Johnson 2004; Van den Bulck 2004; Olds et al., 2006). Given the biological tendency in adolescents for a delayed sleep phase due to a delay in melatonin secretion and subsequent sleepiness (Wolfson, 2003), these poor sleep habits can contribute significantly to a delayed sleep onset and together contribute to the high percentage of Inadequate Sleep Hygiene Syndrome in adolescents. Poor sleep hygiene practices such as these, not only delay sleep onset directly because of their time-consuming nature but also because they may be stimulating enough (e.g. exciting content of video games) to increase alertness, interfering with natural sleep onset at the regular bedtime. Sleep loss is further aggravated in adolescents who must wake early to attend school (Wolfson et al., 2007) which in conjunction with delayed sleep times result in reduced total sleep time, that is - Insufficient Sleep Syndrome. Insufficient Sleep Syndrome in many adolescents could plausibly be the result of both the biologically and socially-induced reduction of sleep time that occurs compared to pre pubescent years.

In summary the most common sleep disorders across the paediatric life span are Sleep Onset Association Disorder, Inadequate Sleep Hygiene Syndrome, Insufficient Sleep Syndrome and parenteral Limit Setting Disorder.

The Effects of Behavioural Sleep Disorders

Treatment outcome studies where daytime performance deficits are ameliorated post intervention (Dahl et al., 1991; Minde, 2002) support the causal relationship between behavioural sleep disorders and daytime performance. Whilst the effects of behavioural sleep disorders on daytime performance, functioning...
and wellbeing are age dependent they can be generally categorised into three main areas—effects on neuropsychological function, physiological function and psychosocial function.

Neuropsychological Function

Only in recent years has it been apparent that behavioural sleep problems not related to a physiological sleep disorder are related to cognitive function and academic performance (Buckhault, 2011) in school aged children. Sleep parameters that have been implicated in deficits in these areas include reduced sleep time, inconsistent sleep wake schedules, late bed (and rise) times and reduced sleep quality (Buckhault, 2011). It is likely that these sleep problems would meet base criteria for sleep disorders described in the ICSD although they are not often classified as such.

Specifically neurocognitive functions that are impaired in children and young people with behavioural sleep disorders include worse short and long term memory performance (Blunden, 2005), selective attention and poorer executive function performance (Sadeh et al., 2002; Sadeh et al., 2003) compared to controls or good sleepers. Other studies have reported that learning and attention skills are significantly compromised in pre adolescent children with insufficient sleep (e.g., Dahl, 1996; Marcotte et al., 1998; Sadeh et al., 2000). Several studies have reported decreased memory or attentional capacity with reduced total sleep time or poorer quality sleep (Steenari, 2003). These are often translated into poorer academic performance which have been subjectively reported in children with Behavioural Sleep Disorders (Owens et al., 1998; Blunden et al., 2005; Blunden, 2010), Insufficient Sleep Syndrome (Kahn et al., 1989; Carskadon et al., 1998) and non-descript sleep disruption (Sadeh et al., 2002). Two studies have assessed the relationship between objectively defined nondescript sleep disruption and neurobehavioral function in otherwise healthy children. Sadeh et al. (2003) reported that children whose sleep was restricted to 30 minutes less than their regular sleep showed greater deficits in complex neurobehavioural tasks which were reversed when sleep returned to baseline. In a similar study, poorer sleep efficiency and longer sleep latency were related to poor performance on working memory tasks (Steenari, 2003).

The relationship between poor academic performance and insufficient sleep has been reported often in adolescents and is seen to compromise school performance with poorer learning, memory, attention and abstract thinking at a time when successful academic performance is paramount (Wolfson, 2003).

Physiological Function

Sufficient sleep is necessary for maintaining the body’s homeostasis. In consequence, sleep loss has been associated with increased stress and locomotive activity, alterations in hormonal activity and body temperature and changes in cytokines and tumor necrosis factor (Kryger et al., 1994). Children with reduced sleep duration are more likely than other children to be overweight or obese (Carter, 2011) and to have changes in appetite regulation, insulin and glucose utilisation which are associated with metabolic syndrome (Spiegel et al., 1999).

Lastly, inadequate sleep duration has been shown to increase the risk of injury among school aged children. Children who slept less than 10 hours per night were more likely to suffer unintentional injury compared with longer sleepers (Valent et al., 2001). Indeed, gross motor tasks seem to be sensitive to sleep loss. Reaction time was reported as significantly impacted after 18 hours of experimental wakefulness, with balance and agility affected after considerably more sleep loss (42 hours of wakefulness) (Copes, 1972).

Psychosocial Function and Behaviour

Problematic behaviour has long been a consequence of insufficient sleep (Pearl et al., 2002; Carskadon et al., 2004; Yokomak, 2008) with sleep loss resulting in increased aggression, irritability, emotional lability and lower frustration tolerance in all age groups. It would appear that any sleep disorder that reduces either sleep quality or quantity has a detrimental effect on behaviour. This has been clearly demonstrated with respiratory sleep disorders (Chervin et al., 1997; Chervin et al., 2002; O’Brien et al., 2004; Gozal et al., 2007). However problematic behaviours are also significant in children with behavioural sleep disorders—that is, with no respiratory or physiological aetiology. These behaviours include parentally reported problematic behaviours such as irritability, emotional lability, hyperactivity (Owens et al., 1998; Minde, 2002; Blunden et al., 2005), aggression, delinquent behaviours (Smedje et al., 2001; Sadeh et al., 2002) and internalising problems (such as anxiety) (Gregory, 2005).

Interestingly, reduced total sleep time has also been associated with an increased likelihood of mood disorders (such as depression) or psychiatric disorders (such as conduct disorder and attention deficit hyperactivity disorder) (Dahl et al., 1991; Dahl, 1996; Ivanenko, 2008) although it has been suggested that these relationships are bidirectional (Dahl et al., 1991) and are likely to have other contributing factors. Certainly the association between sleep loss and depression has been clearly shown in (Hiscock et al., 2001; Hiscock, 2008).

Family Function

Behavioural sleep disorders can have significant effects on family function (Kerr et al., 1994). Those focussed on infants and pre school children have reported significant detrimental effects of poorly consolidated sleep from behavioural sleep disorders. Numerous studies have shown a strong relationship between poor sleep in young children and poor maternal wellbeing particularly maternal depression, with some studies reporting significant impacts of family function (Hiscock et al., 2001; Hiscock et al., 2007). Some reports suggest that the stress of having a child who has poor sleep and additionally consequential poor behaviour during the day has contributed to increase levels of parental discord and even child abuse (Chavin et al., 1980; Kerr et al., 1994). Maternal reports of good sleep quality attenuate this relationship as clearly, children’s disrupted sleep manifestly disrupts parents sleep and resulting in sleep loss for parents. Indeed, the ICSD contains two sleep disorders which patently are focussed on the effects of poor sleep on parents—Adjustment Disorder and Limit Setting Disorder. The effects of poor sleep are additionally felt in families with lower socio-economic income levels (Buckhault, 2011), which would suggest that in households with other stressors, sleep problems in children have both a cause and effect role on the family.

Mechanisms of Negative Sequelae from Behavioural Sleep Disorders

The mechanisms by which sleep pathology, especially sleep
deprivation and disruption, result in daytime performance deficits remain unclear, although some studies on animals are providing some information. Interestingly, some of the cellular and systemic mechanisms that have been implicated in the daytime deficits associated with intermittent hypoxia seen in respiratory sleep disorders such as sleep apnea, may also operate during sleep deprivation (Blunden et al., 2006).

For example, studies have shown that sleep deprivation-induced cellular injury (Everson, 2005), suppression of neurogenesis and long-term potentiation in the hippocampus in animal models (Silva, 2004) effect functioning of these structures that are involved in motivation, goal direction, reward, and attentional capacity (Hanlon, 2005). It has been suggested that the prefrontal cortex, control centre for executive function (Goldberg, 2002), may be compromised when sleep is less than optimal and this may account for the reduced performance in domains directed by that brain area. In fact there is evidence that sleep deprivation following a learning task reduces communication between the hippocampus and the pre-frontal cortex that ordinarily occurs during sleep (Buckhault, 2011). It has also been suggested that whilst suboptimal brain recovery with sleep loss may be a factor in daytime performance deficits, so too, sleepiness per se, due to fatigue and inability to focus on salient material, may be as important to daytime deficits as more cellular and biological mechanisms or brain pathways (Blunden, 2006).

Furthermore, while both slow wave and rapid eye movement sleep are associated with learning tasks (Buckhault, 2011) REM sleep appears to be important for more memory consolidation tasks than NREM (Pihl, 1997). REM sleep is important for emotional and procedural memory while NREM sleep is important for declarative memory (Walker et al., 2004). Behavioural sleep disorders which reduce sleep quantity consequently shortening the amount of REM sleep in the early hours of the morning, may well have a significant impact on learning capacity, due to changes in REM sleep architecture.

Treatment of Behavioural Sleep Disorders

The direction of causality, in which sleep pathology induces or contributes to daytime deficits, is supported by a number of studies on the efficacy of behavioural interventions for these sleep problems. See Table 3 for common sleep treatments options.

For infants and young children, the most common sleep disorders are those that derive from young children’s inability to self-soothe. The American Academy of Sleep Medicine has released a standards of practice document for behavioural treatment of bedtime problems and night wakings in young children (Mindell et al., 2006). Overall, it was found that of 52 treatment studies reviewed, 94% reported that behavioural interventions were efficacious and 80% of children treated demonstrated clinically significant improvement. Common sleep training methods reported in that paper ranged from parents leaving their child to initiate sleep alone while completely ignoring their protests or signalling cries (extinction or often called the “cry-it-out-method”), through graduated extinction or “controlled crying/comforting” where the parent checks their child with minimal interaction at increasingly longer intervals, to the most interactive model where a parent stays with their child in their room (parental presence) and/or gradually withdraws their presence, eventually leaving the room and then adopting the periodic checking paradigm (the “camping out method”).

There is evidence to suggest that these methods are all successful in reducing nighttime protests and encouraging self-settling and sleeping alone without parental intervention, coupled with low relapse rates. However, reports are also common that those behavioural treatments which require some level of ignoring a child’s cries, are difficult for parents to overcome (Mindell et al., 2006; Meltzer, 2010) and can result in abandonment of the process. Some authors suggest this may even be detrimental to the parent/child relationship (Scher, 1999; Murray, 2007; Cortesi, 2008). Alternative methods of behavioural sleep treatments that do not necessitate ignoring a child’s cry, yet achieve similar sleep behaviour outcomes, are emerging (Blunden, 2011).

Although in older children Sleep Onset Association Disorders are also common, extinction techniques are less common. In older children, desensitisation to sleeping alone can be assisted with childhood cognitive behavioural therapy through psychological techniques of desensitisation to reduce anxiety, thereby developing a child’s confidence and self esteem to sleep alone (Sadeh, 2005; Gordon, 2007). Attention to calming and regular bedtime routines in what is essentially improved sleep hygiene with a component of assisting parents in limited setting is also successful (Mindell 2009).

For adolescents, the most common sleep disorders are Insufficient Sleep Syndrome and Inadequate Sleep Hygiene Disorder. It is clear that adolescents have a biological delay in melatonin secretion which results in a delayed onset of sleepiness and subsequent later bedtimes (Carskadon, 1998). Whilst this is not a behavioural sleep disorder, it is important that the sleep patterns of adolescents be understood within this biological context. Precise history taking to estimate circadian phase based on a careful patient history can assist in deciding if the sleep onset delay is primarily biologically driven. If it is, bright light treatment on waking (to suppress melatonin), reduction of bright light in the evening (to enable the rise of melatonin) and in some cases exogenous melatonin administration are considered to be the treatments of choice for these circadian rhythm sleep disorders (Bjorvatn, 2009). Circadian phase needs to be estimated in order to time the exposure to light appropriately as inappropriately timed bright light and melatonin may likely worsen the condition. Whilst measurements of core body temperature or endogenous melatonin rhythms are seldom used in a clinical practice, estimations of circadian phase are usually undertaken during clinical history taking.

Despite the fact that sleep in adolescents is impacted by delayed melatonin onset, as noted above, a large proportion of adolescent sleep problems are also caused by or at the very least exacerbated by, lifestyle factors. Similar to instigating sleep routines in younger children (Mindell, 2009), improving basic sleep hygiene can be a successful in the first instance. Apart from individual psycho-education including maintaining regular sleep wake schedules, reducing media usage at night and the use of strategic napping, some school based sleep education programs are attempting to increase understanding and knowledge of how to minimise the effects of biologically driven changes to sleep wake patterns during adolescence (Cortesi, 2004; De Sousa, 2007; Moseley, 2009). But knowledge alone is not necessarily equating to behaviour change and this remains a challenge for those who are trying to improve sleep health in this group.

In fact, the understanding of sleep problems in the commu-
nity and even amongst health professionals is low (Owens et al., 2001; Archbold et al., 2002; Blunden et al., 2004). Therefore health professionals who work with children may need to consider basic screening for sleep problems before they become problematic. Even a brief evaluation of sleep (e.g. the BEARS screener—Bedtime, Excessive Daytime Sleepiness, Awakenings, Regularity, Snoring) (Owens et al., 2005) is helpful when conducting routine clinical examinations, especially when there are concerns about a child’s daytime functioning.

Finally, clear evidence exists that delaying school start times has resulted in better performance and wellbeing outcomes in adolescents and may well be considered when attempting to avoid chronic sleep loss in adolescents. However, this is logistically outside the influence of health care professionals.

### Conclusion

Behavioural sleep disorders constitute a significant problem in up to 30% of children and their families and even more in adolescents. Given their high prevalence, the impact on many domains of children’s development and wellbeing, the potential for ongoing sleep and broader health problems with age, coupled with significant family impact, the assumption that behavioural sleep disorders are fundamentally ‘benign’ would appear to be misguided. Similarly the belief that paediatric sleep disorders either are inevitably outgrown or are refractory to treatment may also need re-assessment. Indeed, given the tremendous neural, cognitive, and social changes that occur across the childhood years, increasing awareness, education and treatment options need to be considered to eliminate the impact of what is effectively very modifiable health behaviours.

### REFERENCES


