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Abstract

Sleep problems in childhood are associated with a range of cognitive, emotional and behavioural difficulties. Although insomnia is the most common sleep disorder among children, there are several gaps in research regarding its prevalence, stability and predictors in preschool and middle childhood. Applying a general developmental framework combined with adult theories of insomnia, this study examined temperamental negative affectivity and bedtime parent-child conflicts as predictors of DSM-IV defined insomnia. A community sample of Norwegian children was followed biennially from ages 4 to 10 (n = 1,041) capturing insomnia with semi-structured psychiatric interviews of participating children and their parents. At age 4, insomnia was prevalent in 15.9 %, whereas the corresponding numbers for ages 6, 8 and 10 were 20.2 %, 15.5 %, and 13.8 %, respectively. Those with insomnia had 2 - 4 times higher odds of insomnia two years later compared to those not diagnosed with insomnia. Temperamental negative affectivity measured by the parent-reported Child Behavior Checklist (CBQ) at ages 4 and 6 predicted insomnia at ages 6 and 10, while bedtime parent-child conflicts at age 6 forecasted insomnia at age 8, both adjusted for baseline levels of insomnia. This is the first study to demonstrate these two factors to predict later insomnia and takes us one step further in mapping out the factors contributing to the development of insomnia in children, thus informing preventive and treatment efforts.

Keywords: Insomnia, longitudinal, diagnostical interview, community sample, temperament, negative affectivity, bedtime resistance, stability, predictors, preschool, middle childhood

Predictors of insomnia in childhood. A study of Norwegian children followed from age 4 to 10.

Insomnia is the most common sleep disorder among children, with prevalence estimates ranging between 4 % and 21.2 % (Barclay, Gehrman, Gregory, Eaves, & Silberg, 2015; Steinsbekk, Berg-Nielsen, & Wichstrøm, 2013; Steinsbekk & Wichstrøm, 2015; Zhang et al., 2011; Zhang et al., 2009) and is characterized by difficulties with falling or staying asleep (American Psychiatric Association, 1994). The consequences of childhood insomnia are not well documented, but one study has shown that insomnia increased the risk of developing symptoms of several psychiatric disorders (Steinsbekk & Wichstrøm, 2015). Detrimental consequences of more general *sleep problems* on the other hand, are well documented in children, with a range of cognitive, emotional and behavioural difficulties (Astill, Van der Heijden, Van IJzendoorn, & Van Someren, 2012; Erath & Tu, 2011; Gregory, Eley, O'Connor, Rijsdijk, & Plomin, 2005; Gregory & O'Connor, 2002). In preschool and middle childhood sleep problems are associated with several psychiatric disorders, such as depression (El-Sheikh & Arsiwalla, 2011; Ivanenko, Crabtree, & Gozal, 2005; Liu et al., 2007), anxiety disorders (Alfano, Ginsburg, & Kingery, 2007; Steinsbekk et al., 2013) and attention-deficit hyperactivity disorder (Owens, 2005a). In addition, poor infant sleep seems to affect parental mental and physical health (Sadeh, Tikotzky, & Scher, 2010). Research also highlights the negative effects of insufficient sleep on cognitive functioning (Astill et al., 2012), brain maturation (Peirano & Algarín, 2007) and immune functioning (Besedovsky, Lange, & Born, 2012). As insomnia is regarded a more severe condition than more general *sleep problems*, insomnia probably cause similar or greater impairment in several domains of children's functioning. Therefore, successful detection, prevention and treatment of insomnia is needed, but will depend on a clear understanding of the etiology of the disorder.

Children in preschool and middle childhood are an understudied population as most research on insomnia have focused on adolescents (Amaral, de Figueiredo Pereira, Martins, & Sakellarides, 2013; Hysing, Pallesen, Stormark, Lundervold, & Sivertsen, 2013; Johnson, Roth, Schultz, & Breslau, 2006; Ohayon, 2002; Ohayon, Roberts, Zulley, Smirne, & Priest, 2000; Roberts, Roberts, & Chan, 2008). Further, studies typically apply cross-sectional designs; knowledge regarding stability and prospective predictors of insomnia during childhood is therefore sparse. As far as I am aware, no study has followed young children over time to address possible predictors of insomnia as conceptualized in DSM-IV (American Psychiatric Association, 1994), regardless of requests from several researchers within the field (Gregory, 2011; C. J. Harvey, Gehrman, & Espie, 2014; Ohayon, 2002; Roth & Drake, 2004). The

present study aimed to address these shortcomings by examining prevalence, stability and predictors of insomnia following a large community sample of children from the age of 4 to 10 using a structured psychiatric interview to measure insomnia.

Characteristics, Prevalence and Stability of Insomnia in Middle Childhood

No common, well agreed upon conceptualization of insomnia exists. Although the three most widely used diagnostic manuals concur on the core symptoms being distress or impairment due to difficulties of initiation or maintaining sleep diagnosis (American Academy of Sleep Medicine, 2005; American Psychiatric Association, 1994; World Health Organization, 1992), they differ in the length of time, frequency and quality of additional symptoms required to qualify for an insomnia diagnosis (Ellis, Gehrman, Espie, Riemann, & Perlis, 2012). As Ohayon (2002) points out, insomnia has therefore traditionally been studied in four ways; (1) as insomnia symptoms; (2) as insomnia symptoms with daytime consequences, (3) as dissatisfaction with sleep quality or quantity; and (4) as insomnia diagnosis. The symptoms are usually assessed in three ways, as dichotomous (i.e. yes/no), by frequency and/or by severity (Ohayon, 2002). Perhaps not surprisingly, the range of insomnia prevalence rates reported – as indicated above – in the existing sparse literature is therefore rather broad (Barclay et al., 2015; Steinsbekk et al., 2013; Steinsbekk & Wichstrøm, 2015; Zhang et al., 2011; Zhang et al., 2009). In studies not measuring insomnia in accordance with diagnostical manuals, as those abovementioned, but as one or more insomnia symptoms, a higher percentage (approximately 7 – 36 %) is found (Armstrong, Ruttle, Klein, Essex, & Benca, 2014; Barclay et al., 2015; Calhoun, Fernandez-Mendoza, Vgontzas, Liao, & Bixler, 2014; Camhi, Morgan, Pernisco, & Quan, 2000; Gehrman et al., 2011; Jenni, Fuhrer, Iglowstein, Molinari, & Largo, 2005; Kahn et al., 1989; Petit, Touchette, Tremblay, Boivin, & Montplaisir, 2007; Singareddy et al., 2009). In addition, higher prevalence rates are reported when interviews rather than questionnaires are used. Two previous studies from the present sample indicate a prevalence of 17 – 21 % (Steinsbekk et al., 2013; Steinsbekk & Wichstrøm, 2015) in 4 and 6 year olds, respectively, whereas Barclay et al. (2015) found 18 - 20 % of 8 – 18year olds (modal age 8 - 10) to exhibit insomnia (DSM-III-R), captured by diagnostic interviews.

Only two longitudinal studies have documented the stability of diagnosable insomnia in childhood, showing 43% of those with insomnia at age 4 to have insomnia at age 6 (Steinsbekk & Wichstrøm, 2015), and a 15% persistence rate from age 9 to 14 (Zhang et al., 2011). Only the first of these studies applied a diagnostic interview and none examined the early school

years. Based on the same sample as the Steinsbekk and Wichstrøm (2015) study, the present research will therefore explore the stability of insomnia from age 4 to 10.

In sum, more research is needed to determine the prevalence of insomnia and to reveal whether diagnostically defined insomnia is a stable condition in childhood. Such knowledge is needed to inform prevention and treatment. Simply stated: Stable conditions require interventions, whereas highly fluctuating conditions may not. The present inquiry will address this gap in knowledge.

Predictors of Insomnia

Apart from the former study following the present sample from preschool to first grade to examine the reciprocal relation between sleep disorders and psychiatric symptoms (Steinsbekk & Wichstrøm, 2015), no study has, to my knowledge, followed children in preschool or middle childhood over time to address possible predictors of insomnia, as this condition is conceptualized in DSM-IV (American Psychiatric Association, 1994). Research on preadolescent and adolescent samples do exist though, and one study indicated that low paternal education (<12 years), frequent temper outbursts, and daytime fatigue predicted new incidents of insomnia from baseline at age 9 to follow-up around 14 years of age (Zhang et al., 2011). In a community sample of youths (age 11-17), Roberts et al. (2008) found that at least one symptom of insomnia and daytime fatigue were predicted by female sex and impairment due to health and school stress. Another study found family conflicts (at age 7, 9, 13 and 15) to be the factor most strongly predicting insomnia at age 18 (Gregory, Caspi, Moffitt, & Poulton, 2006), when accounting for gender, socioeconomic status, sleep problems at age 9 and self-reported health at age 18.

As indicated above, studies of insomnia differ a great deal when it comes to sample characteristics such as age (4 – 18 years), informants (mother, child) and cultural identity (western, non-western). In addition, different criteria are used to define insomnia, the number of follow-ups varies, as does the timespan between them. A final limitation of earlier research of childhood insomnia is also the lack of a theoretical framework. Although no specific theory or model for development of insomnia in childhood exists, the present inquiry will apply a general developmental framework combined with adult theories of insomnia to understand why some children develop insomnia symptoms whereas other does not.

Theoretical framework. In line with the biopsychosocial model (Engel, 1989), it is well acknowledged that the etiology of sleep problems represents a complex interplay between biological, psychological, developmental and social influences (Owens, 2005b). Research

within other fields of developmental psychology (Belsky & Beaver, 2011) and psychiatry (Patten, 2013) has supported a diathesis-stress model of disorders, which presumes that environmental factors will affect children differently depending on their genetic composition (Belsky & Pluess, 2009; Monroe & Simons, 1991). According to this model, some children are more vulnerable to environmental stressors - for temperamental, biological or other reasons - whereas others are relatively impervious (Belsky, Bakermans-Kranenburg, & van Ijzendoorn, 2007). Further, a developmental-transactional perspective would indicate that the development and course of sleep disorders involve a constant interchange between the child and the immediate environment (Bronfenbrenner, 1977; Sameroff, 1975), most notably the family (microsystem). Research does support such notion, showing children's sleep problems to be sensitive to parent-child interactions (Bell & Belsky, 2008), e.g. among mothers who experienced more negative emotions and were less sensitive in interacting with the child.

Adult models of insomnia can also be useful in understanding the development of insomnia in children. According to one of the most influential models, the 3P behavioral model of insomnia (Spielman, 1986), predisposing, precipitating and perpetuating factors contribute to the development and maintenance of the disorder. The first two factors of the model advance a stress-diathesis conceptualization of how insomnia is expressed and will therefore be included here. Predisposing factors within the biopsychosocial sphere (e.g., tendency to ruminate, hyper reactivity) make some individuals more vulnerable to insomnia. In combinations with precipitating factors such as threats or stressful life events (e.g., family conflicts), the threshold of insomnia might be surpassed causing sleep disruption. Research does highlight the role of stress and stressful life events as a precipitating factor, mediated by certain predisposing factors of personal vulnerability (Bonnet & Arand, 2003; Drake, Richardson, Roehrs, Scofield, & Roth, 2004; Healey et al., 1981; Vein et al., 2002).

Most other insomnia models stress insomnia as a hyperarousal phenomenon (Ellis et al., 2012; A. G. Harvey, 2002; C. J. Harvey et al., 2014; Lundh & Broman, 2000; Perlis, Giles, Mendelson, Bootzin, & Wyatt, 1997; Perlstrom & Wickramasekera, 1998; Riemann et al., 2010). Apart from this, these models provide next to infinite possibilities regarding predisposing and precipitating factors and their relationships. However, since the first demonstration of hyperarousal as a predictor of sleep disturbance (Coren, 1988), evidence is yet to be concluded as definitive (Roth & Drake, 2004). Of specific importance to the present inquiry, it is not known whether hyperarousal is a central tenet in why children develop insomnia.

In sum, I will therefore test whether a developmentally appropriate and modified version of the 3P behavioral model of insomnia is applicable to children. More specifically, I will examine whether temperamentally defined hyperarousal and bedtime parent-child conflict predict future insomnia diagnosis in the preschool and middle childhood years.

Predisposing factors – temperament. Temperament is defined as constitutionally based individual differences in self-regulation and emotional, motor and attentional reactivity (Rothbart, 2007). Constitutional refers to the influence of heritability, maturation and experience on the individuals relatively enduring biological make up (Rothbart, Ahadi, Hershey, & Fisher, 2001). Reactivity indicates responses from the motor, affective and sensory systems, while self-regulation is regarded as the processes that modulate reactivity by increasing or decreasing it (Rothbart et al., 2001). Temperament is a construct that fits the role as a possible contributing factor in accordance to the 3P behavioral model (Spielman, 1986), in lowering the threshold for developing insomnia in childhood.

One can further categorize temperament along three broad dimensions; Surgency, Negative Affectivity and Effortful Control (Rothbart, 2007; Rothbart et al., 2001; Rothbart, Derryberry, & Posner, 1994). The negative affectivity dimension, which is characterized by dysregulated negative emotions (e.g. discomfort, fear, anger/frustration, sadness), angry reactivity and reduced soothability, is conceptually similar neuroticism in adult personality research (Rothbart, 2007; Rothbart et al., 2001; Rothbart et al., 1994). According to C. J. Harvey et al. (2014), neuroticism and poor emotional regulation in adults are associated with increased stress response and sleep disruption. In adults, arousability (measured by Arousal predisposition scale) is reported as one of the most important predictors of new incidents of insomnia (LeBlanc et al., 2007). Research on the continuity of sleep problems in infants have found difficult temperament to increase persistence of sleep problems (Morrell & Steele, 2003). More specifically, Zhang et al. (2011) found frequent temper outburst (yes/no single item question) to predict new incidence of insomnia during adolescence, yet the validity of such a measurement method is highly questionable.

In sum, these links in infant, adolescent and adult research indicate a role for temperamental negative affectivity in children's sleep, through increased arousability and susceptibility towards dysregulated negative emotions, angry reactivity and reduced soothability. Simple stated, arousal is contraindicative of sleep. Given the many negative effects of insomnia and sleep problems, including increased risk to develop several psychiatric disorders (Steinsbekk & Wichstrøm, 2015), we cannot preclude that it also affects emotion

regulation, arousability and reactivity i.e. core components of negative affectivity. Prospective studies that adjust for previous insomnia are therefore essential when studying the link between negative affectivity and insomnia. However, at present it is unknown whether temperamental negative affectivity may have such effect on prospective insomnia in middle childhood.

Precipitating factors - parent-child bedtime conflicts. Although research point in the direction of a significant genetic contribution to insomnia (Barclay et al., 2015; Gehrman et al., 2011; Palagini, Biber, & Riemann, 2014) it is important to stress that individual differences in insomnia are predominately explained by environmental factors (40 – 80 %). Parents play a decisive role in several domains of children's' functioning, and their involvement in sleep is no exception (Erath & Tu, 2011). In the Conceptual model proposed by Erath and Tu (2011) they suggest that parenting may promote disrupted sleep through negative parent-child relationships that produce psychological discomfort (i.e., arousal and vigilance). This is in line with the evolutionary model of sleep put forth by Dahl (1996), viewing sleep as a state of diminished attentiveness and responsiveness. According to this model, vigilance and sleep are incompatible. Paired with the emotional security model of Cummings and Davies (1996), parent-child conflict in proximity to bedtime may be expected to increase arousal and interfere with children's sense of security, and thus, disturb their sleep.

Bedtime resistance, and resulting conflicts with parents, is one of the most commonly reported sleep problems with estimates of occurrence in 6 – 29 % (Archbold, Pituch, Panahi, & Chervin, 2002; Blader, Koplewicz, Abikoff, & Foley, 1997; Bos et al., 2009; Ivanenko, Crabtree, Obrien, & Gozal, 2006; Jenni et al., 2005; Owens, Spirito, McGuinn, & Nobile, 2000; Simola et al., 2012; van Litsenburg, Waumans, van den Berg, & Gemke, 2010). Conflicts may arise when parents are trying to put an unwilling child to sleep, possibly causing arousal in both the parent and the child, consequently hampering sleep. Such reciprocal effects have been found in a longitudinal study following children and their families from age 8 to 10 (Bell & Belsky, 2008). They found that general sleep problems increased more and decreased less over time when the mother-child relationship was characterized by more conflict and less closeness. Additionally, bedtime resistance have been shown to be more common among those with sleep-onset problems (Blader et al., 1997; 80 % vs 27 % overall) and those labeled as "problem sleepers" (van Litsenburg et al., 2010). Notably, as the two latter studies rely on correlational data, no conclusions regarding the effect on insomnia development can be made.

In lack of prospective research on children, a couple of studies on adolescent have explored possible environmental predictors of insomnia, with perceived stress (Roberts et al.,

2008) and family conflicts (Gregory et al., 2006) being the most important predictors. Some of the most common precipitating factors of insomnia found in adults are perceived negative events related to family, health, and work/school (Bastien, Vallieres, & Morin, 2004). As this might indicate that stress or conflict in proximity to bedtime might influence insomnia development, it is not known whether this applies to children.

Based on the applied theoretical framework and the existing research on adults, adolescents and children, the present study examines bedtime resistance as a precipitating factor in the etiology of childhood insomnia.

Summary and Hypotheses

Insomnia is the most common sleep disorder in childhood and negatively affects children and their families. Yet there are still considerable gaps in our knowledge regarding prevalence, stability and predictors of the disorder. More specifically, preschool and school-aged children are an understudied group, rarely examined longitudinally with diagnostical interviews. As no model or theory of pediatric insomnia has been proposed, I draw upon adult insomnia models in combination with a more general developmental framework and hypothesize that temperamental negative affectivity (i.e. a predisposing factor) and bedtime resistance (i.e. a precipitating factor) will predict increased risk of subsequent diagnosed insomnia when following a large community sample of Norwegian children with biennial assessments from 4 to 10 years of age. In addition, as sleep problems preadolescence have shown to vary by gender, with girls displaying higher levels of sleep problems than boys (Calhoun et al., 2014; Johnson et al., 2006; Laberge et al., 2001), gender was included as a covariate of the present study.

Method

Procedure and Recruitment

All children in Trondheim born in 2003 and 2004 were invited to participate through a letter of invitation ahead of an ordinary community health check-up at age 4 years. Included in this invitation was the Strengths and Difficulties Questionnaire (SDQ) for 4- to 16-years old (Goodman, Ford, Simmons, Gatward, & Meltzer, 2000), which the parents completed and brought with them to the check-up. SDQ has proved to be an excellent tool in screening for mental health problem in preschoolers (Sveen, Berg-Nielsen, Lydersen, & Wichstrøm, 2013). The majority consented to participate in the study (82.1 %). To increase variability and thus statistical power children with mental health problems were oversampled. Children were divided into four strata based on their SDQ score: 0 - 4, 5 - 8, 9 - 11 and 12 - 40. The

probability of selection increased with increased SDQ score (37 %, 48 %, 70 % and 89 % from the respective strata) and a random number generator selected the 1250 families invited to interviews (T1; *M* = 4.57 years, *SD* = .25). Data was then collected every second year, when children were 6 (T2, *M* = 6.72 years, *SD* = .19), 8 (T3, *M* = 8.80 years, *SD* = .24) and 10 years of age (T4; *M* = 10.51 years, *SD* = .27). Figure 1 depicts a flowchart describing the sample recruitment procedure and participation rates. All instruments and procedures used in this study were approved by the Regional Committee for Medical and Health Research Ethics.

Participants

Characteristics of the sample are presented in Table 1. Compared to the average of all parents of 4-year-olds in Trondheim, the sample, adjusted for stratification, contained significantly more divorced parents (7.6 %) than the population (2.1 %). The population of Trondheim was at the time of recruitment similar to the national average of Norway on several key indicators, e.g. employment rate, educational levels, gross income (Wichstrøm et al., 2012).

Because we used a full information maximum likelihood estimator all available data was used. Hence, the analytical sample was *n* = 1,041. Attrition at 8 years of age was marginally predicted by insomnia at age 4 ($p = 0.063$, *OR* = 1.40, 95 % CI [0.98 - 2.00]) and parent-child bedtime conflict at age 6 ($p = 0.057$, *OR* = 1.32, 95 % CI [0.99 - 1.78]). Such bedtime conflicts at age 6 did also predict attrition at age 10 at a borderline level ($p = 0.054$, *OR* = 1.33, 95 % CI [1.00-1.77]). No other study variable predicted attrition.

Measures

Insomnia. The Preschool Age Psychiatric Assessment (PAPA) is a developmentally appropriate interview-based instrument for parents of children aged 2 - 6 (Egger & Angold, 2004) and was administered at age 4 (T1) and 6 (T2). The interview follows a structured protocol and allows for optional follow-up questions when necessary, which ensures that the interviewer gets enough information to determine whether the symptoms of insomnia are present or not. PAPA is a revised version of the Child and Adolescent Psychiatric Assessment (CAPA), which is appropriate for older children and their parents, and hence used at age 8 (T3) and age 10 (T4) (Angold et al., 1995). At these measurement points, the children and the parents were separately interviewed, hence providing two sources of information regarding possible insomnia diagnosis. Computer algorithms matched the information from both interviews with DSM-IV criteria (American Psychiatric Association, 1994), as this was the current diagnostic manual in use at the time of data collection. If either the parent or the child

provided information that met the insomnia criteria, the child was included in the insomnia group.

According to DSM-IV, insomnia is characterized by difficulties initiating or maintaining sleep of nonrestorative sleep that lasts for at least one month and causes clinically significant distress or impairment in important areas of functioning (American Psychiatric Association, 1994). However, DSM-IV does not define a cut-off for the frequency of insomnia symptoms (i.e., times per week), of awakenings at night, or length of time to fall asleep required to obtain an insomnia diagnosis. According to a classification of dyssomnias in younger children (Gaylor, Goodlin-Jones, & Anders, 2001), more than 20 minutes to fall asleep 5 to 7 times a week for at least one month is the threshold for an onset sleep disorder for children older than 2 years of age. Johnson et al. (2006) used the frequency guideline from the ICSD-R (American Academy of Sleep Medicine, 2001) to examine 4 times a week as a frequency threshold for a symptom to count as an episode of insomnia, a frequency at which most of the adolescents in their study started to report daytime impairment. Based on these studies and the DSM-IV criteria, insomnia was defined as (1) more than 30 minutes to fall asleep or the use of sleep medication, or (2) 5 or more night awakenings (duration ≥ 10 minutes) a week for at least one month, or (3) nonrestorative sleep: being insufficiently rested after sleep or having difficulty waking up at least 3 times a week for one month.

The interviewers (n = 7) had at least a bachelor´s degree in a relevant field and extensive prior experience in working with children and families. Blinded raters recoded 9 % of the audio-recorded interviews and inter-rater reliability between multiple raters for insomnia was as follows: PAPA k = .90 and CAPA k = .80.

Parent-child bedtime conflict. PAPA generated information about bedtime parent-child conflicts at age 4 and 6. The parent was asked to describe what typically happens when he/she says the child must go to bed. Typical follow-up questions to capture bedtime conflicts concerned whether the child puts up a fuss, gets angry or upset, how long it lasts and how often it happens. Information at hand provided by these questions was the basis of the interviewer's categorization of bedtime parent-child conflict within five categories ranging from 0 to 4, with 0 indicating no resistance going to bed. Mild resistance easily circumvented by parents was coded as 1 and resistance that deteriorated into conflict possibly including child tantrums and crying was coded as 2. If the parents had to struggle more than one hour to get the child to bed, bedtime parent-child conflict was coded as 3. When parents had given up, the bedtime parent-

child conflict was coded as 4. Coding of bedtime parent-child conflict had an inter-rater reliability of .97 (ICC).

Temperamental negative affectivity. The Norwegian translation of The Children's Behavior Questionnaire (CBQ; Rothbart et al., 2001) measured parent-rated temperament dimensions at age 4 and 6. At age 4 the full version (195 items) was used to measure Negative Affectivity (50 items; $\alpha = .88$), whereas the short version (Putnam & Rothbart, 2006) and its subsequent measure of Negative Affectivity was used at age 6 (25 items, $\alpha = .81$). All items reflect child temperament and related behaviors during the previous six months. Mean scores from the negative affectivity subscales of "Sadness", "Fear", "Anger/Frustration" and reversed "Soothability" (Rothbart et al., 2001) was computed (range 1-7), with higher score indicating more negative affectivity. The CBQ have been found to have good convergent and discriminant validity (Putnam, Rothbart, & Gartstein, 2008).

Statistical Analyses

To test whether insomnia was predicted by bedtime parent-child conflict, temperamental negative affectivity and gender, insomnia at ages 6, 8 and 10 years were regressed on these predictors two years earlier while also adjusting for earlier levels of insomnia. Notably though, as bedtime parent-child conflict and temperamental negative affectivity were measured at age 4 and 6 only, insomnia at 10 years were regressed on these variables at age 6. This comprehensive model was tested within a structural equation framework (SEM) using Mplus 7.31 (Muthèn & Muthèn, 1998-2012). Insomnia at age 10 was defined as a categorical variable. Categorical variables that are conditional endogenous, i.e. that serve as both predictors and outcome (insomnia at ages 6 and 8), cannot be specified as categorical in SEM, therefore we report regression coefficients, not odds ratios. The error terms of predictors at the same time-point were allowed to correlate.

As the sample in this study was screen-stratified, probability weights where the number of children in the stratum were divided by the number of participants in the same stratum to arrive at corrected population estimates were used. The attrition analyses indicated that data might not be missing completely at random. Missing data were therefore handled according to a full information maximum likelihood procedure. A robust maximum likelihood estimator was used, which is robust to deviations from normality, which should be expected for bedtime conflicts, and also provides robust and thus corrected error terms with respect to the oversampling.

Results

Prevalence and Course of Insomnia

Table 2 provides the prevalence of DSM-IV defined insomnia at all measurement points, whereas Figure 2 displays the course of insomnia. The prevalence for ages 4, 6, 8 and 10 years were 14.3 %, 18.4 %, 18.2 %, and 12.8 % among boys and 17.5 %, 22.0 %, 13.0 % and 14.7 %, respectively, among girls. To test whether the prevalence differed between boys and girls we compared a model where the prevalence at each time point was restrained to be equal in the two genders and compared it to a model where the respective prevalence was freely estimated, using a Wald test. The results indicated that there was no significant difference in the prevalence of insomnia between the genders at any measurement point. Additional analyses showed that 23.5 % had diagnostically defined insomnia at one time point, 8.2 % twice, 3.3 % three times, and only 1.1 % at all time points.

The 2-year stability coefficients from age 4 to 6 were *OR* = 3.82, 95 % CIs [2.47, 5.92], 6 to 8 years *OR* = 5.52 [3.38, 9.02], and from 8 to 10 years *OR* = 5.18 [2.94, 9.12], all *p*s < .001. To test whether the stability in insomnia differed between the time periods examined, we compared a model where the stability coefficient was restrained to be equal (e.g. between 4-6 and 6-8 years) and compared it to a model where they were freely estimated, using a Wald test. The results revealed no difference in stability between the time periods examined (Age 4-6 vs Age 6-8: Wald = .39, *df* = 1, *p* = .57; Age 6-8 vs Age 8-10: Wald = .26, *df* = 1, *p* = .62; Age 4-6 vs Age 8-10: Wald = 1.22, *df* = 1, *p* = .27). Gender specific analyses showed that the stability among boys were *OR* = 2.84, 95 % CIs [1.48, 5.45], *OR* = 5.57 [2.80, 11.09], and *OR* = 3.42 [1.49, 7.88] for the age 4 to 6, age 6 to 8, and age 8 to 10 years respectively. The comparable stabilities were *OR* = 4.69, 95 % CIs [2.59, 8.49], *OR* = 6.04 [3.09, 11.83], and *OR* = 8.20 [3.71, 18.12] among girls. The difference in age 4 to 6 stability between the genders were Wald = 1.24, *df* = 1, *p* = .27, age 6 to 8 stability Wald = .03, *df* = 1, *p* = .87, and the age 8 to 10 stability Wald = 2.22, *df* = 1, *p* = .14 were not significant. Thus, no gender differences in stability of insomnia were revealed.

Predictors of Insomnia

Table 3 displays the results of the logistic regression analyses for the whole sample. As can be seen, DSM-IV diagnostically defined insomnia at age 6 was predicted by insomnia and temperamental negative affectivity at age 4, adjusted for gender. Insomnia at age 8 was predicted by insomnia and bedtime parent-child conflict two years earlier, whereas insomnia at age 10 was predicted by insomnia at age 8 and negative affect at age 6 exclusively. The model

fit information is as follows: *AIC* = 7496.12, *BIC* = 7694.04 (No traditional fit indices are possible for analyses with categorical outcomes).

Discussion

The aim of the current study was twofold: To examine the prevalence and stability of insomnia, in addition to test whether temperamental negative affectivity and parent-child conflicts at bedtime predicted future diagnosable insomnia in a large community sample of children followed from 4 to 10 years of age, adjusted for gender. Insomnia was prevalent in 13.8 – 20.2 % of children aged 4 to 10 year and those with insomnia had 2 - 4 times higher odds of insomnia two years later compared to those without insomnia. As hypothesized temperamental negative affectivity and bedtime parent-child conflict increased the risk of later insomnia, although at different ages.

Prevalence of Insomnia

The prevalence found in this study was in accordance with those originating from studies using clinical interviews (Barclay et al., 2015; Steinsbekk et al., 2013; Steinsbekk & Wichstrøm, 2015). Uniquely, it is the first study to unveil the prevalence of diagnostically defined insomnia capturing middle childhood. Notably though, the prevalence rates found here is significantly higher than those utilizing questionnaires (Zhang et al., 2011; Zhang et al., 2009), showing insomnia to be prevalent in 13.8 – 20.2 % of school aged children. These differences might be due to that interviews capture insomnia diagnoses in a more precise manner than questionnaires. Further, questionnaires applied to measure insomnia in childhood are mainly parent-reported (Zhang et al., 2011; Zhang et al., 2009), whereas in the present inquiry information from both parents and children were included. As noted above, to be included in the insomnia group at age 8 and 10, it was sufficient that either the parent or the child reported problems that qualified for an insomnia diagnose. Given that the agreement between children and parents regarding the presence of insomnia symptoms is low (Gehrman et al., 2011), including data from both informants may contribute to a higher prevalence rate. As children's independency increases by age, parents will usually be less involved in their children's bedtime routines and thus less informed about how long they spend before the fall asleep or whether they wake up during the night (e.g. the child no longer go to see his/her parent if wakening during night). It is therefore likely to assume that including both self- and parent-reported data generates more precise estimates of insomnia than relying on parent-reports only.

Stability of Insomnia

This study found earlier episodes of insomnia to predict later insomnia in preschool and middle childhood aged children, which accords with studies of adults (Morin et al., 2009). More than one third of those with insomnia at one timepoint have insomnia two years later; a figure consistent from preschool to middle childhood. These results contrast the work of Zhang et al. (2011) who found insomnia to be considerably less stable from 9 to 14 years (15 % persistence rate). Several methodological differences might explain this discrepancy. Zhang et al. (2011) used questionnaires, with different informants (mother/adolescent) at the two measurement points, and did indeed have a longer follow up (5 years), the end point being in adolescence. As noted above, parents typically underestimate their children's sleep problems (Gehrman et al., 2011; Owens et al., 2000; Paavonen et al., 2000), which might have contributed to the relatively low stability rate revealed in the Zhang et al. (2011) study. Further, as regards sleep problems, the longer the time span between measurement points, the lower stability (Armstrong et al., 2014; Fricke-Oerkermann et al., 2007; Petit et al., 2007; Price, Wake, Ukoumunne, & Hiscock, 2012; Simola et al., 2012), which probably applies to insomnia as well. Even though different research designs make interpretation difficult, it is possible that stability is higher in younger compared to older children. Future studies following children from early years to late adolescents are needed to test such assumption.

A reasonable explanation as to why earlier insomnia is one of the strongest predictors of later insomnia is offered by C. J. Harvey et al. (2014) who state that an initial episode of sleep disruption trigger implicit associative learning. The sleeping environment then becomes associated with negative emotions, an association which is not fully extinguished throughout some potential good sleeping periods, thus the child become more easily activated when encountering new stressors. In terms of the 3P model presented above, earlier sleep disorders may serve as both a predisposition and a perpetuating factor. Findings from adult insomnia treatment research supports such explanation as stimulus control, i.e. reestablishing an association between sleep environment and sleep (e.g., getting out of bed when not able to sleep), is shown to be one of the most effective treatments of insomnia (Morgenthaler, Kramer, et al., 2006; Morin et al., 1999).

Finally, it should be noted that although there is moderate stability of insomnia, the majority of children remits from their insomnia, most likely without professional intervention. Considering the negative effects of sleep problems over time, examining the factors contributing to insomnia persistence, and thereby enable interventions, is of great importance.

Negative Affectivity and Parent-Child Bedtime Conflicts as Predictors of Insomnia

This is the first study to demonstrate that temperamental negative affectivity and parent-child bedtime conflicts predict later insomnia, taking us one step further in mapping out the factors contributing to the development of insomnia in children. Furthermore, these findings fit well with current developmental frameworks and adult insomnia theories and research regarding predisposing and precipitating factors contributing to the development of insomnia.

More specifically the current study found negative affectivity at age 4 and 6 to predict insomnia at age 6 and 10. This finding accords with research on infants showing a link between sleep problems and difficult temperament (Morrell & Steele, 2003) and work by Zhang et al. (2011) indicating that frequent temper outbursts increase the risk of insomnia in adolescence. Further, negative affectivity have been found to be similar to neuroticism in adults (Rothbart, 2007; Rothbart et al., 2001; Rothbart et al., 1994), a personality trait that has been strongly associated with adult insomnia (Andenæs, Helseth, Misvær, Småstuen, & Ribu, 2016). More specifically, research suggest that neuroticism *predispose* to sleep disruption (C. J. Harvey et al., 2014), thus mirroring the current findings showing children high in negative affectivity to be more prone to later insomnia. Although not tested in the current inquiry, a plausible explanation is that children with high levels of negative affectivity will experience more negative emotions and may have less effective strategies to downregulate when in distress, and therefore are less able to fall asleep (i.e. the core symptoms of insomnia) than children with lower levels of negative affectivity. Surprisingly though, negative affectivity at age 6 did predict insomnia at age 10 rather than at age 8, and there is no evident explanation for this counterintuitive finding.

Bedtime parent-child conflict measured at age 6 predicted insomnia at age 8, adjusted for baseline levels of insomnia. This findings expand the correlational data of bedtime resistance being more common among those with sleep-onset problems (Blader et al., 1997) and those labeled as "problem sleepers" (van Litsenburg et al., 2010). This might imply a role for bedtime parent-child conflict in the development and persistence of insomnia in children, especially in early school-age. This finding is in accordance with models suggesting that bedtime parent-child conflict might induce insecurity and arousal in the child (Dahl, 1996; Erath & Tu, 2011), states regarded incompatible with sleep, and thereby contributing to increased insomnia risk. Such bedtime resistance may disturb bedtime routines, as bedtime resistance is associated with an inconsistent bedtime and falling asleep away from bed (Blader et al., 1997), although the causality is not investigated.

Notably though, the relation between bedtime conflicts at age 4 and insomnia at age 6 was insignificant. Hypothetically, parents may tolerate bedtime resistance to a higher degree when children are younger because they perceive such behavior as normal, thus being less likely to identify and report such situations as bedtime conflicts. Nevertheless, future research is needed before we can reach the conclusion that the relation between bedtime conflicts and insomnia is age dependent.

Implications of the current findings

Given the stability of insomnia and the detrimental effects of sleep problems, especially more chronic ones (Armstrong et al., 2014; Gregory & O'Connor, 2002), it is important to identify factors affecting the development and persistence of insomnia. From a preventive perspective, it is important to identify those at risk for insomnia, finding children with higher levels of negative affectivity to be at increased risk can therefore inform preventive efforts More specifically, health care personnel should be particular aware when consulting highly negative affective children with insomnia symptoms, as our study indicates insomnia is less likely to remit in these children, interventions may therefore be required. Although negative affectivity is considered a biologically based construct and is thus less suspect to change, environmental factors is shown to explain a substantial part of the variation in temperament (Saudino, 2005). Further, heritability does not preclude behavioral intervention as twin studies only record the impact of natural environmental variations. In fact, there is consistent evidence that high levels of parental responsivity/sensitivity forecast less negative reactivity in the child (Belsky, Fish, & Isabella, 1991; Braungart-Rieker, Hill-Soderlund, & Karrass, 2010), highlighting interventions affecting parenting styles as a possible prevention strategy against childhood insomnia. Likewise, by introducing positive routines characterized by pleasant and soothing activities to establish a behavioral chain leading up to sleep onset one can likely diminish bedtime parent-child conflict over time, as the current practice parameters for behavioral treatment sleep problems recommend (Mindell, Kuhn, Lewin, Meltzer, & Sadeh, 2006; Morgenthaler, Owens, et al., 2006). Indeed, the lack of such routines and sleep hygiene are associated with sleep problems (Mindell, Meltzer, Carskadon, & Chervin, 2009). Our results suggest that children who display high levels of negative affectivity are in particular need for such routines.

Strengths and Limitations

This study has several strengths; involving a large and representative community sample, a longitudinal design, multivariate analyses and the use of standardized diagnostic interviews

(i.e. PAPA and CAPA). Even so, some limitations should be noted. Ideally, objective measures of sleep should be applied. However, in a large community sample study such as the present, the use of polysomnography, actigraphy and video recording is too cost and time consuming. When using self and proxy reports there is always the risk of not only random inaccuracy but also a bias towards socially desirable responses by parents on behalf of their children or by the children themselves. This may have caused subjects to underreport symptoms, although the fact that interviewers were instructed to investigate whether the symptom was present or not may have counteracted this potential bias.

It should also be noted that 84.8 % of the parent informants were mothers, which could bias the reported child symptoms as parents psychological symptoms and relationship with the child have been found to create divergent parental reports (Treutler & Epkins, 2003). Ideally reports from both mothers and fathers should have been gathered, but because the present study is part of a larger, ongoing longitudinal study (The Trondheim Early Secure Study), the data collection is comprehensive and time-consuming (e.g. at age 6 the child and his/her parent spent a whole day doing assessments), and one could not expect both parents to be present during the interview.

Insomnia in children is an understudied phenomenon and the information about cross-cultural variations is thus sparse. Cultural differences in several sleep related behaviors such as napping, bedtime routines and bedtime (Owens, 2005b) may explain variation across cultures. Although such differences are indicated, findings may easily be confounded with design, measurement and age-group differences. As this study mainly consisted of participants of Norwegian ethnicity, generalization to other populations must therefore be done with caution.

Because the data collection of the present inquiry started before DSM-5 (American Psychiatric Association, 2013) was published and the interview manuals used (PAPA and CAPA) were based on DSM-IV criteria (American Psychiatric Association, 1994), it is considered a limitation that the most up-to-date diagnostic manual was not applied to measure insomnia. The data gathered did not provide sufficient information to diagnose according to the new features of DSM-5 (e.g., insomnia manifestation with or without caregiver intervention, early morning awakenings with inability to return to sleep). Notably though, the current study used a stricter criterion regarding frequency of insomnia symptoms than is included in DSM-5 (at least 4 vs at least 3 nights per week) and a longer sleep onset time (>30 min) than used in former studies (e.g. >20 min reported by Gaylor et al. (2001)). Thus, it is reasonable to assume

that the current results do not overestimate insomnia as defined in DSM-5, although future studies are needed to replicate the present findings using the DSM-5 criteria.

Furthermore, it should be noted that several other environmental factors not included in the current inquiry may contribute the development and persistence of insomnia such as other parent-child interactions and parenting practices. Finally, genetic measures were not included in the current study. Temperament and insomnia has been found to have similar genetic contributions (Barclay et al., 2015; Gehrman et al., 2011; Palagini et al., 2014; Saudino, 2005), and negative emotional reactivity has been found to predict internalizing problems (e.g., anxiety, depression; Rothbart & Bates, 2006), in roughly the same manner as genetic factors related to insomnia correlate with those of depression and anxiety (Gehrman et al., 2011). Future studies are needed to explore whether the relation between negative affectivity and insomnia revealed here is due to a genetic overlap.

Conclusions

The aim of the current study was twofold: To examine the prevalence and stability of insomnia, in addition to test whether temperamental negative affectivity and parent-child conflicts at bedtime predicted future diagnosable insomnia in a large community sample of children followed from 4 to 10 years of age, adjusted for gender and baseline levels of insomnia. Insomnia was prevalent in 13.8 – 20.2 % of children aged 4 to 10 year and those with insomnia had 2 - 4 times higher odds of insomnia two years later compared to those without insomnia. In line with adult models of insomnia and a more general developmental framework, temperamental negative affectivity predicted insomnia from age 4 to 6 and age 6 to 10, while bedtime parent-child conflict at age 6 predicted insomnia at age 8, adjusted for baseline levels of insomnia. This is the first study to demonstrate these two factors to predict later insomnia and takes us one step further in mapping out the factors contributing to the development of insomnia in children, thus informing prevention and intervention.

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Figure 1.

Sample recruitment

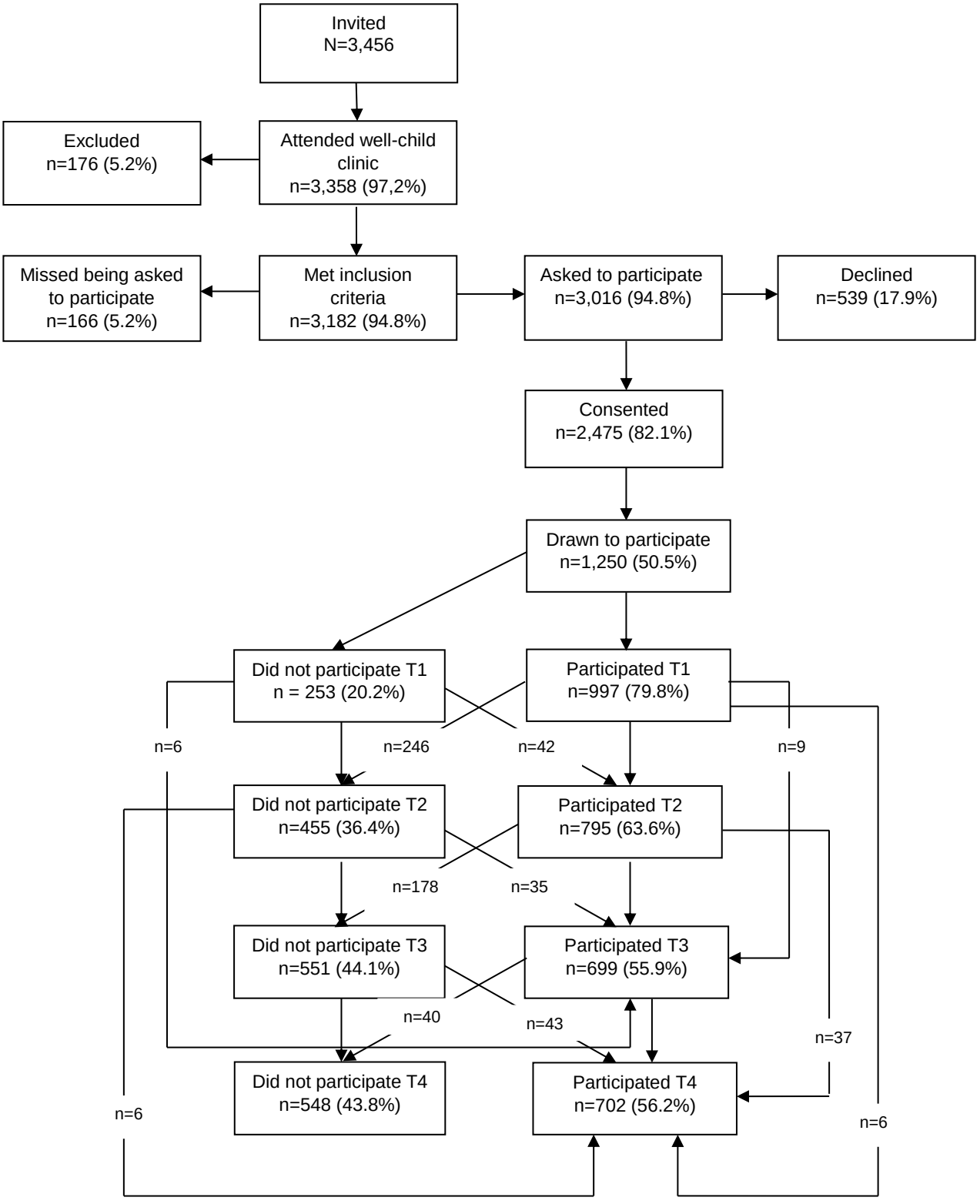


Table 1**.**

Sample characteristics

Characteristic		**%**
Gender of child	Male	49.1
	Female	50.9
Gender of parent informant	Male	15.2
	Female	84.8
Ethnic origin of biological mother	Norwegian	93.0
	Western Countries	2.7
	Other Countries	4.3
Ethnic origin of biological father	Norwegian	91.0
	Western Countries	5.8
	Other Countries	3.2
Childcare	Official day care center	95.0
	Other	5.0
Biological parents' marital status	Married	56.3
	Cohabitating > 6 months	32.6
	Separated	1.7
	Divorced	6.8
	Widowed	0.2
	Cohabitating < 6 months	1.1
	Never lived together	1.3

		%
Informant parent's socio-economic status	Leader	5.7
	Professional, higher level	25.7
	Professional, lower level	39.0
	Formally skilled worker	26.0
	Farmer/fisherman	0.5
	Unskilled worker	3.1
Parent's highest completed education	Not completed junior high school	0
	Junior high school (10^{th} grade)	0.6
	Some education after junior high school	6.1
	Senior high school (13^{th} grade)	17.3
	Some education after senior high school	3.4
	Some college or university education	7.6
	Bachelor degree	6.2
	College degree (3-4 years study)	33.6
	Master degree or similar	20.3
	PhD completed or ongoing	4.4
Households' gross annual income	0 – 225' NOK (0 – 26 500 USD)	3.3
	225' – 525' NOK (26 500 – 62' USD)	18.4
	525' – 900' NOK (62' – 106' USD)	51.6
	900' + NOK (106'+ USD)	26.7
At least one parent has received treatment for mental health problems	None	73.8
	Outpatient only	16.3
	Hospitalized	10.0
Parents received medical treatment for mental health problems	No	87.4
	Yes	12.6

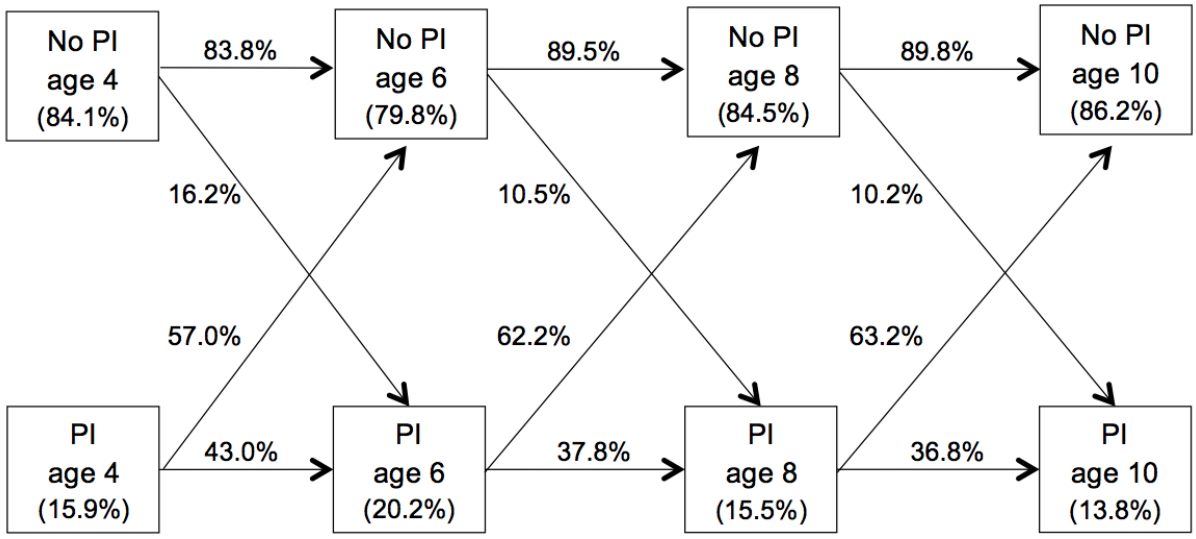
Table 2.

Descriptives of study variables

Study Variable	Mean or Percentage (95 % CI)
Insomnia 4 years	15.9 % (13.5, 18.4)
Insomnia 6 years	20.2 % (17.3, 23.2)
Insomnia 8 years	15.5 % (12.5, 18.4)
Insomnia 10 years	13.8 % (10.9, 16.7)
Bedtime parent-child conflict 4 years (0-4)	0.31 (0.27, 0.35)
Bedtime parent-child conflict 6 years (0-4)	0.31 (0.27, 0.35)
Temperamental negative affectivity 4 years (1-7)	3.63 (3.59, 3.66)
Temperamental negative affectivity 6 years (1-7)	3.73 (3.68, 3.77)

Figure 2.

Course of Insomnia



Note. PI = DSM-IV defined primary insomnia.

Table 3.

DSM-IV diagnostically defined Insomnia at ages 6, 8 and 10 years predicted from parental and child factors (n=1041)

Predictors 2 years earlier[1]	Insomnia age 6			Insomnia age 8			Insomnia age 10		
	B (95 % CI)	*p*	β	B (95 % CI)	*p*	β	B (95 % CI)	*p*	β
Insomnia	0.22 (0.13; 0.32)	≤ .001	0.20	0.23 (0.14; 0.32)	≤ .001	0.26	1.46 (0.85; 2.07)	≤ .001	0.27
Negative affectivity	0.11 (0.05; 0.18)	≤ .001	0.13	0.05 (-0.01; 0.10)	.078	0.07	0.50 (0.05; 0.96)	.030	0.14
Bedtime parent-child conflict	0.04 (-0.02; 0.09)	.237	0.05	0.08 (0.02; 0.14)	.010	0.12	0.30 (-0.15; 0.75)	.186	0.09
Gender	0.02 (-0.03; 0.08)	.418	0.03	-0.07 (-0.12; -0.01)	.019	-0.09	0.21 (-0.31; 0.74)	.423	0.06

[1] Both Temperamental negative affectivity and Bedtime parent-child conflict were assessed only at age 4 and 6, so diagnostically defined Insomnia at 10 years were predicted by these two factors 4 years earlier.

Note. CI = confidence interval, B = unstandardized Beta, β = standardized Beta.