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Insomnia in childhood and adolescence – prevalence, persistence, and predictors

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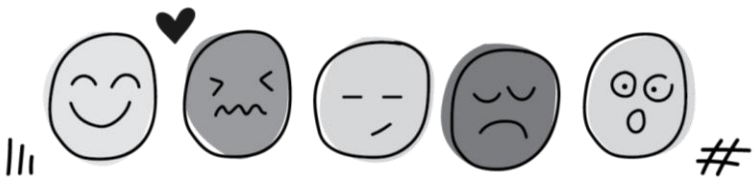

Norsk sammendrag

Denne avhandlingen undersøker forekomst, stabilitet og prediktorer for søvnlidelsen insomni i barne- og ungdomsårene. Insomni kjennetegnes av vanskeligheter med å sovne eller å holde seg sovende og er forbundet med negative konsekvenser for livskvalitet, mental helse og skolegang hos barn og ungdom. Gitt disse negative følgene kan forebyggende tiltak eller tidlige intervensjoner være berettiget. Det forutsetter imidlertid at insomni har en viss forekomst i barneårene og at den vedvarer over tid. Vansker som er ekstremt sjeldne og som går over av seg selv er det mindre behov for å forebygge eller behandle. Målet med denne avhandlingen er derfor å undersøke forekomsten av insomni, hvorvidt de som har fått insomni fortsetter å ha det (stabilitet) og hva som kan forklare at noen utvikler insomni (prediktorer).

To av artiklene i avhandlingen bygger på data fra studien Tidlig Trygg i Trondheim, en longitudinell studie av barns psykiske helse hvor omtrent 1000 barn har blitt undersøkt annet hvert år fra de var 4 til 14 år. Insomni ble kartlagt gjennom diagnostiske intervju av både foreldrene og barna hver for seg. I tillegg ble spørreskjemadata benyttet for å kartlegge en rekke ulike faktorer ved barnet og familien som ifølge teori og tidligere forskning kan være av betydning for insomni. Den siste artikkelen er en gjennomgang og oppsummering av all forskning som har undersøkt prediktorer for insomni fra 4-19 års alder.

Resultatene fra avhandlingens første artikkel tyder på at til enhver tid fra 8 til 14 års alder har omtrent 1 av 10 barn insomni. Forekomsten var lavere da barna var 4 og 6 år gamle, men det kan henge sammen med at vi på disse alderstrinnene bare intervjuet foreldrene. Flere gutter enn jenter hadde insomni frem til 10 års alder, men denne kjønnsforskjellen snudde ved starten av tenårene. Insomni var moderat stabilt, de som hadde insomni på et tidspunkt (i motsetning til de som ikke hadde) hadde større risiko for også å ha insomni to år senere. Artikkel tre nyanserte dette stabilitetsfunnet ved å vise at insomni fortsatte å være en risiko for senere insomni, selv når man kontrollerte for en rekke andre mulige grunner til insomni i en modell som også justerte for alle tidsinvariante faktorer (for eksempel gener). I tillegg viste resultatene at en økning i barnets emosjonelle reaktivitet, ADHD symptomer og en nedgang i emosjonsreguleringsevne bidro til økt risiko for insomni to år senere. Dette gjaldt for hele 10-års perioden (4-14 år). Karakteristika ved familien slik som familiefungering og parkonflikter førte ikke til senere insomni i denne studien. Den siste artikkelen i avhandlingen (litteraturgjennomgang) fant at mentale helseproblemer (i hovedsak tegn til depresjon) og det

å være jente kan øke risikoen for å få insomni. Den viktigste konklusjonen fra denne artikkelen er imidlertid at det finnes veldig få studier som har undersøkt hvorfor noen utvikler insomni i barneårene. Det er videre mange forskjellige faktorer som er undersøkt og det mangler replikasjonsstudier, noe som gjør det vanskelig å konkludere med hva som fører til insomni.

Avhandlingen gir også en oversikt over insomni som forskningsfelt. Teoretiske perspektiver på problematisk søvn presenteres, utfordringene knyttet til å definere og operasjonalisere insomni drøftes, og det gis en oversikt over tidligere forskning inkludert en identifikasjon av eksisterende kunnskapshull. Avhandlingens resultat diskuteres i lys av eksisterende litteratur. Metodologiske betraktninger og mulige implikasjoner av funnene drøftes.

English Summary

This thesis examines prevalence, persistence, and predictors insomnia. Research on sleep problems indicate that they are common and persistent, but we know comparable little about sleep *disorders* such as insomnia. Insomnia is characterized by difficulties initiating and/or maintaining sleep and has detrimental consequences for quality of life, mental health, and schooling. Given these negative outcomes, and if childhood insomnia is prevalent and stable, early intervention may be warranted. To inform preventative efforts and interventions, we need to examine the prevalence of diagnosable insomnia in childhood, to what extent it persists and identify potential predictors. Such knowledge is sparse due to lack of longitudinal studies addressing insomnia with multifactorial models. The present inquiry therefore reports on the prevalence, persistence and predictors of diagnostically defined insomnia from preschool to late adolescence.

In order to do so, two of the Papers (I and III) used data from biannual assessments in the Norwegian longitudinal cohort study the Trondheim Early Secure Study (age 4 to 14 years). Insomnia was assessed through separate clinical interviews of both parents and children. The starting point for insomnia definition and operationalization was the fourth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV) and these criteria were adjusted to concur with the more recent DSM-5. The predictors examined were theoretically linked to insomnia through their influence on hyperarousal, which is assumed to be a main factor in insomnia development and persistence. Paper (II) reviewed the literature of predictors of insomnia (age 4 to 19 years) using a systematic search with strictly defined inclusion criteria.

The results from Paper I demonstrated that insomnia is found in approximately one in ten children at ages 8, 10, 12 and 14 years of age. Fewer had insomnia at ages 4 and 6 years, but at these ages only parents were interviewed, so estimates at older ages were expected to be higher as they relied on both child and parental reports. More boys than girls evinced insomnia in middle childhood, but this shifted to a female preponderance in early adolescence. Insomnia was moderately stable, with those having insomnia (as opposed to those without) being at greater risk of insomnia two years later. Paper III built on this finding and revealed that insomnia forecasted later insomnia even when we adjusted for several other predictors and accounted for all time-invariant unobserved factors. We further found that increases in child emotional reactivity and symptoms of ADHD and decreases in child

emotion regulation skills predicted insomnia two years later across the 10-year period examined. Family factors, in the form of family functioning and marital conflicts, did not predict later insomnia. Lastly, Paper II suggests female sex and mental health problems (most notably depression) may be involved in the etiology of insomnia. However, the diversity of predictors studied in previous reports combined with lack of replication prevent any firm conclusions from being drawn, but the review serves as summary of best available evidence.

The thesis also provides an introduction to research field of insomnia, including the challenges encountered when defining and operationalizing insomnia, theoretical perspectives on problematic sleep, as well as an overview of previous research of childhood insomnia and gaps of knowledge. Findings are discussed in light of the current literature. Methodological concerns and potential implications for research and clinical practice are discussed.

Contents

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Trondheim, December 2020
Jonas Falch-Madsen

List of papers

Paper I

Falch-Madsen, J., Wichstrøm, L., Pallesen, S., & Steinsbekk, S. (2020). **Prevalence and stability of insomnia from preschool to early adolescence: a prospective cohort study in Norway**. *BMJ Paediatrics Open*, *4*(1), e000660. doi: 10.1136/bmjpo-2020-000660

Paper II

Jonas Falch-Madsen, Lars Wichstrøm, Ståle Pallesen, Magnus Rom Jensen, Lene Bertheussen, Solvor Solhaug, Silje Steinsbekk.

Predictors of insomnia in child and adolescent community samples: A literature review

Submitted to Sleep Medicine Reviews.

Paper III

Jonas Falch-Madsen, Lars Wichstrøm, Ståle Pallesen, Bror M. Ranum, Silje Steinsbekk.

Child and Family Predictors of Insomnia from Early Childhood to Adolescence

Submitted to Sleep Medicine.

Acronyms and Abbreviations

ADHD	Attention Deficit Hyperactivity Disorder
BFI	Big Five Inventory
CAPA	Child and Adolescent Psychiatric Assessment
CBQ	Children's Behaviour Questionnaire
CFI	Comparative fit index
CI	Confidence Intervals
CPS	Conflicts and Problem-Solving Scales
DF	Degrees of Freedom
DSM	Diagnostic and Statistical Manual of Mental Disorders
FIML	Full Information Maximum Likelihood
ICC	Intraclass Correlation
ICD	International Classification of Mental and Behavioral Disorders
ICSD	International Classification of Sleep Disorders
JFM	Jonas Falch-Madsen
LB	Lene Bertheussen
M	Mean
MO	Months of age
MRJ	Magnus Rom Jensen
OR	Odds Ratio
PAPA	Preschool Age Psychiatric Assessment
REK	Regional Committee for Medical and Health Research Ethics
RI-CLPM	Random Intercept Cross-Lagged Panel Model
RMSEA	The Root Mean Square Error of Approximation
PSG	Polysomnography
SD	Standard Deviation
SDQ	Strengths and Difficulties Questionnaire
SES	Socioeconomic Status
SOL	Sleep Onset Latency
SRMR	Standardized Root Mean Square Residual
SSol	Solvor Solhaug
TESS	Trondheim Early Secure Study
TLI	Tucker-Lewis fit index
WASO	Wake after sleep onset
X^2	Model Chi Square
Y	Years of age
ΔX^2	Model Chi Square Delta

1 Introduction

Sleep problems in childhood are common (Armstrong et al., 2014; Camhi et al., 2000; Munezawa et al., 2011; Patten et al., 2000; Schlarb et al., 2015), persistent (Armstrong et al., 2014; Fricke-Oerkermann et al., 2007; Simola et al., 2012; Williamson et al., 2019b) and longitudinally linked to increased risk of emotional, behavioural and cognitive problems (Alvaro et al., 2017; Goldstone et al., 2020; Gregory & Sadeh, 2012, 2016; Meijer et al., 2010; Pieters et al., 2014; Shanahan et al., 2014; Stormark et al., 2019; Williamson et al., 2020). Findings from 2002-2014 indicate an increase in the prevalence of sleep-onset difficulties in European children over time (from 17.5% to 20.8%; Ghekiere et al., 2019). Young children with sleep problems use more medical services than children without sleep problems and are a heavy financial burden to the healthcare system, especially when sleep problems are persistent (Quach et al., 2013).

For a substantial share of young people, their sleep problems may not warrant clinical attention because the difficulties lack the severity, duration, intensity or associated impairment typically captured by a clinical diagnosis such as insomnia. At present though, we know comparatively little about the prevalence, stability and predictors of childhood insomnia, which is an important gap in knowledge given that insomnia per definition cause clinically significant distress or impairment in important areas of functioning (American Academy of Sleep Medicine, 2014; American Psychiatric Association, 2013; World Health Organization, 1992) and as such are more severe than sleep problems. The focus of the present dissertation is therefore on the most prevalent and potentially more debilitating condition than mere problems of sleep, namely insomnia disorder.

Research on the impact of insomnia among children are also sparse, but one longitudinal study indicated that insomnia is associated with impaired health-related quality of life (especially psychosocial aspects) and especially so for persistent insomnia (Combs et al., 2016). Additionally, the authors reported insomnia to be associated with an increased risk of developing a chronic medical condition. A second study found preschool insomnia to increase the risk for developing symptoms of major depressive disorder, social phobia and conduct disorder two years later (Steinsbekk & Wichstrom, 2015). Research on the impact of insomnia among adolescents are more common (Shochat et al., 2014) and links insomnia to depression (Johnson et al., 2006a; Lovato & Gradisar, 2014; Roberts & Duong, 2013; Roberts et al., 2002), lower self-esteem and perceived social support (Roberts et al., 2002),

school absenteeism (Bauducco et al., 2015), drinking and driving under the influence (Catrett & Gaultney, 2009) and suicidality (Goldstein et al., 2008; Wong & Brower, 2012). Given these potential outcomes and if childhood insomnia is prevalent and stable, early intervention may be warranted. To provide a platform for preventative efforts and interventions, we need to identify contributors to the incidence and persistence of childhood insomnia. Such knowledge is sparse though, due to lack of longitudinal studies addressing insomnia with multifactorial models, thereby capturing predictors at different levels of influence (i.e., child, family, environment). Further, any predictors and stability of insomnia derived from observational evidence may be open to the influence of confounding, most notably persistent vulnerability (e.g., genes) or unmeasured third variables. Therefore, by applying a statistical method that adjusts for all unmeasured time-invariant confounding, capturing interview measured insomnia at ages 4, 6, 8, 10, 12 and 14 in a community sample of children, the present thesis examines prevalence, stability and predictors of insomnia, thereby providing a novel contribution to childhood insomnia research.

1.1 Defining insomnia

As discussed in detail below (see section 1.1.2), defining insomnia has been a major challenge within the field of sleep medicine for decades. The three reigning diagnostic manuals do agree on the core symptoms of insomnia though, which are *subjective reports of clinically significant distress or daytime impairment due to difficulties of initiation and/or maintaining sleep.*

1.1.1 From sleep problems to insomnia – complicating issues

As highlighted by a task force supported by the American Academy of Sleep Medicine (Owens et al., 2005), the range of sleep behaviors or problems that may be considered normal or pathologic is wide and the definitions are often highly subjective. Problematic sleep in childhood should probably be viewed along a continuum from normal sleep to disorders that meet specific criteria, with individuals more or less likely to move along this continuum in certain ages or developmental periods. Similarly, investigations of problematic sleep in childhood have traditionally ranged from one item inquiries such as "Do(es) you/your child have problems sleeping" to more thorough assessments of diagnostically defined sleep disorders, such as insomnia. Most of the research in childhood relevant for this thesis has focused on problems with sleep initiation or maintenance, and

more behavioral expressions / phenotypes in younger children (e.g., bedtime resistance). These can be viewed as insomnia-related problems which may not have the severity, duration, intensity, or associated impairment that warrants a clinical *diagnosis.* With such a broad spectrum of what is normal and problematic in childhood, it has become challenging to provide accurate age-appropriate estimates of the prevalence, stability and predictors of problematic sleep (i.e., the continuum from sleep problems to insomnia disorder). Differences in estimates relate to several factors, including measurement method (e.g., interviews, surveys, sleep diaries, choice of informant), populations (e.g., age range, clinical or community), time of assessments (e.g., time between measurements) and perhaps most prominently criteria used to determine the sleep problem or disorder investigated (e.g., use of different classification systems, dichotomized or continuous variables, evaluation of frequency and duration of problems/symptoms, evaluation of comorbidities or daytime impairments).

First, as already touched upon, the definition and degree of sleep problems greatly affects estimates. Examining sleep problems with open single items allows for subjectivity, the responder having to interpret what constitute a "sleep problem". Confounding factors such as personality related responding styles or cultural perceptions of what is regarded problematic is obviously a downside of such subjective approach. Cultural comparisons suggest that parents from predominantly Asian regions are more likely to characterize their child's sleep as a problem, especially a severe problem (Sadeh et al., 2011). Efforts to counter such confounding include to specify questions or response alternatives to cover operationalized aspects of the sleep problems (i.e., frequency, severity, duration or associated impairment). Within these aspects of assessment there is further variations; questions regarding frequency can for example have response alternatives in the form of "seldom" or "often" (i.e., opens up for interpretation) as opposed to "once a week" or "2-3 times a week". Such detailed inquires may produce more precise descriptions of the sleep behaviour or problem in focus, yet a possible consequence is moving too far away from the subjectivity that characterizes something as problematic or impairing.

Second, most epidemiological research has relied on questionnaires to capture sleep patterns and problematic sleep, which is understandable for cost-effectiveness reasons. However, concerns have been raised regarding the wide use of sleep questionnaires as few of them fulfil required psychometric properties (Spruyt & Gozal, 2011), especially with regards

to overemphasis on internal reliability, lack of validity and diagnostic power. Further, insomnia-specific questionnaires are lacking in pediatric sleep assessment (Lewandowski et al., 2011). Other forms of assessments include sleep-diaries, interviews and observational/objective measures (i.e., actigraphy, video, polysomnography). Notably, these assessments normally cover different time-periods when they probe for problems. Observational data are seldom collected for more than a week, sleep diaries for a couple of weeks, while questionnaires and interview may inquire sleep patterns for months prior to the assessment. Examining longer periods of time may be less affected by short-time fluctuations in sleep but may invite retention problems in the informants, especially children. Although objective measurements often have been considered the gold standard of sleep assessment, it does not include the unique subjective experience of sleep as problematic which is essential in diagnostic criteria of insomnia (American Academy of Sleep Medicine, 2014; American Psychiatric Association, 2013; World Health Organization, 1992). Indeed, this is recognized by the third edition of the International Classification of Sleep Disorders (ICSD-3; American Academy of Sleep Medicine, 2014) which emphasizes that use of polysomnography (PSG) to evaluate insomnia is unnecessary, although useful in ruling out other sleep disorders. Empirical longitudinal research using PSG found no differences in sleep architecture or sleep parameters (e.g., sleep latency, time in bed, total sleep time, sleep efficiency) between children with or without ICSD-2 defined insomnia (Combs et al., 2016). Comparisons between objective and subjective measures of problematic sleep tend to yield different results (Lang et al., 2013; Pesonen et al., 2014; Sateia et al., 2000). Therefore, observational measures of sleep difficulties along the continuum from normal sleep to insomnia continuum are relevant to inform factual elements of sleep (e.g., sleep duration), but seem to be less relevant in assessing sleep disorders such as insomnia which includes diagnostic criteria related to the subjective experience of a sleep disturbance. Clinical interviews and sleep diaries on the other hand, are recommended tools in clinical settings when evaluating insomnia (Brown & Malow, 2016; Mindell & Owens, 2015; Owens et al., 2005; Sateia et al., 2000), but the latter have also been criticised for poor psychometric properties (Spruyt & Gozal, 2011). Clinical interviews stand out as the preferred method when addressing diagnoses but is seldom used in research. Thus, there seem to be a discrepancy between research and clinical practice and when it comes to assessment of childhood insomnia. Because the latter relies on the first, this is unfortunate.

Third, another factor making estimates within the field of sleep problems challenging is the trade-off concerning the use of informants, namely whether a parent or the child is the most reliable source in evaluating sleep problems. Parents may be well positioned in evaluating young children's sleep because children are likely to signal when facing problems sleeping. Moreover, parents are involved in bedtime routines. Additionally, one can question whether young children have the capacity to evaluate the occurrence and duration of sleep problems given the proximity to the clouded state of mind transition to sleep is characterized by. However, as children's independency increases by age, parents will usually be less involved in their children's bedtime routines and thus less informed about potential problems with sleep onset or nocturnal awakenings (e.g., the child no longer signals his/her parent). Parents may nevertheless still be able to observe or be informed of potential daytime impairments across settings. A recent review of the limited evidence on this topic indicates that parents typically have poor knowledge of children's sleep (age 3mo-17y; McDowall et al., 2017). More specifically, McDowall et al. (2017) found that parental knowledge of daytime sleepiness, sleep practices and settling problems were somewhat better than knowledge regarding nocturnal sleep problems. Additionally, empirical evidence in middle childhood (age 8-10 years) indicate that when sleep initiation and maintenance problems are serious and/or frequently occur, they are typically under-reported by parents, although not prominently so for minor/mild problems (Fricke-Oerkermann et al., 2007; Gregory et al., 2006b; Paavonen et al., 2000). In contrast, reports of daytime sleepiness are more common in parent-reports relative to reports of their offspring (Gregory et al., 2006b). Consistent with parents typically under reporting serious sleep problems compared to their offspring, Gehrman et al. (2011) found that children (aged 8-16 years) reported substantially more insomnia according to the revised third version of the Diagnostic and Statistical Manual of Mental Disorders (DSM-III-R) than their parents did on their behalf (19.5% vs 6.6%). In detail, parents and children agreed upon the occurrence of insomnia in 79.8% of the time (77.1% absence and 2.7% presence by both informants). Notably though, parents reported no symptoms in 85.9% of the cases where children reported insomnia (i.e., 85.9% of the 19.5% child-reported insomnia). Additionally, parents reported insomnia in 4.5% of cases where children reported no problems. In all, this may indicate that parents do tend to miss their children's insomnia symptoms or that the definition of problematic sleep according to parents is influenced by the amount of disruption caused to parents' sleep (typically less as independency develops). An alternative explanation is that children differ from their parents in their subjective threshold of what is regarded problematic (especially severe and frequent

problems). This is worrying considering that in most studies addressing the continuum from sleep problems to insomnia, parents act as sole informants.

In sum, the above noted methodological and conceptual issues and challenges identified in research on problematic sleep also applies to the study of childhood insomnia, and thereby forms an important backdrop of the present work.

1.1.2 Insomnia according to diagnostic manuals

Defining sleep problems based on diagnostic criteria can contribute to a more uniform conceptualization, and because medical decisions are based on diagnoses, the use of diagnoses in research will conform to medical decision-making processes. The three most widely used diagnostic manuals when assessing insomnia in adults are, in their most recent editions, DSM-5 (American Psychiatric Association, 2013), International classification of mental and behavioral disorders (ICD-10; World Health Organization, 1992), and ICSD-3 (American Academy of Sleep Medicine, 2014). The former two are diagnostic manuals covering mental disorders according to American and European expert work groups named by American Psychiatric Association and World Health Organization, respectively. The latter is a sleep-specific diagnostic manual developed by worldwide international sleep experts and published by the American Academy of Sleep Medicine. Overall, these three diagnostic manuals concur on the core symptoms of insomnia in adults being *clinically significant distress or daytime impairment due to difficulties of initiation and/or maintaining sleep and/or early-morning awakenings with inability to return to sleep*. Further, they agree on these symptoms to occur at least 3 times a week and not better explained by other sleep sleep-wake disorders. To be appreciated, in previous editions, these manuals diverged more in their detailed insomnia criteria, which have made research on the epidemiology of insomnia challenging.

Definitions of childhood/pediatric insomnia have historically been absent in the abovementioned diagnostic manuals, except for behavioral insomnia of childhood in the ICSD classification. So in order to clarify which clinical situations the use of pharmacotherapy may be appropriate, a task force supported by the American Academy of Sleep Medicine (Owens et al., 2005) arrived at the following key components in a consensus definition for pediatric insomnia (mainly aimed at children below 12y):

> *"Pediatric insomnia may be defined as difficulty initiating or maintaining sleep that is viewed as a problem by the child or caregiver. The significance of the sleep problem may be characterized by its severity, chronicity, and frequency and associated impairment in daytime function in the child or family. The sleep problem may be due to a primary sleep disorder or occur in association with other sleep, medical, or psychiatric disorders."* (Owens et al., 2005, p. 50)

Although developed to guide when pharmacological treatment may be appropriate, this definition has guided the descriptions of child-specific phenotypical characteristics of insomnia in DSM-5 and ICSD-3. In general, these manuals and ICD-10 indicate that caregiver- or self-report of symptoms and impairment is sufficient for a diagnoses and underscore that sleep initiation and maintenance may be expressed as bedtime resistance, or camouflaged by conditioning (e.g., only falls asleep with favorite teddy bear) or caregiver intervention (e.g., only falls asleep with parent presence). Typically, when considering insomnia in childhood, the diagnosis is based on a detailed clinical history from interviews of both the child and the parent or caregiver (Maski & Owens, 2016; Owens et al., 2005). Additionally, the use of a sleep diary is recommended, kept by the patient or caregiver for 2 consecutive weeks, that includes sleep-wake patterns, estimated sleep onset latency (SOL), timings of nocturnal awakenings, wake after sleep onset (WASO), and naps (Brown & Malow, 2016; Maski & Owens, 2016).

Of relevance to the current thesis, DSM-IV (American Psychiatric Association, 1994) and ICSD-2 (American Academy of Sleep Medicine, 2005) were the prevailing editions in addition to ICD-10 when the Trondheim Early Secure Study (TESS) started in 2007, which is the study that the present work is based on. Therefore, the insomnia definition applied here was partly guided by the data collection decisions taken in TESS. Because TESS addresses the development of mental health and disorders, the current edition of the generic research-based DSM-IV was reasonable to apply when the study was launched, as opposed to a pure sleep disorder manual such as ICSD. Therefore, DSM-IV was used as a starting point for insomnia conceptualization in this thesis, but with the updated knowledge within DSM-5 and ICSD-3 the aim has been to adapt the DSM-IV criteria to the newer DSM version when deemed possible, to make the research as relevant and up to date as possible (e.g., symptom frequency criteria of 3 times per week). Therefore, the insomnia criteria applied here, which are presented in detail below (chapter 2.1.7.1) and discussed in the methodological

considerations section (chapter 4.5.1.3), aim to provide operationalization of DSM-IV insomnia criteria concurring with present DSM-5 and ICSD-3 when possible.

1.2 Theoretical considerations

1.2.1 Sleep and insomnia in a developmental perspective

Research on sleep initiation and maintenance problems from preschool to late adolescence indicates that these problems are prevalent (8-36% at least three times a week; Armstrong et al., 2014; Calhoun et al., 2014; Camhi et al., 2000; Chaput et al., 2018; Martin et al., 2007; Munezawa et al., 2011; Patten et al., 2000; Pesonen et al., 2014; Schlarb et al., 2015; Singareddy et al., 2009; Spruyt et al., 2008), which suggest that problematic sleep is not necessarily atypical in certain developmental periods. As described by Mindell and Owens (2015), the evaluation of insomnia in childhood requires a basic understanding of what constitutes "normal" sleep. Characteristics of normal sleep and typical difficulties with sleep initiation and maintenance from infancy to adolescence varies greatly as sleep development progresses. In general, sleep patterns and behaviours are evolved and modified by both intrinsic (e.g., biological systems and their maturation, genetics, personality) and extrinsic (e.g., parenting practices, school start times, cultural influences) processes throughout development.

Sleep is not simply rest, but an active process where some brain regions show corresponding or increased activity as during wakefulness (Dahl & Lewin, 2002; Siegel, 2017). It is regarded a recurring state characterized by altered consciousness and reduced physical activity. Sleep is the primary activity in infancy and by school start children have typically spent more time asleep than awake (Galland et al., 2012; Iglowstein et al., 2003; Sadeh et al., 2009; Schlarb et al., 2015), although with great individual and cultural variations (Dias et al., 2018; Galland et al., 2012). Two great and complex developmental milestones for infants (and their parents) is to learn to settle in (i.e., *sleep regulation*) and sleep through the night (i.e., *sleep consolidation*). As sleep-wake rhythms starts to mature around 3 months of age (Rivkees, 2003), infants start merging their nocturnal sleep periods which leads to the longest sleep period being 5-8 hours (Henderson et al., 2011; Sadeh et al., 2009), enabling most infants to sleep through the night within their first year (Galland et al., 2012; Henderson et al., 2011). During the first year of life self-regulating skills starts to develop which enables self-soothing and settling progress (Rothbart et al., 2011). This can be observed in decreases

in mean SOL from 40 to 20 minutes during the first year of life (Galland et al., 2012; Paavonen et al., 2020). At night-time, it is normal for infants to awaken 2-3 times lasting a total of 15-60 minutes during the night, typically decreasing through the first year of life to once a night and 10-15 minutes (Galland et al., 2012; Paavonen et al., 2020; Rivkees, 2003; Sadeh et al., 2009). It should be noted that these studies of normal sleep development report considerable variability, with the upper problematic 5% (+2 SD) of 1-3-year olds estimated to experience as much as 45 minutes SOL, 4-6 awakening lasting a total of 1 hour per night. Of note, the most prominent predictor of a parent-reported sleep problem in children's first three years of life are reported to be number of nocturnal awakenings, followed by prolonged sleep onset latency (Sadeh et al., 2011; Sadeh et al., 2009), both regarded symptoms of insomnia in later ages.

Thus, when entering preschool age, an age period captured in this thesis, many children struggle with SOL and frequent and/or enduring night awakenings that parents experience as problematic, with more similarities than differences across cultures (Owens, 2005). During preschool age, children have usually halted napping during the day (Galland et al., 2012; Iglowstein et al., 2003) which can further consolidate the night-time sleep period. Focusing on the normal development of more insomnia-related phenomena, mean SOL is indicated to be 10-20 minutes across age and measures (Galland et al., 2018; Goodwin et al., 2007; Gradisar et al., 2011; Ozgun et al., 2016; Russo et al., 2007; Spruyt et al., 2008) and fairly stable entering adolescence according to objective measures (Ohayon et al., 2004), with more conflicting results regarding decrease (Russo et al., 2007) or increase (Spruyt et al., 2008) according to child reports. Between 6-28% of all children use 30 minutes of more to fall asleep (Petit et al., 2007; Russo et al., 2007; Simola et al., 2012), whereas the corresponding number for adolescents is 12-20% (Chung & Cheung, 2008; Ozgun et al., 2016; Russo et al., 2007). Of note though, sleep onset latency does not necessarily denote an experience of sleep initiation as problematic, but it is reported to be an important predictor of parent-reported sleep problem and the most important predictor in school-aged children (Williamson et al., 2019a). Another measure that encompasses sleep onset latency as problematic is reports of difficulties initiating sleep. From preschool through early adolescence, which is the age period captured in the present work, estimates of problems with sleep initiation or bedtime resistance fall between 2-26% (BaHammam et al., 2006; Fricke-Oerkermann et al., 2007; Ghekiere et al., 2019; Jenni et al., 2005; Lehmkuhl et al., 2008; Paavonen et al., 2000; Schlarb et al., 2015; Simola et al., 2012; Smedje et al., 2001; Spruyt et

al., 2008; Williamson et al., 2020). In late adolescence, the corresponding range is suggested to be 5-17% (Abdel-Khalek, 2004; Chung & Cheung, 2008; Gradisar et al., 2011; Hayley et al., 2015; Kaneita et al., 2006; Ohida et al., 2004; Schlarb et al., 2015).

Less research have addressed nocturnal awakenings, but both subjective and objective measures indicate a decrease in awakenings and time awake at night from toddlerhood through adolescence (Jenni et al., 2005; Ohayon et al., 2004; Petit et al., 2007). Approximately one-third of 2.5-year-old children and one-fourth of children aged 4 years awaken every night, decreasing to 13% and 5% at ages 6 and 10 years, respectively. Another study found only 2% to awaken more than twice a night from age 3 to 11 years of age (Simola et al., 2012). Reports of difficulties maintaining sleep is suggested to be less prevalent than problems with initiating sleep, with prevalence estimates of 1-18% from preschool through early adolescence (BaHammam et al., 2006; Fricke-Oerkermann et al., 2007; Lehmkuhl et al., 2008; Paavonen et al., 2000; Schlarb et al., 2015; Simola et al., 2012; Spruyt et al., 2008; Williamson et al., 2020) and 7-12% in late adolescence (Abdel-Khalek, 2004; Chung & Cheung, 2008; Kaneita et al., 2006). However when prevalent, nocturnal awakenings often lead to parents experiencing and reporting offspring sleep as problematic (Williamson et al., 2019a).

Finally, non-restorative sleep (insomnia symptom in DSM-IV) and daytime sleepiness (potential marker of sleep as non-restorative) have been found to greatly vary across studies, ranging from 4% to 46% from preschool through late adolescence (Abdel-Khalek, 2004; BaHammam et al., 2006; Camhi et al., 2000; Choi et al., 2009; Munezawa et al., 2011; Ohida et al., 2004; Simola et al., 2012; Spruyt et al., 2008; Williamson et al., 2020). Of note, daytime sleepiness may also be an indication of insufficient sleep duration (i.e., not necessarily insomnia) as adolescents typically develops an ‘eveningness’ in their circadian preference (Russo et al., 2007). Their shifting to later bedtimes without being able to stagger rise times because of school is a likely contributor to daytime sleepiness, as indicated by some studies (Chan et al., 2018; Gariépy et al., 2017).

To sum up, what constitutes normal in terms of initiating and maintaining sleep varies considerably by age. Simply stated, it is more typical than untypical to display difficulties with sleep initiation and maintenance in some developmental periods. This fact underlines the importance of assessing diagnostically defined sleep problems when aiming to identify

prevalence, persistence and predictors of atypical or pathological sleep difficulties, as is the case of the present work on childhood insomnia.

1.2.2 Theories of insomnia development and persistence

According to one of the most influential models of insomnia, the 3P behavioral model (Spielman, 1986) predisposing, precipitating and perpetuating factors contribute to the development and maintenance of the disorder. Predisposing factors (e.g., personality, temperament) make some individuals more vulnerable to insomnia and in combination with precipitating factors typically perceived as threatening or stressful (e.g., family conflicts), leading to the threshold of insomnia to be surpassed causing sleep disruption. Perpetuating factors can both be predisposing and precipitating, or in the form of maladaptive strategies when trying to cope with insomnia (e.g., daytime napping, earlier bedtimes). The 3P model has in many ways provided a framework that remains relevant for insomnia theories and models. Because most of the theoretical work has focused on adults, a brief review of this theorizing is warranted before moving on to models of childhood insomnia.

1.2.2.1 Insomnia in adults

Despite considerable heterogeneity in insomnia models and theories, most include some sort of hyperarousal as a potential inhibitor of the sleep system (Bonnet & Arand, 1997; Harvey, 2002; Harvey et al., 2014; Lundh & Broman, 2000; Riemann et al., 2010). Hyperarousal can be divided in cognitive-emotional and physiological hyperarousal, that take place in autonomic or cortical areas, and may be viewed in a trait (i.e., predisposing/personality) or state (i.e., precipitating/acute stressors) manner. Throughout the recent years there is growing evidence that support hyperarousal as a an important contributor to insomnia (Bonnet & Arand, 2010; Drake & Roth, 2006; Riemann et al., 2010), yet there is still some uncertainty whether hyperarousal leads to insomnia or vice versa insomnia provokes arousal or whether there is a bi-directional relationship. Some have suggested insomnia to be a 24-h disorder of hyperarousal (Roehrs et al., 2014), but which vulnerabilities or processes that lead to hyperarousal are not that well investigated and thus poorly identified. Hyperarousal has been proposed to be a characteristic of an increased stress-reactivity (Drake & Roth, 2006), in the sense that stressful events leads to greater sleep disruption. This stress-diathesis approach accords with the 3P model in acknowledging predisposing, precipitating and perpetuating factors are in play.

In a review of such a vulnerable phenotype of insomnia, Harvey et al. (2014) outlined a psycho-bio-behavioral model of insomnia backed by empirical evidence, noting that genetics and negative affect (neuroticism) and poor emotional regulation lead to disrupted sleep via augmented and underregulated responses to stress. More specifically, certain specific genes (e.g., 5HTTLPR - a likely modulator of responsiveness to stress; Harvey et al., 2014) or a pool of genes may contribute to our physiological stress response, whereas certain personality traits guide how we perceive and cope with stress. Physiological and psychological stress/arousal are thought to lead to increased stress-reactivity and are likely to trigger each other. If an individual have poor regulation skills when facing stress, arousal may be further increased (i.e. not able to downregulate) and thus contribute to the negative conditioning between the sleep setting and not being able to sleep which is typically seen in insomniacs (Robertson et al., 2007). In the most effective treatments for insomnia, such conditioning is successfully targeted by stimulus control and sleep restriction (Riemann et al., 2017). As outlined here, genetics do seem to play a role as do a propensity toward negative effect (along with poor regulation skills) when it comes to precursors of hyperarousal and insomnia development and persistence. The above-cited research does not discuss whether these potential mechanisms also apply to childhood insomnia.

1.2.2.2 Insomnia in childhood and adolescence

To my knowledge, there are no well-recognized insomnia specific theoretical models for children. Some models do aim to explain development of childhood sleep though, which may be relevant for our understanding of problematic sleep and thereby insomnia. Further, as stated by Owens (2005), sleep-related behaviors and problematic sleep in children are likely to result from a complex interplay between biological, psychological, developmental, social, and cultural influences. Thus, in line with Spielman's original framework, childhood insomnia can most likely be explained by a combination of predisposing, precipitating and perpetuating factors (Mindell & Owens, 2015). That is, some children are more vulnerable to insomnia for genetic, physiological, or psychological reasons and thereby are more prone to negative effects from physiological or environmental stressors that may trigger insomnia. If insomnia is already present, perpetuating factors such as conditioning and maladaptive sleep habits and cognitions may cause persistent insomnia.

1.2.2.3 The Two-process model of sleep regulation

The Two-process model of sleep regulation (Borbély, 1982) describes the interaction between sleep drive and the circadian timing system to determine the timing of sleep and waking and acknowledges biological contributors to problematic sleep. According to the theory, sleep drive/pressure builds during wakefulness and is drained during sleep, whereas the circadian timing fluctuates with a near perfect 24-hour cycle influenced by the light–dark cycle. In this model's view, the need for daytime naps in young children indicates that they accumulate sleep drive quicker than older children, who can sustain wakefulness during the day (Jenni & LeBourgeois, 2006). Also, delaying bedtime 1h later in middle childhood has been reported to lead to shorter sleep latency and less night awakening (Sadeh et al., 2003), most likely as a consequence of increase sleep drive. The sleep-phase delay in the transition to adolescence observed across cultures is understood as a combination of delayed circadian timing, changed sensitivity to evening and morning light, and slower rise in sleep pressure. Thus, the preferred biological bedtime is delayed, but in the context of most educational systems across the world adolescents are expected to go to bed at biologically unfavorable times, potentially leading to prolonged SOL and daytime sleepiness at school days.

1.2.2.4 Arousal regulation and sleep

From a more evolutionary perspective on problematic sleep in children, Dahl (1996) conceptualized sleep and vigilance as opponent processes in a larger system of arousal regulation. According to this perspective, the brain regularly cycles through patterns of higher and lower arousal states influenced by abovementioned biological contributors. On a moment-to-moment basis however, these arousal states are influenced by attentional and emotional threats, demands or experiences. Therefore, on the pendulum of arousal between the mutually exclusive states of sleep and vigilance it is adaptive for sleep to occur at physical places with minimal need for vigilance. Way back in history and compared to other species, humans have lacked nocturnal locations that were physically ideal (i.e., nests, burrows), thus safety from predators was organized through protective social groups (Dahl, 1998). The social-emotional context, that is feelings of social connectedness and its related emotions, have therefore been strongly linked to the sense of safety required for sleep. Today, this harmonizes with effective pediatric sleep recommendations that encourages parents to meet children's emotional needs during the day, ensure a consistent bedtime routine and a positive atmosphere in the children's living environment (Allen et al., 2016).

1.2.2.5 Other theoretical considerations

Additionally, although not incorporated in theoretical models *per se*, several prominent researchers and clinicians within pediatric sleep medicine point towards the contribution of inadequate sleep hygiene to development of sleep initiation and maintenance problems (Lipton et al., 2008; Mindell & Owens, 2015; Moore, 2012). Ineffective sleep hygiene can be divided into two factors: sleep behaviors and sleep cognitions. The former includes maladaptive sleep habits, including excessive time in bed, irregular sleep-wake schedules and daytime napping. Daytime napping is likely to lower evening sleep pressure, whereas excessive time in bed(room) may weaken the conditioning to sleep. Irregular sleep-wake schedules (e.g., later rise times in weekends) may defer the accumulation of sleep drive and increase the risk for long sleep latency before weekdays. Most of these sleep behaviors have received support in an evaluation of recommended sleep practice (Allen et al., 2016). Sleep cognitions typically include beliefs and attitudes regarding sleep (e.g., "I'll never fall asleep") and the possible consequences of poor/less sleep (e.g., "I'll get bad grades"), and might then contribute to sleep initiation problems through cognitive or emotional arousal that arises from these cognitions (Harvey & Greenall, 2003).

As seen above, insomnia related theories mainly address factors at the level of the individual and the family. From a cultural perspective though, several other factors beyond the microsystem are also thought to contribute to insomnia development and persistence (Owens, 2005). For example, rise- and bedtimes are largely culturally dependent (e.g., school start times, work vs family time after school), and may therefore influence a child's sleep drive at sleep onset. Other examples are accessibility to, and culture specific norms related to use of electronic devises, as are differing conditions for perceived safe sleeping environments.

In sum, several theories and models may inform our understanding of insomnia development and persistence. The insomnia specific contributions arise from the more established field of insomnia in adults, while more sleep generic perspectives are found within research on children and adolescents. In general, hyperarousal or factors contributing to hyperarousal (i.e., physiological, cognitive, emotional) at both the level of the individual (e.g., temperament/personality, regulatory skills) and its surroundings (e.g., family system, bedroom environment) seem to predict insomnia.

1.3 Insomnia – Empirical evidence and gaps of knowledge

What is known about prevalence, stability and determinants of insomnia in childhood? As described, previous research falls along the continuum from sleep problems to diagnostic insomnia. As Ohayon (2002) points out, insomnia has traditionally been studied in four ways; (1) as dissatisfaction with sleep quality or quantity, (2) as insomnia symptoms, (3) as insomnia symptoms with daytime consequences, 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). Here, the focus will primarily be on empirical evidence regarding insomnia diagnosis and its prevalence, stability and determinants from childhood through early adolescence. Please note that if relevant, insomnia-near findings and research in late adolescence will also be included. Studies examining insomnia applying diagnostic criteria and clinical interviews and/or sleep diaries will be highlighted, because this is most similar to clinical practice.

1.3.1 Prevalence of Insomnia

Regarding the developmental period captured in the present work, no studies have assessed insomnia according to diagnostic manuals and by means of interviews. However, three interview-based studies have used insomnia measures without assessing whether daytime impairments were present (Barrios et al., 2018; Steinsbekk et al., 2013; Steinsbekk & Wichstrom, 2015). Two of these stem from the TESS (Steinsbekk et al., 2013; Steinsbekk & Wichstrom, 2015) reporting parent-reported prevalence of DSM-IV insomnia without considering daytime impairment to be 16.6% and 21.2% in 4 and 6 year old children, respectively. Lastly, as part of a validation study in a sample of children where half of the biological mothers had a lifetime history of depression, Barrios et al. (2018) reported DSM-IV insomnia prevalence in 4 and 7 year old children to be 12% and 18.5%, respectively.

By also including samples that consists of children *and* older adolescents (14+ years of age), four studies from US (Barclay et al., 2015; Gehrman et al., 2011; Johnson et al., 2006b; Roberts et al., 2006), and one from China (Chung et al., 2014) have examined insomnia applying diagnostic criteria and interviews. Gehrman et al. (2011) examined DSM-III-R defined insomnia at baseline in the Virginia Twin Study (age range 8-16, mean 12 years) and found a prevalence of 19.5% and 6.6% according to child- and parent-report, respectively. Barclay et al. (2015) also used data from the Virginia Twin Study but used age

groups spanning from 8 to 18 years on all follow-ups (modal ages 11, 14 and 15 years), and found child-reported insomnia rates of 17.9%, 17.4% and 11.5%, respectively. Barclay and colleagues used an insomnia duration of at least 1h as a threshold for difficulties initiating and maintaining sleep, as their measures only permitted duration in hours. The same probably applies to Gehrman and colleagues using the same data and sample, although not explicitly stated in their article. Using data from the Teen Health study, Roberts et al. (2006) found insomnia prevalence to be 6.7% in a sample of 11 to 17 years old adolescents (4.7% when excluding mood, anxiety and substance disorders) with no differences across ethnicities. Specifically, they reported estimates of insomnia at each age which revealed considerable variation across age (e.g., 24% at age 11-12 and 5-6% at age 13-14) despite a solid sample size (e.g., around 1,100 aged 11-12 years). Of note, the authors used DSM-IV criteria, but instead of the usual 3 symptoms (i.e., trouble falling asleep, nocturnal awakening(s) with trouble falling asleep, nonrestorative sleep) they used 5 symptoms of insomnia (adding frequent nocturnal awakenings with no trouble falling asleep and waking up very early). A study by Johnson et al. (2006b), examined DSM-IV insomnia in 13 to 16-year-olds using 4 times a week as a frequency criteria of symptoms, arriving at child-reported prevalence estimates of 9.4% and 10.7% for current and lifetime insomnia, respectively. They used this frequency threshold as this was the threshold where most adolescents began to report daytime impairment. Lastly, in a validation study of three questionnaires, Chung et al. (2014) reported a DSM-IV-TR insomnia prevalence of 9.3% among 12-19-year-olds. Symptom criteria thresholds was operationalized based on clinical experience as sleep onset latency $\geqslant$ 20 min, wake after sleep onset $\geqslant$ 10 min, and early morning awakening $\geqslant$ 20 min, respectively, that occurred $\geqslant$3 days per week in the past month.

Additionally, one Australian study (Dohnt et al., 2012) using a comprehensive questionnaire battery in combination with sleep diaries is worthy of a mention. Triumphing the abovementioned studies in their compliance to diagnostic criteria (i.e., controlling for other sleep disorders, mental disorders, substance use opportunity in bed), they found 7.8%, 10.9% and 3.4% to meet criteria for DSM-IV primary insomnia, ICSD-2 general insomnia, and ICSD-2 psychophysiological insomnia, respectively. As can be seen, prevalence rates of diagnostically defined insomnia by means of interviews differ from 4.6% to 24.3% and varies by age, criteria and informants used.

1.3.2 Stability of Insomnia

No previous studies have addressed the stability of insomnia according to diagnostic manuals with interviews within the age span addressed here, but two studies come close. Steinsbekk and Wichstrom (2015) report that children with DSM-IV insomnia without assessing daytime impairment at age 4 were 4 times more likely (odds ratio) of having insomnia at age 6, compared to those who did not have insomnia at age 4 years. This indicated that 43% of those with insomnia at age 4 had insomnia two years later. A one-year follow up of youths aged 11-17 showed that 34.7% of the adolescents who originally had DSM-IV insomnia continued to have it one year later (23.6% when excluding mood, anxiety and substance disorders; Roberts et al., 2008).

In addition to two studies using interviews, some studies have measured insomnia diagnoses using questionnaires. In a study set in Hong Kong, Zhang et al. (2011) captured parent-reported 12-month DSM-IV insomnia without considering daytime consequences at age 9 years and used child-reports at follow-up 5 years later and found insomnia to persist in 15% of those with insomnia at age 9 years. In the TuCASA study, Combs et al. (2016) examined ICSD-2 defined insomnia including daytime consequences and opportunity for enough sleep, but without any frequency thresholds for symptoms. Insomnia was based on parent-reports at age 9 years, child-reports at ages 13 and 15 years. Persistent insomnia was defined as insomnia at all time-points or at the latter two time-points, resulting in 7% reporting persistent insomnia according to the age-adjusted ICSD-2 near insomnia definition. What these longitudinal studies of insomnia in general report, is that insomnia is persistent in 7% to 43% of original cases and varies by persistence definition (e.g., all or subsequent measurements), time between and informants across measurements (i.e., different informants typically yield lower persistence).

1.3.3 Determinants of Insomnia

Not surprisingly, because few studies address insomnia longitudinally and apply diagnostic criteria there is sparse knowledge regarding predictors of diagnostically defined insomnia in childhood. Steinsbekk and Wichstrom (2015) investigated psychiatric disorders as risk factors for insomnia without assessing daytime impairment. When adjusted for insomnia at age 4, they found symptoms of attention-deficit hyperactivity disorder, oppositional defiant disorder, and major depressive disorder to increase the risk for insomnia

at age 6. In unadjusted models (i.e., not accounting for symptoms of other psychiatric disorders), symptoms of generalized anxiety disorder and separation anxiety at age 4 years were statistically linked to insomnia at age 6 years. However, unadjusted models do not take into consideration contribution of previous insomnia on later insomnia (see the above stability) and the impact of comorbid conditions of the disorder in question and on later insomnia.

Although no other diagnose-near interview-based studies exists, some surveys should be mentioned. In the above noted follow-up study Roberts et al. (2008) report that incidence of DSM-IV insomnia (both with/without excluding mood, anxiety, and substance use disorders) was significantly predicted by female sex, more perceived limitations in daily life activities due to physical health, and more perceived stressfulness of the social environment at school (e.g., violence, crowdedness, teacher treatment) one year earlier. Incidence of DSM-IV insomnia without excluding mood, anxiety, and substance use disorders was additionally predicted by perceived diminished life satisfaction and lower mental health. In a study measuring DSM-insomnia by interviews only at age 18 years, but adjusting for sleep problems at age 9 years, Gregory et al. (2006a) reported the mean level of family conflict at ages 7, 9, 13, and 15 years to predict insomnia at age 18. Further, they revealed a dose-response relationship, with increased insomnia risk for every previous measurement point reporting “high” family conflict. Of note, sleep problems at age 9 years were not linked to insomnia at age 18 years. In a 5-year follow-up study (baseline age 9 years) of chronic insomnia (present for 12 months, but daytime consequences was not included in criteria), Zhang et al. (2011) found incidence of insomnia to be associated with baseline lower parental education, frequent temper outburst and daytime fatigue. Persistent insomnia was linked to baseline chronic medical conditions.

1.3.3.1 Genes and biological sex in Insomnia development and persistence

As noted in models of insomnia, genetics is expected to be involved in the etiology of insomnia (Barclay & Gregory, 2013). Findings from the baseline of a twin study by Gehrman et al. (2011) indicate that genetic factors play a modest role in the etiology of insomnia symptoms (30.7% heritability) in 8-16-year-old children and that these genetic effects overlap with those of depression and overanxious disorder. Thus, common genes may contribute to both insomnia *and* other psychopathology or traits. Addressing the genetic contribution to insomnia stability in a longitudinal study stemming from the same twin cohort, Barclay et al.

(2015) report genetic influence at modal age 8 years (baseline) to contribute to insomnia at all later measurements, indicating stability of the genes at play at different ages.

In adults, a female preponderance has been identified (Zhang & Wing, 2006), thus biological sex has been viewed as a potential contributor to insomnia. In a review of insomnia in adolescence de Zambotti et al. (2018) reveals that a sex difference in insomnia or insomnia symptoms emerge with the onset of menses (Hysing et al., 2013; Johnson et al., 2006b; Zhang et al., 2016), indicating that changes due to puberty might be involved in the etiology of insomnia. Notably though, it is plausible that it is the behavior rather than the sex that matters, that some behaviors which happens to be sex-differentiated, constitutes predictors of insomnia. Such an example is perceived school stress, a predictor of insomnia (Roberts et al., 2008) which is higher in girls than in boys (Klinger et al., 2015) and thus may influence the sex difference in insomnia. Also, female adults tend to engage in more help seeking behavior for insomnia (Morin et al., 2006), which may contribute to more registered insomnia cases. However, one study investigating help-seeking behavior in children and adolescents found no sex difference (Liu et al., 2016). Lastly, although investigated cross-sectionally, Zhang et al. (2016) found sex differentiated behaviors to be associated with insomnia symptoms: Boys engaging in more maladaptive lifestyles (e.g., alcohol, energy drinks) and girls experiencing more emotional and relationship difficulties were at increased risk for insomnia. In all, an emerging sex-difference in insomnia prevalence may be linked to puberty, thus indicating a role for biological sex. However, the association between sex-differentiated behaviors and insomnia suggests that there may not necessarily be biological processes behind the observed sex difference, thus indicating a role of gender (i.e., social construction).

1.3.3.2 Hyperarousal in Insomnia development and persistence

As illustrated in the theories and models presented above, hyperarousal is likely to play role in the development and persistence of insomnia. Whether there indeed exist empirical evidence of links between problematic sleep or insomnia symptoms and cognitive-emotional or physiological hyperarousal should therefore be scrutinized. Cortical hyperarousal during non-rapid eye movement sleep (NREM) in childhood has been identified as a precursor of incidence of insomnia symptoms in adolescence (Fernandez-Mendoza et al., 2019), providing preliminary evidence for the role of hyperarousal in childhood insomnia. Further, individuals who experience negative emotions more frequently and intensely than others are likely to be more prone to emotional hyperarousal than those who experience fewer

negative emotions. Children with high levels of difficult temperament, or later in the form of emotional instability (i.e., neuroticism), are – compared to others – characterized by a tendency to more frequent and intense negative emotions, worse response when facing stressors, and a poorer soothability from emotional arousal (Boyce & Ellis, 2005; Rothbart, 2007; Widiger, 2009). Indeed, difficult temperament has been reported as a well-established risk factor in a systematic review of more general sleep problems in children (Newton et al., 2020), although the authors note a trend of diminishing associations beyond infancy. In adults, however, research back neuroticism as a predisposing factor of insomnia (Harvey et al., 2014), possibly through increased perceived stress and poor coping styles (Connor-Smith & Flachsbart, 2007). Thus, although indicated by the above to be of importance, there is a gap of knowledge regarding the role of negative affectivity or neuroticism as a hyperarousal-vulnerability for insomnia in childhood and adolescence. The current thesis aims to address this gap of knowledge.

Moreover, as difficult temperament and neuroticism are characterized by negative emotions that are intense and enduring and impair sleep, adaptive emotion regulation strategies may protect against insomnia through the ability to (down)regulate emotions and thereby arousal. Such a relationship is proposed in the above-mentioned psycho-bio-behavioral model of insomnia (Harvey et al., 2014). Preliminary findings among children and adolescents indeed suggest an association between emotion regulation and general sleep problems (Palmer et al., 2018; Williams et al., 2017), yet no causal interpretations can be made. In adults, the link between emotion regulation and insomnia has been reviewed (Cerolini et al., 2015), with results indicating that sleep deprivation impairs the use of adaptive emotion regulation strategies and increases negative emotions. Whether emotion regulation may protect against insomnia in children in accordance with a hyperarousal understanding of insomnia remains to be investigated and will be a focus of the current thesis.

Additionally, available evidence suggests that sleep, arousal and attention have underlying neurochemical and anatomical systems that overlap. Therefore, it is perhaps not surprising that problematic sleep and attention-deficit/hyperactivity disorder (ADHD) have been found to be highly comorbid. The relationship between them is a highly complex one and subject of several reviews (Cortese et al., 2009; Cortese et al., 2006; Gruber, 2009; Kirov & Brand, 2014). Children with ADHD experience more bedtime resistance, sleep onset difficulties and frequent night awakenings (Kirov & Brand, 2014; Owens, 2009). However,

the direction of influence has been difficult to elucidate, as it is probable that the experienced sleep loss from sleep disruption may contribute to further manifestation of their cognitive, attentional and behavioral symptoms. Some have reported bidirectional relationships between problematic sleep and externalizing problems in childhood (Quach et al., 2018), but not in adolescence (Pieters et al., 2014). However, others have suggested a bidirectional relationship between impulsive behavior, a core symptom of ADHD, and problematic sleep in adolescence (Bauducco et al., 2019). Whether children with symptoms of ADHD in the community also are at risk for later insomnia due to attentional, cognitive or motoric arousal is unknown, and will be addressed in this thesis.

Cognitive-emotional or physiological hyperarousal forecasting sleep difficulties may also be generated by stressors in the family system. Three longitudinal studies indicate a role for family regularity and family/marital conflicts in later sleep problems (Kelly & El-Sheikh, 2011; Koopman-Verhoeff et al., 2019) and late adolescent insomnia (Gregory et al., 2006a). In a meta-analysis of 12 to 18-year-olds, child sleep onset latency lengthened with a more negative family environment (Bartel et al., 2015). Whether this holds true for insomnia in childhood is unknown, especially when also considering child vulnerabilities (e.g., difficult temperament, neuroticism). The current thesis will investigate the role of such family factors while accounting for possible child vulnerabilities.

1.3.4 Summary of insomnia research and gaps in knowledge

As presented above, few studies have addressed childhood insomnia with diagnostic criteria and clinical interviews in normally developing children and even fewer have followed these children longitudinally. Both prevalence and stability estimates diffuse greatly across studies and there is a paucity of data in middle childhood (7-12 years). Regarding determinants of insomnia from preschool to adolescence, there is several issues need to be determined.

First, because insomnia looks to be moderately heritable and have genes that overlap with mood (and potentially other) disorders, it would be ideal to be able to control for the effect common genes when examining potential environmental or trait-like predictors of childhood insomnia. A novel statistical method allows us to partly do this, by adjusting for all unmeasured time-invariant confounding (e.g., genes, unchanging environmental/social factors). This enables us, while acknowledging the uncertainty caused by time-variant

confounding being at play, to investigate whether the potential stability of insomnia is attributable to persistent vulnerability (e.g., genes) or previous insomnia.

Second, and perhaps most notable is the lack of coherence between theoretical frameworks and subsequent empirical investigations. Theoretical models of adult insomnia point to a disorder of hyperarousal, and preliminary findings link cognitive-emotional and physiological hyperarousal to problematic sleep in children and adolescents. Phenotypes of hyperarousal may be the underlying vulnerability in studies reporting stress in family or social (school) settings to predict later insomnia. In sum, few attempts have been made to measure trait-like, behavioral aspects or environmental stressors linked to hyperarousal when trying to predict insomnia development or persistence from preschool to adolescence.

1.4 Aims of the thesis

Based on the literature and knowledge gaps outlined above, the overall aim of the current thesis was to investigate the (1) prevalence and (2) stability of diagnostically defined insomnia from preschool to adolescence, as well as to (3) review the literature in order to investigate possible determinants of insomnia from preschool through adolescence, and (4) empirically test child, parent and family predictors of insomnia while adjusting for time-invariant confounding (most notable genes).

2 Methods

2.1 Paper I and III

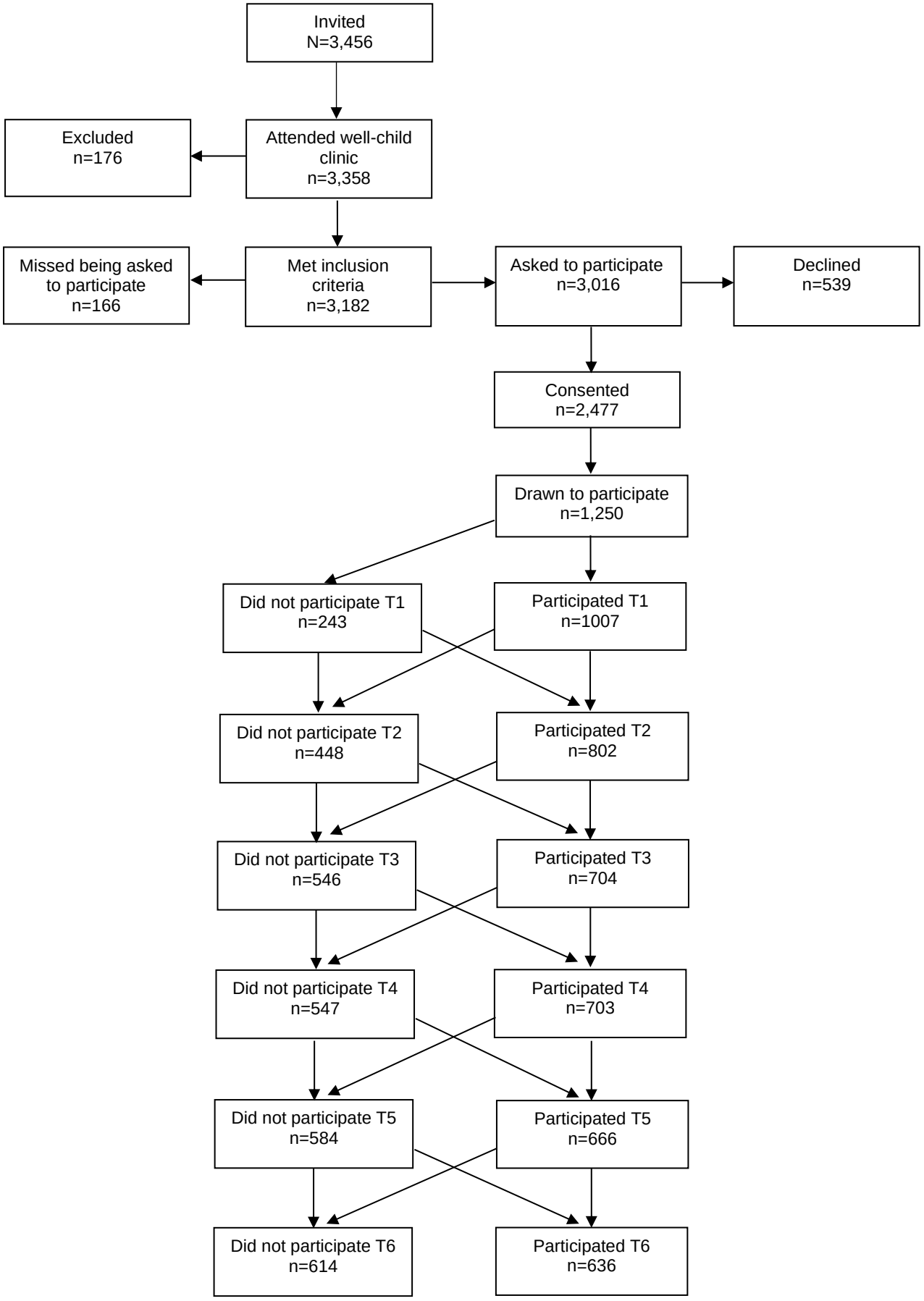
2.1.1 Study design and procedure

The Trondheim Early Secure Study (TESS) is an ongoing longitudinal study from preschool to adolescence that captures mental health, psychosocial development and health behavior (Steinsbekk & Wichstrom, 2018). TESS aims to cover both the breadth and depth of factors of importance for child development, by collecting information from the child, parents, and teachers, through observations, tests, clinical interviews, and questionnaires. Two birth cohorts (born in 2003 and 2004; N = 3,456) in Trondheim, the fourth most populated city in Norway (Statistics Norway, 2019), were enrolled at age 4 (2007) and are examined biennially. The population of Trondheim was, at the time of recruitment, similar to the national average of Norway on several key parameters (e.g., employment rate, educational levels, gross income; Statistics Norway, 2012). Paper I and III uses data from the first 6 data waves (ages 4, 6, 8, 10, 12 and 14 years). Procedure and participation rates are displayed in Figure 1.

2.1.2 Recruitment and screening

Ahead of a mandatory routine health check for all children aged 4 years, parents of the children in the two cohorts (N = 3,456) received a letter with information regarding the study an invitation to participate and a screening assessment for emotional and behavioral problems, the Strengths and Difficulties Questionnaire (SDQ; 31 items) version 4-16 years (Goodman et al., 2000). Parents were encouraged to complete the SDQ and bring it to the health check-up. At the well-child clinics (14 in Trondheim), the parent(s) (n = 3,358 attending; 97.2 %) received further information regarding TESS from the health nurses, in accordance with procedures approved by the Regional Committee for Medical and Health Research Ethics (REK). Parents with insufficient proficiency in Norwegian to complete the SDQ were excluded (n = 176), and some were missed being asked to participate by the health nurses (n = 166). Among the parents asked to participate (n = 3,016), 2,477 (82.1 %) accepted and gave written consent to the health nurses, while 539 declined (17.9 %).

Figure 1. Flowchart of Recruitment and Follow-up



Note. Number of participants at the various assessment points is based on the number of participants invited to participate (n=1,250) minus those who did not participate at the respective measurement points (i.e. T1, T2). Parents with insufficient proficiency in Norwegian to fill out the SDQ were excluded.

2.1.3 Study sampling

With 97.2% of the population attending the well-child clinic, the TESS sample is regarded a community sample (Steinsbekk & Wichstrom, 2018). As the overall aim of TESS was to investigate factors that may positively or negatively affect mental health and psychosocial development, children with such problems were oversampled to increase sample variability and thus statistical power. The probability of being drawn to participate increased with increasing score on total difficulties in the SDQ (20 items; scored 0-2; assessing conduct problems, peer relationship problems, and emotional symptoms). The SDQ is well-positioned to screen for emotional and behavioral problems in 4-year old's (Sveen et al., 2013). However, as Norwegian parents tend to under-report or under-recognize emotional problems on the SDQ (Heiervang et al., 2008), the overall cut-offs points were therefore lowered as well. We divided the children in the following four strata based on their SDQ total difficulties score: 0-4 (44.2% of the population), 5-8 (29.5%), 9-11 (18.5%), and 12-40 (7.8%). The drawing probability within the respective strata was 37%, 48%, 70% and 89% and the draw of a total of 1,250 children was conducted with a random number generator. Of these, 1,007 (80.6%) participated at baseline.

2.1.4 Data collection procedure

A few weeks after the well-child clinic visit, the parent and child were invited to the university clinic at the Norwegian University of Science and Technology for test and interviews. At the clinic the families were assigned two research assistants (one being dedicated to the child, the other to the parent), who had (i) at least a bachelors degree in relevant field, (ii) extensive experience working with children and families, and (iii) substantial training in diagnostic interviewing and coding (n = 7). The test day involved questionnaires, interviews, parent-child cooperative tasks, anthropometric measures, cognitive tests, and accelerometer handouts, and normally took 4-5 hours in total.

Completed questionnaires from the day care- or schoolteacher who knew the child best were obtained in the weeks after the test day, if the parents gave their permission to collect such information. The test day procedure described here was repeated biennially from September 2009 to August 2019 (T1-T6), although the content of the testing differed (e.g. developmentally appropriate measures). The same research assistants met with the family at every assessment point, if possible.

2.1.5 Participants

Unweighted sample characteristics (n = 1,007) at baseline are displayed in Table 1. There was an equal balance between boys and girls (49.9%) and the mean age at each wave was as follows: T1: M_{age}=4.59, *SD*=.25; T2: M_{age}=6.72, *SD*=.19; T3: M_{age}=8.79, *SD*=.23; T4: M_{age}=10.51, *SD*=.17; T5: M_{age}=12.50, *SD*=.14; T6: M_{age}=14.35, *SD*=.16. The sample, adjusted for stratification, is representative of the Norwegian population regarding parents' level of education (Statistics Norway, 2012) and family variables (The Norwegian Directorate for Children, 2017), except for a higher divorce rate (7.6% vs 2.1%; Statistics Norway, 2017). The differences in rates of occupational categories between the sample and the city were negligible, below 3.6% (Steinsbekk & Wichstrom, 2018).

Table 1. Sample Characteristics from T1 (age 4 years) of the TESS (*n* = 1007)

Characteristic	**%**	***n***
Sex of child		
Male	50.5	507
Female	49.5	497
Sex of parent informant		
Male	15	149
Female	85	845
Ethnic origin of biological mother		
Norwegian	92.4	908
Western Countries	3.3	32
Other Countries	4.3	42
Ethnic origin of biological father		
Norwegian	90.5	887
Western Countries	6.2	61
Other Countries	3.3	32
Biological parents' marital status		
Married	54.7	536
Cohabitating	33.6	330
Divorced/Separated	9.8	96
Other	1.9	19
Informant parents' socioeconomic status		
Leaders	5.7	54
Higher professionals	25.5	242
Lower professionals	39.3	374
Skilled workers	25.8	245
Farmers/fishermen	0.5	5
Unskilled workers	3.2	30
Households' gross annual income		
0 – 225' NOK (0 – 25' USD)	3.4	33
225' – 525' NOK (25' – 59' USD)	18.3	180
525' – 900' NOK (59' – 102' USD)	51.8	509
> 900' NOK (> 102' USD)	26.5	260
Childcare		
Official day care center	94.8	942
Other	5.2	52

Note. Unweighted sample characteristics. Note that there are missing data and that percentages are without missing data. NOK to USD exchange rate from 29th November 2020.

2.1.6 Attrition rate

Those who did not attend at baseline albeit consenting to participate at the well-child clinic (and drawn to participate) did not differ from those who attended regarding SDQ-strata group classification or sex (Steinsbekk & Wichstrom, 2018). In general, there is low selective attrition in TESS (Steinsbekk & Wichstrom, 2018). As all non-participating families were re-invited at later measurement points unless withdrawn, attrition in Paper I and III was defined as not participating at the data wave in question. Attrition (i.e., not participating) was therefore examined by regressing attrition on every study variable on the preceding time points. In Paper I being a boy predicted attrition at age 10 (OR=1.39, 95% CI 1.07 to 1.81), 12 (OR=1.31, 95% CI 1.01 to 1.69) and 14 years (OR=1.40, 95% CI 1.09 to 1.80). However, each of these predictions explained a negligible part of the variance in not-attending (R^2 values <1%). In Paper III attrition (i.e., not participating at that timepoint) was unsystematically predicted by symptoms of ADHD, low emotion regulation, poor family functioning, insomnia and male sex, and are displayed in Table 2. Each R^2 value were <5%.

Table 2. Significant Attrition Predicted by Study Variables.

Attrition	**OR**	**95% CI**
Age 8 years		
Symptoms of ADHD (age 4 years)	1.08	1.01, 1.15
Emotional regulation (age 6 years)	0.57	0.35, 0.94
Age 10 years		
Symptoms of ADHD (age 6 years)	1.09	1.02, 1.17
Symptoms of ADHD (age 8 years)	1.11	1.01, 1.23
Emotional regulation (age 6 years)	0.47	0.29, 0.76
Male sex	1.39	1.07, 1.81
Age 12 years		
Symptoms of ADHD (age 6 years)	1.07	1.00, 1.14
Emotional regulation (age 6 years)	0.46	0.29, 0.73
Male sex	1.31	1.01, 1.69
Age 14 years		
Symptoms of ADHD (age 12 years)	1.13	1.02, 1.25
Emotional regulation (age 6 years)	0.40	0.26, 0.63
Male sex	1.40	1.09, 1.80
Insomnia (age 12 years)	2.75	1.20, 6.31
Family functioning (age 12 years)	2.88	1.48, 5.58

Note. Attrition defined as not participating at that timepoint. The R^2 values were all <5%.

2.1.7 Measures

2.1.7.1 Insomnia

The Preschool Age Psychiatric Assessment (PAPA; Egger & Angold, 2004) is a parent-reported developmentally appropriate semi-structured diagnostic interview for children aged 2 to 7 years old and was therefore administered at baseline and T2 (TESS participants being 4 and 6 years old, respectively). PAPA is a revised version of the Child and Adolescent Psychiatric Assessment (CAPA; Angold et al., 1995) which is appropriate for older children and their parents, and hence used at T3-T6 (ages 8, 10, 12 and 14 years). At these latter measurement points, the children and the parents were separately interviewed, hence providing two sources of information regarding symptoms of psychiatric disorders.

Both PAPA and CAPA covers diagnoses according to DSM-IV (American Psychiatric Association, 1994) and captures debut, duration, frequency, and degree of symptoms. These semi-structured interviews contain mandatory and optional follow-up questions, interviewers' probe until they can decide whether a symptom is present or not according to incorporated symptom definitions and instructions for coding, which facilitates diagnostical symptom decisions. The length of the interviews, which were conducted by the research assistants, varies by the extent of symptoms reported and explored. Research assistants (n = 7) had at least a bachelor's degree in a relevant field, a two-week intensive training course in PAPA and CAPA interviews in addition to extended training. Training was conducted by the team who developed the CAPA/PAPA at Duke University.

Included in PAPA and CAPA interviews were questions exploring insomnia symptoms by DSM-IV criteria (see Appendix A). According to DSM-IV (American Psychiatric Association, 1994), insomnia is characterized by (Criteria A) difficulties initiating or maintaining sleep or nonrestorative sleep that lasts for at least one month and (B) causes clinically significant distress or impairment in important areas of functioning. Further, (C) the sleep disturbance does not occur exclusively during the course of other sleep disorders or (D) another mental disorder. Lastly, (E) the disturbance is not due to the direct physiological effects of a substance. As previously mentioned, the DSM-IV insomnia criteria served as a starting point for the applied insomnia criteria in Paper I and II. However, to make the findings more comparable and relevant for present and future research we decided to assimilate the criteria with the updated DSM-5 and ICSD-3. Thus, several notable adjustments were made that diverge from a full DSM-IV diagnosis of insomnia, but which

was justified to approximate, to the degree that our measure permits, the present operationalization of insomnia to the updated DSM-5 and ICSD-3 criteria.

Several considerations were made in this process and will be outlined here. First, because there are no defined cut-offs for the frequency (i.e., times per week) or length (i.e., minutes required) of insomnia symptoms in DSM-IV, some thresholds to quantify insomnia were applied. Although an attempt of quantifying insomnia criteria exists (Lichstein et al., 2003), it is tailored for adults and lean on studies applying former diagnostic criteria (i.e., DSM-IV and ICSD-2). Therefore, we turned to current diagnostic manuals and sleep experts within pediatric sleep medicine when considering appropriate thresholds. Because DSM-5, ICSD-3 and ICD-10 all use the frequency threshold of at least 3 times a week this was applied for all three symptoms in criteria A. To define cut-offs for sleep latency and time awake after sleep onset we drew on the suggested quantitative criteria in DSM-5 of 20-30 minutes and expert consensus recommendations from the Sleep Quality Consensus Panel assembled by National Sleep Foundation (Ohayon et al., 2017). According to these recommendations there is reason for concern for poor sleep quality when sleep onset latency exceeded 30 minutes and when time awake after sleep onset exceeded 20 minutes, from preschool through adolescence. Therefore, thresholds of 30 minutes or more of sleep onset latency and 20 minutes or more of time awake after sleep onset occurring at least 3 times a week were applied. In the clinical setting such quantified criteria may be redundant due to the emphasis on the subjective experience and impairment presented by the patient. However, in the research setting such quantified criteria may contribute to diagnostic consistency, ensuring more homogenous and seriously affected samples. Indeed, diagnostic inconsistency may be the case for epidemiological estimates of insomnia in adults as they vary widely depending on definition (2-48%; Ohayon, 2002). Consequently, studies addressing treatment efficacy may be misleading if criteria for research entry are inconsistent. However, quantifying the symptoms of sleep onset length and wake after sleep onset may have reduced the generatability to clinical practice whether such criteria seldom are employed. Additionally, these cut-offs do not address the symptom of non-restorative sleep. Further, thresholds are no better than their ability to discriminate between actual cases with and without insomnia and such data for insomnia in childhood does not exist.

A second notable consideration regarding the operationalization of insomnia is that the reports of clinically significant distress or impairment in important areas of functioning

(Criteria B) were not insomnia specific in the TESS data collection. The reason for this decision is that it is often difficult to pinpoint with certainty whether it is insomnia or other comorbid psychiatric disorders (e.g., depression or ADHD) that causes impairment, thus parents and children's hypotheses regarding this were not noted and included in the data. Based on the same line of argument we also did not assess whether insomnia was 'primary' or 'secondary' to other mental disorders. The DSM-5 and ICSD-3 do no longer apply this terminology. A separate part of the interview (i.e., not integrated in PAPA/CAPA) addressed reports of clinically significant distress or impairment in one of 11 important areas of functioning (e.g., parent relation, school, play).

Third, TESS measures did not enable to exclude for insomnia being due to (E) substance use, other (C) sleep or (D) mental disorders across the study period. That is, TESS collect data regarding mental disorders, but the PAPA/CAPA interview does not assess these psychiatric conditions up against each other in order to determine which is the most predominant or whether insomnia occurs exclusively during other disorders. Thus, Paper I and III did not account for insomnia explained by other mental disorders, which may have been the case for some individuals. Most research indicates that up to one third of total insomnia cases have comorbid psychiatric disorders (Chung et al., 2014; Ohayon et al., 2000; Roberts et al., 2008), with one study indicating two thirds (Dohnt et al., 2012). To which degree insomnia is a predominant complain or occurs exclusively during these disorders is unknown. The consequences of the inability to exclude insomnia due to substance use is likely to be negligible, as comorbid substance us is rare (<0.5%; Chung et al., 2014; Dohnt et al., 2012). Similarly, others have reported that comorbid sleep disorders (e.g., delayed sleep phase disorder) are a minor issue with negligible effects on estimates of insomnia (Chung et al., 2014; Johnson et al., 2006b; Ohayon et al., 2000) and symptoms of insomnia (Calhoun et al., 2014), although many with sleep disorders also qualifies for insomnia diagnosis (Sivertsen et al., 2013). In all, the inability to exclude by due to substance use, other sleep or mental disorders may have led to a minor overestimation of insomnia cases.

Taken together, our insomnia criteria capture the core diagnostic criteria from DSM-IV insomnia. The criteria were formed to approximate newer diagnostic manuals and operationalized in order to contribute to diagnostic consistency in research with thresholds formed by DSM-5 and sleep experts. The final DSM-IV insomnia criteria applied were as follows: (1) ≥30 min sleep onset latency (SOL) or the use of sleep medication; (2) ≥20 min

time awake after sleep onset (WASO); or (3) non-restorative sleep (insufficiently rested after sleep). The symptom(s) had to be perceived as problematic at least three times a week and had to be present over the previous 3 months, as this is the inquiry period of PAPA and CAPA, and be accompanied by reports of clinically significant distress or impairment in important areas of functioning.

Please note that at age 8 years (T3), CAPA only probed for SOL and WASO data in hours rather than minutes. At that measurement point a ≥1-hour cut-off (opposed to 30 and 20 minutes) were applied and it should therefore be viewed as yielding a conservative, proxy insomnia measure. At the fourth measurement in TESS, when children were 10 years of age, we corrected this data collection flaw by adding an extra sleep interview to the CAPA procedure which enabled us to return to the SOL and WASO criteria of 30 and 20 minutes, respectively. Additionally, this interview expansion also allowed for capturing insomnia more in line with the DSM-5 criteria. Therefore, insomnia according to both DSM-IV and DSM-5 could be provided from age 10 years onwards. More specifically, in DSM-5 early awakenings without being able to return to sleep has replaced the DSM-IV symptom of "nonrestorative sleep". Early morning awakenings is suggested to involve awakening at least 30 minutes before the scheduled time and before total sleep time of 6.5 hours in adults. Our measures did not permit estimates of how many minutes before scheduled times, but questions in the interview (see Appendix A) probed for information if the awakening was earlier than necessary and if the child could have profited from more sleep. To ensure this measure did not capture those who went to bed early and therefore awakened early because their sleep need was met, the symptom of early morning awakenings was combined with total sleep time and had to be 8 hours or less. Sleep duration of 8 hours is below recommended amount in children aged 10-12 years and borderline for age 14 years (Hirshkowitz et al., 2015). Mean sleep duration was used and it was calculated by using typical bed- and risetime subtracted for sleep onset latency and time awake after sleep onset. Moreover, it is stated that symptoms of problematic sleep initiation and maintenance in children may be camouflaged by caregiver intervention (e.g., only falls asleep with parent presence). In the added sleep interview, these new aspects of diagnostic criteria were assessed. Thus, DSM-5 insomnia criteria applied were as follows: (1) ≥30 min SOL or the use of sleep medication or "always" (opposed to "sometimes" or "no") parental intervention when difficulties initiating sleep; (2) ≥20 min WASO or "always" parental intervention; or (3) early morning awakenings without being able to return to sleep and sleep duration of 8 hours or less. The symptom(s) had to be

perceived as problematic at least three times a week and had to be present over the previous 3 months and be accompanied by reports of clinically significant distress or impairment in important areas of functioning.

Combined data from parent and child reports of insomnia symptoms and impairment were used (i.e., parent could report of symptoms while offspring reported of impairment, or vice-versa). This was done because of the expressed concern regarding the use of parents as sole informants in sleep research (chapter 1.1.1) and since the use of multiple informants has been found to increase the reliability and validity of psychiatric diagnoses among adolescents (Cantwell et al., 1997; Jensen et al., 1999).

2.1.7.2 Symptoms of ADHD

The number of parent-reported DSM-IV defined ADHD symptoms in PAPA and CAPA were summed at each measurement point (total of 18 symptoms). The three core problem areas of ADHD include hyperactivity (6 symptoms, e.g., "Always on the go"), inattention (9 symptoms, e.g., "Difficulty organizing tasks/activities") and impulsiveness (3 symptoms, e.g., "Interrupts or intrudes"). The interrater reliability (ICC) between multiple pairs of raters were computed from double coded audio recordings of interviews (9% of PAPA, 15% of CAPA) by blinded raters and were .96 for symptoms of ADHD in PAPA and .90 in CAPA.

2.1.7.3 Emotional reactivity

To obtain a measure of the tendency to be negatively emotionally reactive to internal and environmental stimuli, with regards to the frequency and intensity of experiencing negative affect, we used two different age appropriate measures of emotional reactivity from preschool to preadolescence. At age 4 and 6 we used the negative affectivity dimension of temperament (Rothbart, 2007; Rothbart et al., 2001) in the parent-reported Children's Behaviour Questionnaire short form (CBQ-SF; Putnam & Rothbart, 2006). Negative affectivity was calculated as the mean of the five subscales 'fear', 'sadness', 'anger/frustration', 'discomfort', and 'soothability' reversed, where higher scores indicate more negative affectivity (total of 31 items; [α]=.78-.82). The negative affectivity dimension of temperament (measured at age 4 and 6) has been proposed as a precursor to the Big Five Inventory (BFI; John et al., 1991; Soto et al., 2008) dimension of Neuroticism (Rothbart et al., 2001; Rothbart & Bates, 2006), which we applied at ages 10-14 years. Neuroticism is characterized by an enduring tendency or disposition to experience negative affect (e.g.,

anxiety, anger, irritability and depression) and typically responds poorly to environmental stress (Widiger, 2009). The BFI dimension of Neuroticism consists of short statements (e.g., "Worries a lot") rated on a 5-point Likert scale (from 1=*strongly disagree* to 5=*strongly agree)* where higher scores indicate disposition towards emotional negative affectivity (8 items, age 10 [α]=.60, age 12 [α]=.72, and age 14 [α]=.80). To make the metrics of the two emotional reactivity measures comparable across time, they were converted to z-scores. Please note that because CBQ is applicable to age 7 only and self-reported neuroticism were not measured prior to age 10, no measure of emotional reactivity was included at age 8 (T3).

2.1.7.4 Emotional regulation

The emotional regulation teacher-reported subscale of the Emotion Regulation Checklist (ERC; Shields & Cicchetti, 1997) was used to capture emotional regulation skills from age 6 onwards. The subscale measures how frequently children were able to apply effective and socially appropriate emotion regulation skills (e.g., "Responds angrily to limit-setting by adults") rated on a 4-point Likert scale (from 1=*almost always*, to 4=*never*). Mean scores were computed where higher scores indicate good regulation skills (8 items, [α]=.71-.74).

2.1.7.5 Family Functioning

The parent-reported General Family Functioning scale (Byles et al., 1988) of the Family Assessment Devise (Epstein et al., 1983) was used at all measurement points. The subscale assesses family functioning within six dimensions of family life: problem-solving, communication, roles, affective responsiveness, affective involvement, and behavior control. Parents rated the accuracy of family statements (e.g., "We are able to make decisions about how to solve problems") on a 4-point scale (strongly agree, agree, disagree, strongly disagree), and a mean score was calculated where higher scores indicate worse family functioning (12 items, [α]=.87-.90). The General Functioning scale has previously been used independently as a measure of overall family functioning with favorable reliability and validity (Byles et al., 1988).

2.1.7.6 Marital conflict

The parent-reported Conflicts and Problem-Solving Scales (CPS; Kerig, 1996) was used to assess marital conflict from children's age 6 onwards. The CPS is divided in reports of strategies used in conflict by (i) oneself (43 items; [α]=.88) and (ii) the partner (43 items;

[α]=.90-.91). A higher mean total score represents more frequent use of the following detrimental marital conflict strategies: stonewalling (e.g., withdraw love or affection, 'silent treatment'), avoidance/capitulation (e.g., ignore the problem, leaving the scene, giving in to escape argument), involving the child in marital conflicts (e.g., arguing in front of the child, involving the child in the argument, and arguing about child-related matters), physical aggression (e.g., threatening or inflicting harm), verbal aggression (e.g., yelling, accusing, insulting), and cooperation (e.g., trying to reason, talking about the issue, expressing thoughts and feelings) reversed.

2.1.8 Data analyses / Statistics

All data management and processing were conducted in Stata MP 15 (StataCorp, 2017), including algorithms that calculated whether insomnia disorder was present or not according to the abovementioned criteria. All other analyses were performed in Mplus, version 8 (Muthèn & Muthèn, 1998-2017). As the sample in Paper I and III was stratified by the initial SDQ score (see chapter 2.1.3), analyses used population weights to arrive at estimates representative for the population rather than our sample which was oversampled for mental health problems. This was achieved by weighting data proportional to: the number of children in the stratum divided by the number of participating children in that specific stratum. Thus, children with low initial SDQ-scores were "weighted up" and high SDQ-scorers was "weighted down" to represent more and fewer children than in the sample, respectively, depending on the drawing probability. A robust maximum likelihood estimator was used in both studies, which does not presuppose multivariate normality of residuals and provides corrected error terms with respect to oversampling. We had diagnostic insomnia information on at least one time-point for 1,037 participants, thus forming the analytical sample. Of note, 1,007 participated at baseline but some participants missed the first assessment and participated when older (revisit Figure 1). Missing data were handled by using a full information maximum likelihood approach (FIML; Enders & Bandalos, 2001) under the assumption of missing at random. In FIML, all available data is used to estimate the most likely values for the population parameters in the model. FIML has been shown to produce less biased estimates that alternative procedures, and on par with multiple imputation (Schafer & Graham, 2002). The inversed weighting back to population estimates in combination with FIML strengthens the generalizability of the results from the two studies (Lesko et al., 2017).

2.1.8.1 Paper I

To investigate the stability of insomnia we performed bivariate regressions (i.e., insomnia age X+2 regressed on insomnia age X), because Mplus cannot model a categorical variable as both predictor and outcome, which insomnia was at ages 6, 8, 10 and 12 years. To examine sex differences in both the prevalence of insomnia at each age and the stability between time points, we applied Wald tests to compare the fit of two competing models, one in which prevalence/stability was constrained to be identical in the two sexes and one where prevalence/stability was freely estimated. Significant Wald test results indicate that the competing models are different, hence the parameter in focus differ between boys and girls. To assess the growth of parent and child reports of insomnia prevalence over the study period, we did post-hoc linear growth curve analyses where the slopes reflected yearly mean change (M_{growth}) in insomnia prevalence (i.e., %). The intercepts were set at age 4 years for parents and age 8 years for children and the growth was allowed to correlate with the intercept. Post-hoc Wald tests were also performed to investigate sex differences in symptoms of insomnia to elucidate any sex differences found at disorder level.

2.1.8.2 Paper III

To examine predictors of insomnia in childhood, we applied a random intercept cross-lagged panel model (RI-CLPM; Hamaker et al., 2015). The RI-CLPM, in contrast to traditional cross-lagged prospective designs, disentangles within- from between-person effects (i.e., intra-individual from inter-individual). Within-person processes are often in focus of theories and models addressing the aetiology of disorders, as these offer clues for interventions on the individual level. More specifically, the within-person level examines whether changes in a child's own level of a predictor (e.g., depression) from one's overall level of the predictor during the study period at a given time point predict changes in that same child's (risk of) insomnia relative to that child's overall rate of insomnia during the study period. However, in research the associations between predictors and outcomes are typically tested by approaches combining information from the between-person level (e.g., increase in mean group level depression forecasts group level rise in later insomnia) and the within-person level. It is therefore difficult to determine to what extent parameters are due to between-person effects or within-person effects (Hamaker, 2012). Only statistical models that can separate these two levels of effects may inform within-person processes, which regular cross-lagged panel models does not. Research that relies on a combination of within- and between-person variance may generate conclusions regarding the within-person associations

that are imprecise (i.e., effects may be overestimated, underestimated or reversed; Berry & Willoughby, 2017; Hamaker et al., 2015). The RI-CLPM enables us to separate estimates of within-person and between-person effects. Between-person effects may identify target populations whereas within-person effects may indicate starting points for interventions (i.e., possible mechanisms). In separating these two sources of information, the RI-CLPM implicitly adjusts for all time-invariant confounders. In less statistical terms, it can account for factors that are stable across the study period. Thus, we test whether children high on insomnia also score high on emotional reactivity, ADHD-symptoms, emotion regulation, family functioning, and marital conflict – i.e., a between-person analysis. That said, the main focus is on investigating whether deviations from one's own mean level of these variables predict later deviations from one's own mean level of these variables (i.e., within-person effects). The current model allows for examining all possible directions between the included variables, but the present focus is on the effects of the predictors (i.e., emotional reactivity, ADHD-symptoms, emotion regulation, family functioning and marital conflict) on later insomnia.

In the following section more details are given regarding the statistical specifics of the current RI-CLPM model (Figure 2). Between-person time-invariant effects were captured as we created one latent variable for each construct, with factor loadings from all observed scores (i.e., all time points) of that construct set to 1. Thus, the stability of these observed scores were transferred to that construct's factor. These time-invariant random intercept factors, one for each construct, were allowed to correlate. The within-person cross-lagged effects were modeled by creating one latent variable for each observed score (all variables at all time-points) with a factor loading of 1 and variance of the observed score set to 0. Thus, the variance of the observed scores is transferred to the latent variables and they represent the deviations from that child's average level of the construct at hand. These latent variables at a certain time-point, representing change from the mean level across time, are then regressed on all the constructs latent variables (deviations) 2 years earlier. The exceptions were: 1) emotional reactivity (T4), because there was no measure of emotional reactivity at T3 it was regressed on emotional reactivity 4 years earlier (T4 on T2); and all T2 measures were regressed on only insomnia, emotional reactivity, ADHD symptoms and family functioning at T1, because emotion regulation and marital conflict were not measured at T1. The error terms of all predictors were allowed to correlate at each time point. As a result, at the within level we have estimates of whether deviations from one's mean level of a predictor forecasted

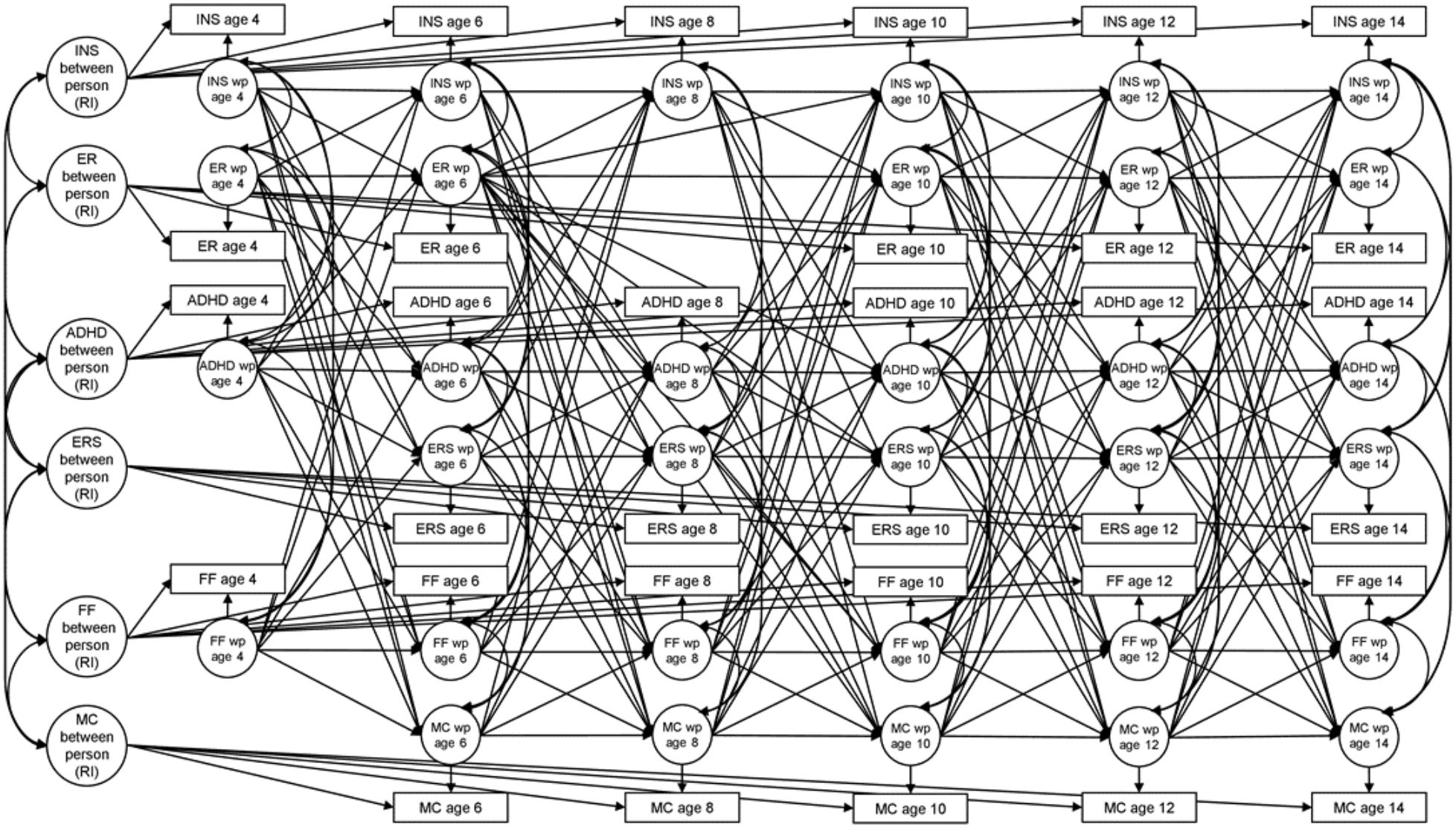
the outcome 2 years later, adjusted for time-invariant stable measured and unmeasured confounders associated with these constructs.

To investigate the importance of predictors across childhood, rather than at age-specific time-points which are more prone to variation across significant thresholds, we compared the fit of a RI-CLPM model where cross-lagged paths were set to be of equal magnitude across ages 4 to 14 to a model with time varying effects using the Sattora-Bentler scaled χ^2 test. The model where predictors were set to have equal importance across childhood showed good fit (χ^2 (267)=341.354, p<0.01, RMSEA=0.016, SRMR=0.034, CFI=0.988, TLI=0.977) and not worse than the model with time varying effects (ΔX^2=16.93, df=22, p=.77). Therefore, RI-CLPM model where cross-lagged paths were set to be of equal magnitude across ages 4 to 14 was used.

2.1.9 Ethics

TESS was approved by the Regional Ethical Committee for Medical and Health Research (REK) in Mid-Norway. All parents of participating children provided consent on their behalf as children below age 16 are not considered legally competent to give consent to research participation. However, from age 12 onwards children were specifically informed about the study. Regardless, the TESS research assistants have focused on the well-being and comfort of the children through the various tasks they are performing throughout the test day to minimize the occurrence of negative experiences for the children. If research assistants are concerned about the participants' wellbeing (e.g., information about sexual abuse), they are obliged to discuss this with the principal investigator who is also specialized in clinical psychology and who will take further steps if needed. If the participant/parent experience emotional difficulties due to the data collection, they are offered a consultation with a clinical psychologist not involved in TESS. The clinical interviews and most of the test day found place in soundproof rooms with one-way mirrors and built in video recording systems at the university clinic. All video tapes and data files were saved on an own server on a local network only accessible for the research group and IT-personnel. In accordance with REK standards, no notable regular reward was given for TESS participation, but children received a small gift at the end of the day (e.g., water bottle, power bank) and parents got a 300 NOK gift-card (about 33 US$). Compensation for travel expenses or parking fee was also given. There was however a family travel gift-card lottery price of 40,000 NOK (4,400 US$) among participating families after each measurement point.

Figure 2. Random intercept cross-lagged panel model (RI-CLPM) of predictors of insomnia



Note. INS=DSM-IV defined Insomnia. ER=Emotional reactivity. ERS=Emotion regulation skills. ADHD=Symptoms of ADHD. FF=Family functioning. MC=Marital conflict. WP=Within-person. RI=Random intercept. Curved arrows are correlations, straight arrows predictions. The six left-most circles marked with (RI) are latent variables for each construct, with factor loadings from all observed scores (i.e., all time points) of that construct (set to 1).

2.2 Methods Paper II

2.2.1 Type of review

Efforts have been made to classify this review among most common types of reviews (Grant & Booth, 2009). In many ways, a systematic review fits well with the systematic search, appraise and synthesis of evidence conducted here, yet the current review falls short on some aspects. No protocol was registered in PROSPERO and the main part of study selection and study quality assessment was conducted by only one reviewer. Therefore, a systematized review may be a more accurate classification, emphasizing the strengths of the systematic search applied but acknowledging it does not adhere to the full criteria for a systematic review. The PRISMA guidelines were adhered to when attainable (Appendix B; Moher et al., 2009). A meta-analysis was not conducted due to the few included studies.

2.2.2 Literature Search

A systematic search process was conducted, using the following databases: Scopus (Elsevier) Web of Science (ISI), Medline (Ovid), PsycINFO (Ovid), and Psychology and behavioural science collection (Ebsco). The search process was performed by two Research Librarians with expertise in searching academic databases at two occasions: 5th of February 2020 by Solvor Solhaug and updated 20th of September 2020 by Magnus Rom Jensen. Search terms are displayed in Table 3 and were grouped in three search strings capturing the main aspects of the review: (i) insomnia in (ii) children and adolescents and (iii) longitudinal studies. Search strings were customized to each database and therefore differed slightly between them (example given in Table 4). To reduce the number of irrelevant hits and increase accuracy of the searches, a proximity operator was used.

Table 3. Search terms to capture the main aspects of the review.

Outcome	Age group	Design/method
(insomnia OR insomnias OR "Sleep onset insomnia" OR "Night waking insomnia" OR "Sleep onset insomnias" OR "Night waking insomnias" OR "Late insomnia" OR "Middle insomnia" OR "Early insomnia" OR "Sleep disorder" OR "Sleep disorders" OR "Sleep disturbance" OR "Sleep problem*" OR "Behavioral insomnia*" OR "Sleep onset latency" OR "Night waking" OR "sleep initiation and maintenance")	(toddler OR preschool OR toddlerhood OR children OR childhood OR preadolescent OR preadolescence OR "School age*" OR paediatric OR pediatric OR youth OR child OR adolescent OR adolescence)	(longitudinal OR follow-up OR "Follow-up study" OR "Follow-up studies" OR prospective OR course OR stability OR continuity OR "Over time" OR forecast OR predict*)

Note. These formed the basis for database-specific search strings in Table 4.

Table 4. Search string from Scopus.

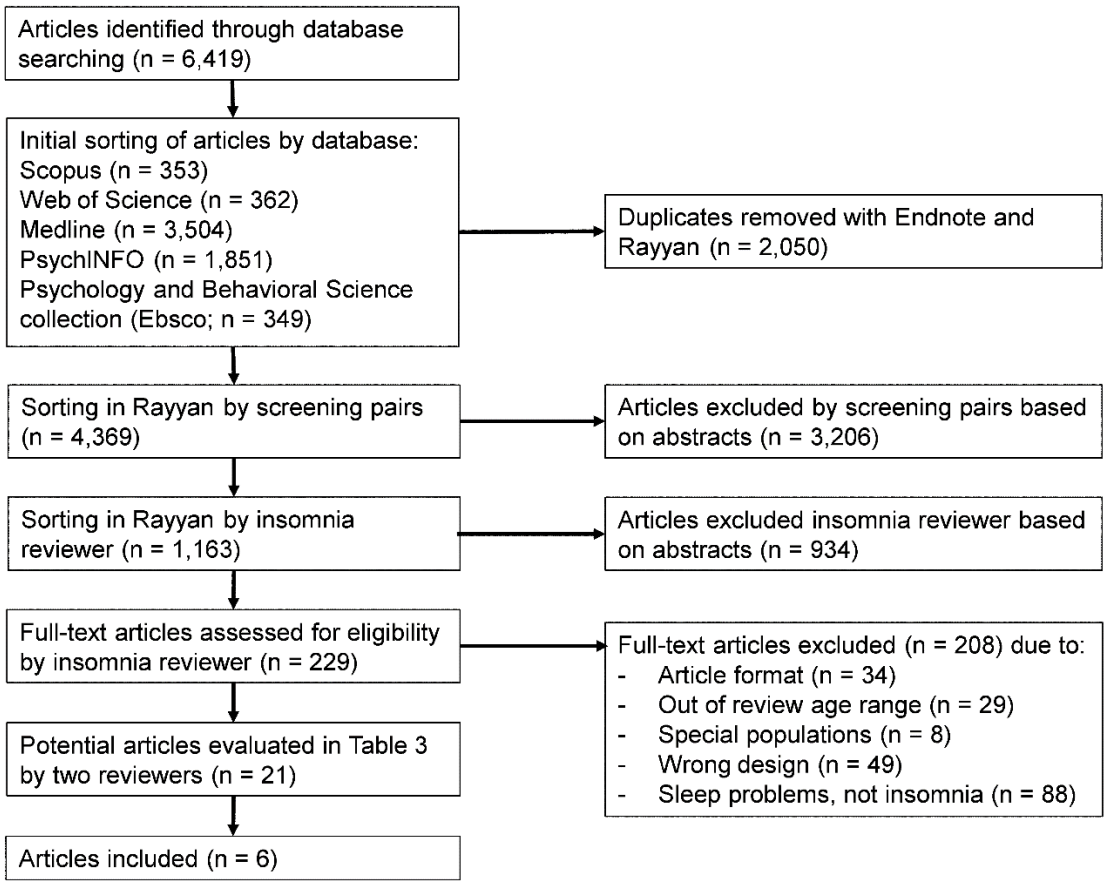
Database	Rationale	Search string
Scopus (Elsevier)	Covers research in the natural sciences, medicine and social sciences. A stricter proximity search was used with Scopus.	TITLE-ABS-KEY ((insomnia OR insomnias OR "Sleep onset insomnia" OR "Night waking insomnia" OR "Sleep onset insomnias" OR "Night waking insomnias" OR "Late insomnia" OR "Middle insomnia" OR "Early insomnia" OR "Sleep disorder" OR "Sleep disorders" OR "Sleep disturbance" OR "Sleep problem*" OR "Behavioral insomnia*" OR "Sleep onset latency" OR "Night waking" OR "sleep initiation and maintenance") W/10 (toddler OR preschool OR toddlerhood OR children OR childhood OR preadolescent OR preadolescence OR "School age*" OR paediatric OR pediatric OR youth OR child OR adolescent OR adolescence) W/10 (longitudinal OR follow-up OR "Follow-up study" OR "Follow-up studies" OR prospective OR course OR stability OR continuity OR "Over time" OR forecast OR predict*))

Note. Different search strategies were made based on the contents and search syntax of the databases. In the multidisciplinary databases proximity search was used to reduce the number of irrelevant hits and increase accuracy for the searches. In Medline, which is a medical database allowing the use of keyword registers and where the keywords are arranged hierarchically, we customized the search to the database structure and content to make use of these features. The search was also expanded with the Explode command, to capture the extended keywords.

2.2.3 Inclusion/exclusion criteria

Included studies were (i) peer-reviewed articles (ii) available in English with a (iii) longitudinal design in a (iv) normally developing children and adolescents with (v) mean age at follow up between 4 and 19 years of age and (vi) diagnostically defined insomnia according to subjective reports as the outcome. Further, included studies captured (vii) predictors of insomnia while (viii) controlling for previous insomnia. Thus, studies were excluded if the sample consisted of an abnormal population (e.g., developmental disorders, mental/medical conditions) due to limited generalizability. Further, intervention studies were excluded as the aim was to summarize predictors of insomnia development and persistence, opposed to the effective components of interventions.

Figure 3. Flow chart of coding and article selection



2.2.4 Coding and article selection

As illustrated in Figure 3, a total of 6,419 identified studies formed the basis of the article selection process. After duplicates were removed (n = 2,050), two thirds of titles and abstracts (n = 2,972) were screened and coded by the two research librarians conducting the first search (Magnus Rom Jensen and Solvor Solhaug). However, due to excused absence of Solvor Solhaug (SSol), the remaining abstracts were screened by Magnus Rom Jensen (MRJ) and another Research Librarian with expertise in review screening, Lene Bertheussen (LB; n = 1,397). All three were drilled in the inclusion/exclusion criteria and blind to each other's coding. Screeners had the opportunity to flag articles as `include`, `maybe` or `exclude` (Tables 5a-5b). Interrater agreement according to weighted kappa with quadratic weights (Table 5c; Cohen, 1968; Warrens, 2013) was fair, both between MRJ and SSol (n = 2,972; 71% agreement; k_w=.29, 95% CI .26 to .32) and MRJ and LB (n = 1,397; 89% agreement; k_w=.36, 95% CI .28 to .44). As illustrated by the contingency tables (5a-5b), there was skewed rater disagreement between MRJ and SSol, and prevalence bias is present for both coding pairs because of the few eligible studies. To illustrate the possible effect of such bias we used a prevalence and bias adjusted Kappa-ordinal scale calculator (Vannest et al., 2016) which yielded estimates of substantial and almost perfect agreement between the two respective screening pairs (.69 and .87, Table 5d).

Table 5a. Contingency table for MRJ and SSol (n = 2,966)

	'Include'	'Maybe'	'Exclude'
'Include'	33	35	103
'Maybe'	60	88	45
'Exclude'	10	599	1993

Table 5b. Contingency table for MRJ and LB (n = 1,397)

	'Include'	'Maybe'	'Exclude'
'Include'	7	7	12
'Maybe'	6	22	76
'Exclude'	11	42	1214

Table 5c. Applied quadratic weights to estimate weighted kappa

	'Include'	'Maybe'	'Exclude'
'Include'	0	1	4
'Maybe'	1	0	1
'Exclude'	4	1	0

Table 5d. Applied weights in the bias adjusted kappa calculator

	'Include'	'Maybe'	'Exclude'
'Include'	1	0.75	0.125
'Maybe'	0.75	1	0.75
'Exclude'	0.125	0.75	1

Note. Value of 0 not available for disagreement in the online calculator, but likely to be negligible for this illustration of the possible influence of prevalence and disagreement bias.

To ensure that no relevant articles were excluded, all articles categorized as 'maybe' or `include` by one screener (n = 1,163) underwent a second review by a coder with insomnia expertise (Jonas Falch-Madsen; JFM; not blinded). This resulted in 229 full-text requests, and by reviewing these, 208 were excluded due to reasons displayed in Figure 3 (e.g., outside age range, wrong design, sleep problems rather than insomnia). The remaining were evaluated (by Silje Steinsbekk and JFM) according to the inclusion criteria and especially considering their compliance to insomnia criteria. Table 6 displays a visualized evaluation of the studies (n = 21) ability to capture the frequency and duration of the three core symptoms of insomnia, along with daytime impairment and adjustment for previous insomnia. This evaluation led to the final inclusion of 6 studies (Table 7).

2.2.5 Data extraction and quality assessment

All included studies reported one or more factors statistically significantly associated with the insomnia outcome, and we report estimates from multivariate analyses. Further, we report all estimates which may inform on the etiology of insomnia, regardless of being presented as covariates, control variables or risk factors. To assess the quality and risk of bias in the included studies we used the Newcastle-Ottawa scale for cohort studies (Stang, 2010), illustrated with stars in Table 7.

Table 6. Articles evaluated according to the inclusion criteria by two reviewers (*n* = 21).

First author (year)	Difficulties initiating sleep	Nocturnal awakenings	Early morning awakenings (EMA) or nonrestorative sleep (NRS)	Frequency per week of symptoms	Daytime impairments in insomnia criteria	Duration	Outcome (adjustment for previous insomnia)
Roberts et al. (2008)	Yes	Yes (two versions)	Both	"Almost every day"	Daytime fatigue or daytime sleepiness	≥ 4 weeks	Insomnia (yes)
Roberts and Duong (2013)	Yes	Yes (two versions)	Both	"Almost every day"	Daytime fatigue or daytime sleepiness	≥ 4 weeks	Insomnia (yes)
Steinsbekk and Wichstrom (2015)	Yes (≥ 30 min)	Yes (≥10min or ≥5 awakenings per week)	NRS	≥ 3	No	≥ 1 month	Insomnia (yes)
Zhang et al. (2011)	Yes	Yes	EMA	≥ 3	No	≥ 12 months	Insomnia (yes)
Tokiya et al. (2017)	Yes	Yes	EMA	"Often or always"	No	≥ 1 month	Insomnia (yes)
Gregory et al. (2006a)	Yes	Yes	EMA	≥ 3	At least "some" interference	≥ 4 weeks	Insomnia (partly)
Foerster et al. (2019)	Yes	Yes	Both	"Often"	No	Not specified	Sleep problems
Taylor et al. (2017)	Yes	Yes	EMA	Not specified	No	Not specified	Insomnia, but criteria not presented (no)
Quach et al. (2018)	Yes	Yes	No	≥ 4	No	'Usually'	Sleep problem factor (yes)
Williams et al. (2017)	Yes	Yes	No	≥ 4	No	'Usually'	Sleep problem factor

Williamson et al. (2020)	Yes	Yes	NRS	≥ 4	No	'Usually'	Sleep problem factor (yes)
Murcia et al. (2019)	Yes	Yes	No	≥ every other day	No	≥ 1 month	Insomnia symptom (yes)
Barclay et al. (2015)	Yes	Yes	Both	≥ 3	Yes	≥ 3 months	Insomnia, no predictors
Sivertsen et al. (2017)	Yes	Yes	No	≥ 3	Sleepiness/tiredness	≥ 3 months	Insomnia, no predictors
April-Sanders et al. (2020)	Yes	Yes	EMA	Not specified	Yes	'Last year'	Insomnia symptoms
Chen and Gau (2016)	Yes	Yes	No	≥ 1 and ≥ 2	No	≥ 1 month during last 6 months	Insomnia symptoms
Fernandez-Mendoza et al. (2020)	Yes	Yes	No	'often' / 'very often'	No	Not specified	Insomnia symptoms
Fernandez-Mendoza et al. (2019)	Yes	Yes	No	'often' / 'very often'	No	Not specified	Insomnia symptoms
Johnson et al. (2004)	Yes	Yes	EMA	Not specified	No	'Usually'	Insomnia symptoms
Liu et al. (2015)	Yes	Yes	EMA	≥ 3	Excessive daytime sleepiness	≥ 1 month	Insomnia symptoms (no)
Shanahan et al. (2014)	Yes	Yes	Both	Not specified	Yes	≥ 3 months	Insomnia symptoms or summed as sleep problems

Note. EMA = Early morning awakenings; NRS = nonrestorative sleep. Studies with outcomes categorized as "Insomnia symptoms" predicted symptoms of insomnia separately.

Table 7. Descriptive characteristics of included studies and quality assessment according to The Newcastle-Ottawa quality assessment Scale.

First author (year)	Setting	Age baseline and follow-up	Prospective sample size (% female)	Selection (maximum 4 stars)	Compar-ability (maximum 2 stars)	Outcome (maximum 3 stars)	Insomnia outcome (% with outcome)	Significant predictors in multivariate analyses adjusted for previous insomnia (other variables in the multivariate model)	Adjusted/Multivariate odds ratio (95% CI)
Roberts et al. (2008)	Houston, US	11-17y and 1y	3,134 (49%)	****	*	**	Incidence of DSM-IV insomnia with (13.9%) and without DI (5.5%)	Of insomnia without daytime impairment: Female sex Age Somatic health limitations Life satisfaction Perceived mental health Depressed mood School stress Father stress Of insomnia with daytime impairment: Female sex Somatic health limitations Life satisfaction Perceived mental health School stress School stress (Ethnicity, family income, perceived somatic health, impact of somatic illness, neighborhood stress, mother stress)	 1.31 (1.07-1.58) 0.72 (0.57-0.91) 1.33 (1.09-1.63) 1.73 (1.34-2.23) 1.46 (1.11-1.93) 1.25 (1.02-1.53) 1.84 (1.39-2.43) 1.58 (1.19-2.10) 1.91 (1.35-2.69) 2.12 (1.45-3.10) 1.65 (1.11-2.45) 1.82 (1.17-2.84) 2.93 (1.61-5.32) 5.01 (2.75-9.13) All insignificant
Roberts and Duong (2013)	Houston, US	11-17y and 1y later	3,134 (49%)	****	*	**	Incidence of DSM-IV insomnia with (13.9%) and without DI (5.5%)	Of insomnia without daytime impairment: Depression symptoms Of insomnia with daytime impairment: Major depression (Age, sex, family income, previous insomnia)	 1.39 (1.14-1.68) 2.31 (1.01-5.28) Estimates not reported
Gregory et al. (2006a)	Dunedin, New Zealand	7y and age 9, 13, 15 and 18y	936 (49%) with insomnia data 18y	**	*	**	Later DSM-IV insomnia with DI (15%)	Family conflict Male sex Health problems 18y (concurrent) (SES, previous sleep problems)	1.42 (1.17-1.73) 0.53 (0.36-0.80) 0.62 (0.46-0.83) All insignificant

Steinsbekk and Wichstrom (2015)	Trondheim, Norway	4.4y and 6.7y	795 (49.9%)	****	**	**	Later DSM-IV insomnia without DI (21.2%)	Symptoms of ADHD Symptoms of ODD Symptoms of MDD (Symptoms of conduct disorder, GAD, separation anxiety disorder, social phobia and specific phobia)	1.08 (1.02-1.15) 1.15 (1.03-1.29) 1.28 (1.07-1.52) All insignificant
Zhang et al. (2011)	Hong Kong, China	(9.0y ±1.8) // (13.7y ±1.8)	1,611 (50.9%)	***	*	*	Incidence (6.2%) or persistence (0.6%) of chronic (12mo) DSM-IV insomnia without DI	Incident insomnia: Paternal education Frequent temper outbursts Feeling tired during daytime Persistent insomnia: Chronic medical disorders (Sex, family income, maternal education, hyperactivity, feeling unrefreshed/ headache after waking up, difficulty getting up in the morning, morning dry mouth)	 2.49 (1.13-5.51) 1.85 (1.16-2.97) 2.17 (1.24-3.77) 10.2 (1.99-52.5) All insignificant
Tokiya et al. (2017)	Japan	7th and 10th graders and two years later	3,473 (not specified); 776 junior (52.2%) and 2,697 senior high students (42.3%)	***	*	**	Incidence of DSM-IV insomnia without DI in junior (7.8%) and senior high students (9.2%)	Incident insomnia (junior high school): Sleep paralysis Poor mental health Incident insomnia (senior high school): Extracurricular learning Mobile phone use Nightmares Poor mental health (Sex, sleep duration, skipped breakfast, habitually consumed coffee, exercise habits)	 3.59 (1.19-10.83) 2.69 (1.41-5.15) 2.10 (1.23-3.60) 1.39 (1.03-1.89) 4.46 (2.36-8.42) 1.62 (1.18-2.21) All insignificant

Note. Approx.= Approximately; mo = Month(s); y = Year(s); CI = Confidence interval; DSM-IV = Fourth edition of the Diagnostic and Statistical Manual of Mental Disorders; SES = Socio-economic status; ADHD = Attention-deficit hyperactivity disorder; ODD = Oppositional defiant disorder; MDD = Major depressive disorder; GAD = Generalized anxiety disorder. Sample size refers those followed longitudinally.

3 Results – overview of papers

3.1 Paper I – "Prevalence and stability of insomnia from preschool to early adolescence: a prospective cohort study in Norway"

In Paper I we aimed to explore the prevalence and stability of childhood insomnia through the following hypotheses: 1) Insomnia prevalence will be U-shaped from age 4 to 14 years, with a dip in prevalence in middle childhood; 2) a female preponderance will arise in early adolescence; and 3) the two-year stability of insomnia will be of medium effect size. We remained open to how the new DSM-5 insomnia criteria would compare to DSM-IV criteria.

In preschool (4 and 6 years) approximately 2.5% of children met the DSM-IV insomnia criteria, whereas prevalence from age 8 to 14 years were 8.3%, 10.2%, 7.9% and 10.5%, respectively. This may look like a sharp increase, however, bear in mind that children were not included as informants before the age of 8 years. Growth curve analyses for parents, which served as informants at all measurements, showed no increase in insomnia prevalence with age (M_{growth}=−0.05, p=0.56). Close to every fifth child had insomnia by DSM-IV criteria at one point during the 10-year period examined (18.7%). Prevalence of DSM-IV criteria were comparable to DSM-5 criteria (age 10-14 years). The prevalence of the specific symptoms of DSM-IV and DSM-5 insomnia showed that no children displayed the new DSM-5 symptom of early morning awakenings.

Sex differences were apparent with DSM-IV, but not DSM-5, criteria. At age 8 years insomnia were more common in boys (11.5%) than among girls (5.4%; *p*=0.006). When comparing overall means for insomnia for the two sexes in early and middle childhood (ages 4–10 years; *p*=0.003), insomnia was more common among boys (8.1%) than girls (4.5%). In contrast, at age 14 years insomnia was more common in girls (13.7%) than in boys (6.7%; *p*=0.009), and marginally more when comparing the mean prevalence of insomnia in girls (11.4%) compared to boys (7.1%) in early adolescence (ages 12–14 years; Wald=4.02, df=1, *p*=0.045).

Both Insomnia by DSM-IV and DSM-5 criteria proved stable. Persistence rates for those with an insomnia diagnosis at one time point who experienced the same disorder two years later were 32–40%, with the transition from age 4 to 6 years being the exception (23%).

Stability effect sizes were moderate to high, with current insomnia increasing the probability of subsequent insomnia (OR) by 5.1 to 15.3 times compared with not having insomnia at the preceding time point. There was no difference in two-year stability between DSM-IV- and DSM-5-defined insomnia.

3.2 Paper II – "Predictors of insomnia in child and adolescent community samples: A literature review"

In Paper II we aimed to synthesize results from longitudinal studies examining predictors of insomnia among children and adolescents in the community. A total of 6 articles from 5 separate studies from 5 different countries were included (US, New Zealand, Norway, China, and Japan), consisting of a total sample of 9,949 children and adolescents aged 6 to 18 years at follow-up. Within sociodemographic predictors four studies reported mixed results for the predictive role of female sex (Gregory et al., 2006a; Roberts et al., 2008; Tokiya et al., 2017; Zhang et al., 2011), while other sociodemographic predictors were largely unrelated to later insomnia and not replicated (e.g., age, ethnicity, family income, SES, neighborhood stress, parental education). Among indicators of mental and somatic health, the most consistent result arises from four studies of 7,402 individuals aged 4 to 18 years and suggests that poor general mental health and depression and markers thereof predict insomnia (Roberts & Duong, 2013; Roberts et al., 2008; Steinsbekk & Wichstrom, 2015; Tokiya et al., 2017). Others reported predictors of insomnia in adolescence to include family conflict (Gregory et al., 2006a) and school stress (Roberts et al., 2008).

Taken together, the results suggest that female sex and mental health (most notably depression) may be involved in insomnia etiology. However, the diversity of predictors studied combined with lack of replication prevent any firm conclusions from being drawn.

3.3 Paper III – "Child and Family Predictors of Insomnia from Early Childhood to Adolescence"

In Paper III we aimed to empirically test child, parent and family predictors of insomnia while adjusting for time-invariant confounding. The following hypotheses were stated: 1) Increased emotional reactivity (i.e., negative affectivity and neuroticism), 2) more symptoms of ADHD, 3) decreased emotion regulation skills, and 4) more stress in the family system (i.e., poorer family functioning and more frequent and detrimental marital conflicts)

will predict later insomnia when previous insomnia and all time-invariant confounding variables are adjusted for. We remained open to whether the stability in insomnia persists when controlling for time-invariant confounding variables.

Random intercept cross-lagged analyses revealed that at the between-person level, insomnia was highly correlated with ADHD-symptoms and emotional reactivity, less so with emotion regulation, family functioning and marital conflict. As hypothesized, at the within-person level (i.e., relative to the child's typical levels across childhood), increased emotional reactivity and ADHD-symptoms and decreased emotion regulation skills consistently but modestly predicted later insomnia, when adjusting for previous insomnia and all time-invariant confounding variables. In contrast to our hypotheses, family functioning and marital conflict did not predict later insomnia across the 10-year period investigated. Previous insomnia predicted later insomnia.

4 Discussion

This thesis has three main aims: 1) To describe the prevalence and stability of diagnostically defined insomnia from preschool to adolescence; 2) to review the literature of possible predictors of insomnia in childhood; 3) and empirically test child, parent and family predictors of insomnia. Results from the thesis aspire to generate new knowledge about the development and etiology of insomnia in the general population, capturing the years from childhood to adolescence. The outcome of the present work can also inform preventative efforts and treatment and indicate when and for whom such interventions are warranted. This is important given the likely detrimental consequences of insomnia in childhood and adolescence. In the present chapter, findings will be discussed in light of theoretical perspectives outlined above, mainly focusing on insomnia as a hyperarousal phenomenon, and with regards to previous empirical findings. Strengths, limitations and suggestions for further research are also discussed.

4.1 Insomnia is prevalent and moderately stable

4.1.1 Insomnia prevalence

The present study adds to previous knowledge by reporting prevalence of diagnosable insomnia with interviews from preschool through early adolescence in the community, which no study has done before. I contrast to stated hypotheses, insomnia prevalence did not show the expected U-shape from age 4 to 14 years, with a dip in prevalence in middle childhood. Paper I did however confirm the two other hypotheses, a female preponderance emerged in early adolescence and insomnia persistence were of medium effect size.

Three former studies have interviewed parents of 4 to 7-year-old children regarding prevalence of offspring insomnia without daytime impairment. These studies reported insomnia to be prevalent in 12-17% and 19-21% of 4- and 6-year-old children, respectively. These rates are far higher than the 2.5% parent-reported prevalence in preschool found in Paper I. The discrepancy may be due to several factors, perhaps the most likely one being that the above-noted studies used insomnia criteria without considering daytime impairments. The insomnia measure may then capture children having one or more insomnia symptoms, rather than children also experiencing the impact on daytime functioning characteristic of *disorders*. We found 14-15% of the preschoolers to have at least one symptom of insomnia in Paper I,

which indeed accord with the findings from the previous studies examining insomnia without daytime impairments.

All research involves uncertainty and in the following I will discuss the uncertainty of the results in Paper I. The estimates revealed in the present work suggest parent-reported insomnia to prevalent in roughly 2.5% of 4 and 6-year-old children (2.6% and 2.4%, specifically). That is, 2.5% is the estimate most compatible with our data (Amrhein et al., 2019). However, considering the confidence intervals, a prevalence ranging from 1.5% to 3.5% is also reasonably consistent with our data, given the assumptions. Further, values outside this range are not incompatible, they are just less suitable, or likely. Likewise, the reported prevalence of insomnia from age 10 to 14 years with both DSM-IV (7.9-10.5%) and DSM-5 criteria (7.5%-12.3%) are compatible with values diverging nearly 3% (i.e., 95% CI) from their most likely estimates, not ruling out prevalence's beyond these values. Thus, there is some uncertainty regarding these estimates, without condemning the quality of Paper I. Such uncertainty should be considered when discussing these results and comparing them with prevalence's (with accompanying uncertainty) from other studies.

The results of Paper I, both by DSM-IV and DSM-5 criteria, suggest joint scores of parent- and child-reported insomnia to be, simply put, prevalent in one out of ten children from age 10 years onwards. Recall that our 8-year measure was a conservative one, using 1 hour threshold of symptoms, so results from 10 to 14 years of age may be more comparable to other studies using interviews to examine insomnia in samples that consists of children *and* older adolescents (Barclay et al., 2015; Chung et al., 2014; Gehrman et al., 2011; Johnson et al., 2006b; Roberts et al., 2006). All these studies include child-reported insomnia prevalence ranging from 7% to 20%, thus fairly in line with compatible estimates of insomnia prevalence reported in Paper I. Among the above cited papers, those applying insomnia criteria most like ours report a prevalence estimate of 9% in children aged 12 to 18 years (Chung et al., 2014; Johnson et al., 2006b). However, because they used child-reported prevalence of insomnia and given the low agreement between child- and parent report, these estimates would probably be higher if a joint score was used, as in Paper I. Among former studies, Roberts et al. (2006) report the lowest child-reported insomnia estimate (7%) in adolescents aged 11 to 18 years, however their prevalence among 11-12-year-olds were as high as 24%. The highest mean estimates are found in the two papers based on the same sample (Barclay et al., 2015; Gehrman et al., 2011). They found child-reported insomnia to be prevalent in 17-19% on

three assessments (ages 8-18) and 11.5% at the last time point (ages 14-18). Please note that these authors used a conservative 1-hour threshold for the durations of insomnia symptoms. In all, these studies indicate that the prevalence of insomnia reported in Paper I, especially when considering they include child- *and* parent-report, is somewhat lower than presented elsewhere. This lower prevalence may reflect the lower rate of psychopathology found in Nordic children (Heiervang et al., 2008; Heiervang et al., 2007; Wichstrøm et al., 2012). Additionally, the estimates may have been affected by methodological aspects, such as the use of semi-structured diagnostic interviews or the applied diagnostic criteria (discussed further in section 4.5.1.3).

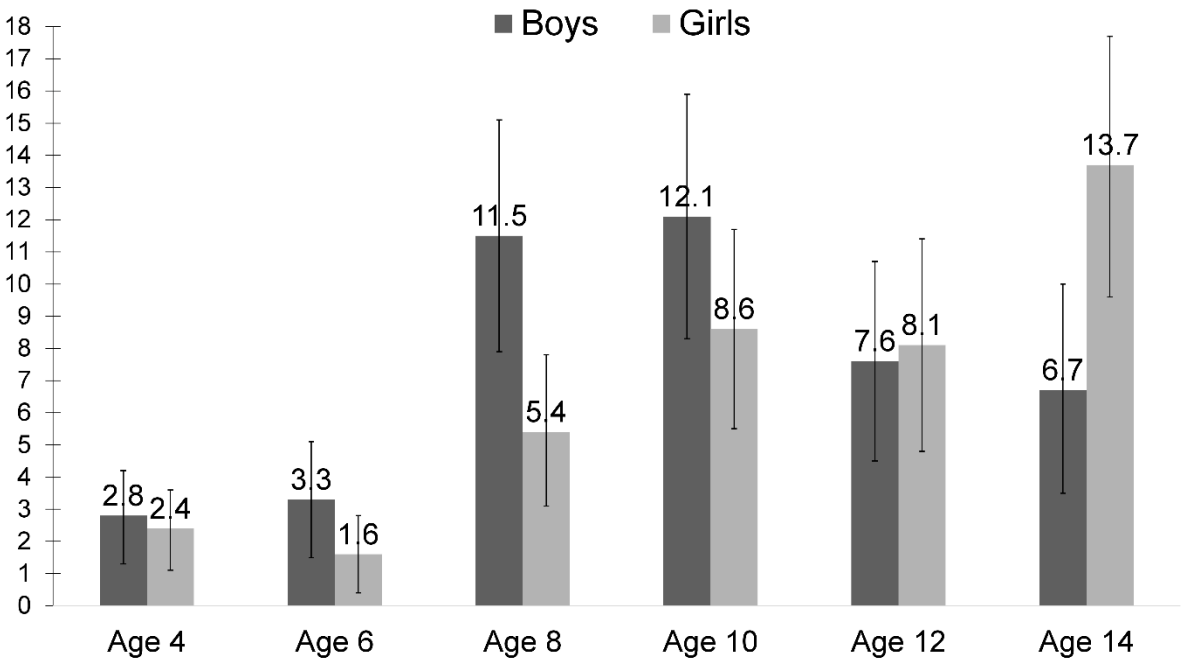
4.1.2 Sex difference in prevalence

We revealed a sex difference in the prevalence of insomnia at two specific measurements in Paper I, with a male preponderance at age 8 years and shifting to a female preponderance at age 14 years. However, bucketing results into 'statistically significant' and 'statistically non-significant' sex differences at specific measurements in a dichotomous way may be considered misleading (Amrhein et al., 2019). For instance, two samples with the same sex difference in prevalence (e.g., boys 10%, girls 5%) may produce p-values of 0.048 and 0.051 depending on the sample size. These two hypothetical studies with identical sex-difference are arguably not categorically different results because of the p-values, but rather the p-value describes the degree of certainty and likelihood of a real-world sex-difference. Therefore, discussing the trends in the data while emphasizing the uncertainty illustrated by upper and lower 'limits' of estimates may be more informative. Taken together, the estimates in middle childhood in Paper I (i.e., not just age 8 years) suggest that boys have more insomnia than girls (Figure 4).

At age 8 years, the sex difference in our data is compatible with, in terms of confidence intervals, as much as 15% of boys having insomnia compared to 3% of girls (i.e., upper end CI boys, lower end CI girls). However, if using the end of the confidence interval where the sexes are most similar (i.e., lower end CI boys, upper end CI girls), both sexes may have an 8% prevalence of insomnia. Although these two presented scenarios are more unlikely than the reported estimates, these are also suitable with the reported data (not excluding further deviations) and depicts the uncertainty of these results. Of note, examining the degree of overlap between CI does not imply significant group differences (i.e., non-overlap equals difference, but overlap does not equal no group difference). Significant group

differences are determined by the distribution of the difference in mean prevalence between the sexes and whether its 95% CI contains the value of zero (Schenker & Gentleman, 2001). Returning to the trend in data, both DSM-IV and DSM-5 suggest a shift in sex difference to pivot around age 12 years and an emerging female preponderance at age 14 years. Analyses of DSM-5 data did not reveal any significant sex differences but may be a consequence of more missing data (than with DSM-IV criteria) and thus reduced statistical power, illustrating one of the perils of mainly glancing through significance levels.

Figure 4. Prevalence of DSM-IV defined Insomnia by sex.



Note. Error bars represent 95% confidence intervals.

In all, the male preponderance reported in Paper I is not reported elsewhere and should therefore be interpreted with caution. The reported female preponderance at age 14 years is in line with the proposed role for the onset of puberty (de Zambotti et al., 2018; Johnson et al., 2006b). Other research in children and adolescents yield mixed results. The previous research on TESS data trend towards girls having more insomnia in preschool, although not significantly more (Steinsbekk & Wichstrom, 2015), and similar results are reported by others (Chung et al., 2014; Johnson et al., 2006b). The estimates in the study by Barclay et al. (2015) move from no sex-difference (modal age 8 years) towards an increasing significant female preponderance with age (modal age 11, 14 and 15 years). Chung et al. (2014) report no sex-difference in prevalence but note that onset age was 2 years earlier in

girls (12 years) compared to boys (14 years), which may further support the puberty hypothesis as girls enter puberty earlier. Both contrasting and supporting this finding,, Johnson et al. (2006b) report boys to have earlier insomnia onset (10 years) than girls (12 years) but revealing that after the onset of puberty, compared to pre menses, girls had a near 3-fold increased risk of insomnia development. Several aspects of puberty might be involved in the sex difference of insomnia. In general, puberty is associated with increased emotional distress (Mendle, 2014), which may arise from both biological (e.g., hormones, neural maturation) and social changes (e.g., relationships/roles, expectations), as well as the interplay between them. Further, there is a tendency towards asynchronous maturation of systems (Mendle, 2014): Early in puberty adolescents show heightened reactivity to socioemotional cues, while regions involved in cognitive control (e.g., prefrontal cortex) develops more independently of puberty. This lack of synchronicity in maturation may contribute to emotion dysregulation and thereby an increased risk of insomnia. However, while these factors may explain why a sex difference occurs in puberty (i.e., because girls enter puberty prior to boys), it does not explain why the same female preponderance is found in adults (Zhang & Wing, 2006). It has been shown that it cannot solely be explained by the higher prevalence of anxiety and depression, nor genetics. Notably though, it has been suggested that a sex difference in symptom endorsement (i.e., ability to detect and report symptoms; Zhang & Wing, 2006) may partially explain why more women than men report to have insomnia. Finally, lending some support for a male preponderance before adolescence, Johnson et al. (2006b) report cumulative incidence of lifetime insomnia to be higher in boys than girls prior to age 11 years. In sum, sex-differences reported in Paper I add to existing data and reveal a switch from male to female preponderance from preschool to early adolescence, which should be subject for replication as well as examining the reasons for this change.

4.1.3 Persistence of Insomnia

This thesis adds to previous knowledge by examining stability across a 10-year period with six biannual assessments, providing data to inform on the persistence of insomnia. The results from Paper I tell two seemingly conflicting yet coherent stories regarding the stability of insomnia across childhood; (i) for the majority (i.e., 60-77%) insomnia diminishes below diagnostic threshold over a two-year period and (ii) insomnia exhibits moderate to high stability (odds ratio 5-15) over a two-year period. The odds ratio results describe how many

more times likely you are as an “insomniac” to have insomnia 2 years later compared to those children who did not have insomnia at baseline. The stability estimates from age 4 to 6 years (OR=15) showed the widest confidence intervals, a likely result of low insomnia prevalence at these two measurements. Here, the results are also compatible with odds ratios as low as 4 and as high as 53. That is, a child that has insomnia at age 4 is 4 to 53 times as likely to have insomnia at age 6 years, as opposed to a child that did not have insomnia at age 4 years. There is less uncertainty regarding the latter periods, yet odds ratios range from 2 to 20.

Although few have addressed the stability of insomnia by means of interviews, their results are largely in line with the results presented here (Roberts et al., 2008; Steinsbekk & Wichstrom, 2015). These report that 43% of preschoolers with insomnia (without assessing daytime impairments) have the same disorder two years later, and a 1-year persistence rate of DSM-IV insomnia in adolescence is reported to be 35%. Further, results from a study on DSM-IV insomnia measured by questionnaire in early adolescence indicate the same pattern. A measure of chronic insomnia (i.e., present for 1 year) was persistent in 15% over a 5-year period (Zhang et al., 2011), but the lower persistence here may be explained with longer time until follow-up and different informants on the two measurements. Also, Combs et al. (2016) report estimates which are not directly comparable as persistence was defined as insomnia at the two last or all three measurements (age 9, 13, and 15 years). Nevertheless, they found 7% of the total sample to have persistent ICSD-2 defined insomnia and because their prevalence was 10%, 19%, and 8%, respectively, this constitute a fairly similar persistence as reported elsewhere. Taken together, this implies that current insomnia provides a risk of later insomnia, suggesting that insomnia may warrant clinical attention.

Although Paper I found insomnia to be stable, this does not necessarily imply that previous insomnia is involved in the etiology of later insomnia. Because insomnia seem to be moderately heritable (Barclay et al., 2015), this stability may well be caused by confounding factors (e.g., persistent vulnerability to insomnia across childhood). Paper III extends previous research by reporting insomnia to be stable even in a more comprehensive model where time-invariant confounding is adjusted for. This finding indicates that consolidating mechanisms beyond stable vulnerabilities (e.g., genes) are in play. Such mechanisms may be in the form of changes in behavior (e.g., daytime napping, going to bed earlier, electronics use in bed) that are efforts to cope with insomnia or the consequences of insomnia (e.g., daytime sleepiness). Alternatively, sleep related mental processes (e.g., exaggerating the

importance of sleep, increased sleep-related worry) may increase arousal levels which further consolidate or escalate insomnia. Such mechanisms are very much in line with Spielman's original model (Spielman et al., 1987) and fits well with the insomnia treatments found to be effective (Blake et al., 2017; Riemann et al., 2017), which is aimed at altering these behaviors and their corresponding mental processes.

4.2 Predictors of diagnostically defined insomnia in the community – a paucity of studies

Paper II adds to previous knowledge by reviewing predictors of diagnostically defined insomnia in longitudinal studies of children and adolescents in the community. The results from the review indicate that female sex and poor mental health (most notably depression), may be involved in the etiology of insomnia. However, only a total of 6 articles from 5 separate studies were identified and the diversity in predictors examined yields uncertainty regarding the role of the suggested predictors.

Perhaps the most consistent finding was that indicators of depression (i.e., both at disorder and symptom level) forecasted insomnia. It is plausible that depressive tendencies may contribute to insomnia through increasing cognitive processes such as pre-sleep rumination or worry (Bartel et al., 2015; Newton et al., 2020) and thereby cognitive-emotional hyperarousal. Further, emotions such as sadness, hopelessness and guilt are symptoms of depression, in addition to thoughts of death (or suicide thoughts) and may produce emotional hyperarousal which hampers sleep. However, several considerations cast doubt on the suggested prospective relationship between depression and insomnia. First, sleep problems are also one among the nine (DSM) symptoms of depression and none of the included studies state that this symptom-overlap has been adjusted for. Therefore, the seeming prediction of insomnia may instead tap into the stability of depressive disorders and symptoms (Morken et al., 2020). Second, a meta-analysis examining the development of more broadly defined sleep disturbance in adolescence found little support for the predictive role of depressive symptoms, although also relying on few studies (Lovato & Gradisar, 2014). Lastly, two of the three included papers addressing the depression-insomnia link stem from the same study and sample, which may misguide the consistency of available data.

Apart from the possible role of female sex which has previously been discussed (section 4.1.2), two other findings from this review should be noted. First, school stress was a

predictor in all three measures of insomnia (with and without daytime impairment, and without comorbid disorders) in Roberts et al. (2008) study of adolescents. A notable strength here was the comprehensive multivariate models used adjusting for several plausible predictors. The indicator of school stress used includes 11 conditions at school (e.g., violence, weapons, drugs, crowdedness, teacher treatment, prejudice and discrimination) and whether the youth perceive these as problematic. Because this measure captures perceived stress, as opposed to observed stressors, it may reflect a vulnerability towards a tendency to perceive environments as stressful (e.g., personality). While it is likely that social school environments that is perceived as stressful may induce feelings of fear and dismay when going to bed before school nights, this novel finding needs to be replicated. Second, because children and adolescents go to sleep at home, the family environment may contribute to both downregulation and increased arousal. The results reported by Gregory et al. (2006a) suggest that levels of family conflict during childhood (7-15 years) may be of importance for later insomnia. Furthermore, a dose-response relationship was found. The greater number of assessments a participant's family scored in the top quartile for conflict, the more likely was the participant to experience insomnia at 18 years. Although no other studies included in our review addressed similar predictors, these results are supported by reviews of risk factors of general sleep problems in children (Newton et al., 2020) and adolescents (Bartel et al., 2015). These reviews identified marital conflict and negative family environment as 'emerging' risk factors, albeit not family problems and parenting stress. However, due to the correlational nature of these reviews and their reporting of unadjusted estimates, they are susceptible to confounding and the direction of influence cannot be determined. Although it seems reasonable that family distress may produce disruptive emotions or rumination/worry in children and adolescents, which in turn may lead to difficulties initiating and maintaining sleep, results summarized here are far from conclusive.

In sum, despite the lack of longitudinal studies and diversity in the predictors examined across studies, the current review serves as a summary of best available evidence. Perhaps the most important finding from Paper II is that the vast number of excluded studies highlights that longitudinal studies of predictors of insomnia defined according to diagnostic manuals are lacking in children and adolescents. Because clinical practice (e.g., prevention and treatment) relies on research, more high-quality prospective studies is vital for our understanding of predictors of insomnia.

4.3 Child predictors of importance for insomnia from preschool through early adolescence

Paper III examined predictors of diagnostically defined insomnia from ages 4 to 14 years. The predictors addressed were mainly from the theoretical perspectives of Dahl's regulation of arousal (Dahl, 1996, 1998) and Harveys psycho-bio-behavioral model (Harvey et al., 2014), focusing on child and family factors which may influence hyperarousal and thereby insomnia. The results indicate a role for child, rather than family, factors: Within-person increase (i.e., relative to the child's typical levels across childhood) in emotional reactivity and symptoms of ADHD and decrease in emotion regulation skills predicted later insomnia during the 10 year period investigated after adjusting for previous insomnia and all time-invariant confounding variables. Thus, although these within-person predictions should be considered modest in terms of effect size, they may inform involved processes in insomnia development and targets of interventions. Below the findings will be discussed in more detail.

Recall that RI-CLPM separates within- from between-person effects and that we applied a model where cross-lagged paths were found to be of equal magnitude, that is, we investigated the impact of the predictor in question across ages 4 to 14 rather than at each time-point separately. This influences our understanding of the results and should therefore be highlighted. First, the between-person results indicate that all the predictors were correlated with insomnia. Although significant, most of these correlations were marginal. The correlations between ADHD-symptoms and emotional reactivity with insomnia were however more prominent. Thus, on a group level across age 4 to 14 years we can expect these factors to correlate, but this does not inform possible etiological relationships. However, the within-person effects illustrate that when an individual deviate from his/hers mean level of a predictor (i.e., mean level from age 4 to 14 years), that predicts the individual's risk of altered insomnia two years after that deviation.

Some notable results from the studies reviewed in Paper II may be linked to the above-mentioned results from Paper III. Focusing on child factors first, Zhang et al. (2011) reported frequent temper outburst at age 9 years to predict incident insomnia at age 15 years. Frequent temper outburst may be an indicator of emotional reactivity or occur as a consequence of poor regulation skills. Further, in preschool Steinsbekk and Wichstrom (2015) found insomnia without assessing daytime impairments to be predicted by ADHD-symptoms. Paper III adds to these results by suggesting a role for problems with

hyperactivity, inattention and impulsivity for diagnosable insomnia, through early adolescence and adjusted for time-invariant factors. In contrast, hyperactivity at age 9 years has been found to be unrelated to chronic insomnia at age 15 years (Zhang et al., 2011). Among family factors, Gregory et al. (2006a) reported family conflict from middle childhood through early adolescence to predict insomnia in late adolescence. Reviews on general sleep problems in childhood and adolescence suggest that marital conflict and negative family environment increases the risk for problematic sleep (Bartel et al., 2015; Newton et al., 2020). However, in Paper III, and in contrast to our hypotheses, we did not find family functioning or marital conflicts to predict diagnosable insomnia across the 10-year period investigated. This may suggest that detrimental family or marital environments do play a role in the development of general sleep *problems*, but their impact is not evident in more strictly defined insomnia *disorder*. Alternatively, former results may be attributed to unmeasured stable third-variables common to parents and children (e.g., genes, response style). Also, some of the child and family factors included in Paper III were correlated, such as emotional reactivity and marital conflict. If emotional reactivity is of importance for later insomnia and marital conflict not, but a study only includes marital conflict when they are correlated, the results may mistakenly suggest a role for marital conflict (i.e., confounding). Recall that Gregory et al. (2006a) did not adjust for child variables in their study that revealed family conflict to predict later insomnia. Therefore, their results are more at risk of confounding, which Paper III in general to a greater degree account for. This is not to say that other family factors not investigated in Paper III may not be of importance for the etiology of insomnia.

The child predictors identified in Paper III may influence the risk of insomnia in several ways. An increase in the tendency towards emotional reactivity may cause the child or adolescent to react more strongly and negatively than usual to stimuli in proximity to sleep onset (e.g., social media, disturbing noises or thoughts, family conflict). Also, such a tendency may heighten the risk of problematic parent-child interactions in proximity to bedtime. The consequence may be an emotionally aroused child or adolescent going to bed and trying to sleep. As opposed to these children's 'normal' level of emotional reactivity, they may now require more downregulatory capacity in order to obtain sleep. The results presented here, that decreased emotion regulation skills predicted insomnia in addition to increased emotional reactivity, indicate that arousal may stem from both higher emotional reactivity and lack of ability to downregulate emotional arousal once it occurs. If unable to downregulate arousal at bedtime, sleep latency is likely to prolong. Over time, associative

learning may consolidate between the sleep setting and arousal and the inability to fall asleep. Further, because individuals with insomnia tend not only to be hyper aroused at bedtime, but also throughout the day and night (Roehrs et al., 2014), it may be that such emotional arousal makes them more prone to night awakenings or negative reactions to these awakenings. Because of their impulsivity and distractibility, children with increased symptoms of ADHD may struggle to comply to regular bedtime routines and avoiding electronic media use. These two aspects of sleep hygiene are included in evidence-based pediatric sleep recommendations (Allen et al., 2016). Bedtime conflicts are also more frequent among youth with ADHD symptomology (Kirov & Brand, 2014), so an increase in ADHD symptoms might increase the likelihood of bedtime conflict and emotionally provoked arousal in proximity to sleep onset.

An alternative view on the three child predictors of later insomnia found in Paper III, is a 'dysregulation phenotype' that has been described over the last decades (Leibenluft, 2011; Stringaris, 2011), which has similarities with disruptive mood dysregulation disorder in DSM-5 (American Psychiatric Association, 2013; Copeland et al., 2013). Characteristics of such a phenotype is severe affective and behavioral dysregulation, including irritability, aggression, 'affective storms', hyperarousal and mood instability. It has been suggested that many children and adolescents with psychiatric disorders display these characteristics and that they often are categorized as suffering from ADHD (Legenbauer et al., 2012). Indeed, children showing such a dysregulation profile or sub-clinical levels experience more sleep disturbances (i.e., range of sleep related problems; Legenbauer et al., 2012), although prospective studies are warranted to further investigate this proposed link.

In sum, the results presented in Paper III indicate that individual deviations in emotional reactivity (increase), ADHD-symptoms (increase) and emotional regulation skills (decrease) influence the risk of insomnia two years later. These results are partly supported by previous literature, but comparisons are difficult due to variations in insomnia definition (e.g., sleep problems, insomnia with or without daytime impairments), measurement methods (e.g., questionnaires, interviews), informants (e.g., parental or self-report), age, time between measurements, outcome (incidence, persistence or later insomnia). Insomnia to be influenced by tendencies towards negative reactivity and poor emotion regulation are perhaps most in line with Harvey's psycho-bio-behavioral model (Harvey et al., 2014). These results

contribute to our understanding of insomnia etiology and adds to the research reviewed in Paper II.

4.4 Implications for research and clinical practice

The aim of this thesis was to examine prevalence, stability, and predictors of insomnia to inform preventive efforts and interventions. To suggest direct practical implications based on the research in this thesis may be premature, but the results presented here combined with what is previously known, may inform future research and health care policy.

The findings indicate that insomnia is prevalent and stable between age 4 and 14 years, suggesting that insomnia warrants clinical attention to prevent the detrimental consequences of the disorder. Clinical attention relies on the ability to detect disorders, it is therefore worrying that studies indicate very few children and adolescents (0.05% to 1.2%) receive an insomnia diagnose in primary care (Meltzer et al., 2010; Meltzer et al., 2014). A similar tendency was reported in a new study among adolescents seeking mental health services in Norway (Hysing et al., 2020). The rate of diagnosed sleep disorders according to registry data was 0.6%, while 34.4% of the same adolescents self-reported insomnia according to DSM-5 defined criteria. In line with the previous understanding in DSM-IV, many health workers may still regard insomnia as secondary to other disorders, or symptoms thereof. Nevertheless, the prevalence and stability of insomnia revealed in the current thesis calls for improved detection of insomnia in order to provide treatment or target preventive efforts. By detecting and successfully treating insomnia, research indicates that these children and adolescents may experience several beneficial effects, including increased health-related quality of life (Combs et al., 2016) and decreased risk of depression (Johnson et al., 2006a; Lovato & Gradisar, 2014; Roberts & Duong, 2013; Roberts et al., 2002), school absenteeism (Bauducco et al., 2015), and suicidality (Goldstein et al., 2008; Wong & Brower, 2012).

The findings presented in Paper I indicate that insomnia is persistent for about a third of children and adolescence only, which triggers the question: For whom? Identifying who is at risk of developing insomnia and characteristics of those with current insomnia who are at risk of persistent insomnia may enable preventive efforts to be targeted at those at risk. As revealed in Paper II, there are alarmingly few studies that can inform such practice, which prevents recommendations of targeting certain children and adolescents at risk. Our own findings from Paper III adds to this sparse foundation of knowledge, by indicating that

individual increases in emotional reactivity and ADHD-symptoms and decreases in emotional regulation skills may serve as such markers of insomnia risk.

If future research can confirm the role of emotional reactivity, ADHD-symptoms, and emotional regulation skills in later insomnia, it is reasonable to assume that interventions aimed at improving these predictors may protect against or prompt remittance from insomnia. For example, although supported by preliminary evidence only, interventions aimed at improving parents' meta-emotion philosophy (i.e., feelings and thoughts about one's own emotions and one's children's emotions) may be appropriate for reducing the effect of emotional reactivity and/or improving children's regulation skills (Gottman et al., 1996). Here, the parents are encouraged to practice emotion coaching, which includes to view their children's negative emotion as an opportunity for intimacy and teaching. As opposed to deny or ignore emotions, the goal here is to validate and label their children's emotions and discuss how to cope with the situation that led to the emotion. In essence, parental meta-emotion philosophy may greet rather than avers negative emotions and use these situations as opportunities to improve emotional understanding and regulation. Related to this, high maternal sensitivity (i.e., supportive, respectful, and hostility reversed) are reported to improve future bedtime problems in toddlers with high negative emotionality, whereas low maternal sensitivity predicted the opposite (Conway et al., 2018). Other interventions have proven promising in helping parents promote emotion regulation skills in children (England-Mason & Gonzalez, 2020) and managing their child's temperament (Cameron et al., 2013), which both may positively impact hyperarousal and thus sleep. Finally, given that Paper III indicates ADHD symptoms to predict insomnia, effective treatment for ADHD may also inform possible interventions, presupposed that the relation with insomnia also applies to ADHD diagnosis (Subcommittee on Attention-Deficit/Hyperactivity Disorder & Management, 2011).

4.5 Methodological considerations – strengths and limitations

Quantitative research is fraud with risk of bias, or systematic error, which may cause observed measures to differ from their true value (Gerhard, 2008; Rothman et al., 2008). It is therefore important to understand, and when possible make efforts to limit, the potential impact of these biases on the results and conclusions made. Similarly, such bias in the design or execution of a review may also impede valid conclusions. In the following section the

strengths and limitations of the present thesis will be discussed, and separately so for the two observational studies (Paper I and III) and the literature review (Paper II).

4.5.1 Validity in Papers I and III

The opposite of bias is validity, which refers to accuracy of study results and inferences drawn, and how well these describe the real world (Rothman et al., 2008). Validity can be challenged through all stages of the research process and can broadly be categorized in internal and external validity. Internal validity refers to whether the results are representative of true associations in the source population (i.e., from which the study population originated) and is typically challenged by three forms of systematic errors; selection bias, confounding, and information bias (Gerhard, 2008; Rothman et al., 2008). External validity refers to the whether the results are representative, or generalizable, beyond the source population and to different settings (Gerhard, 2008; Rothman et al., 2008). Therefore, especially in studies aiming to inform causation, internal validity is often regarded prerequisite for external validity. The following sections will discuss validity concerns in more detail.

4.5.1.1 Selection bias

A general strength of the empirical papers in this thesis is the use of data from TESS, a large-scale well-designed study well-positioned to investigate prevalence, stability, risk factors, and potential causal mechanisms. However, in all studies where some form of selection of participants occurs (by procedure/design or attrition) internal validity may be threatened. Selection bias refers to factors that influence study inclusion (participation or non-participation) and attrition when in longitudinal studies such as TESS (Gerhard, 2008; Rothman et al., 2008). The fact that all potential participants in the target population were invited to participate (2003-2004 cohort, revisit Figure 1) are likely to have reduced selection bias. However, parents with insufficient proficiency in Norwegian to fill out the screening questionnaire were excluded (176 of 3,456 invited) and may have contributed to the low ethnic variation in the TESS sample. Some participants were missed being asked to participate in the study and although likely to be a random error, we do not have data to confirm this. A more likely source to selection bias may be those who declined to participate (self-selection) and while it is not possible at this time to account for the study characteristics of these non-participants, it may be reasonable to assume that parents declining invitation to participate (half a day plus travel) may have less resources or a more stressful everyday life. However, most of those invited accepted to participate (82%) and drop-out after consent did

not differ by child problems (SDQ-score) or sex. Among those participating, the only characteristic found to differ between the sample and the source population (Trondheim municipal) is rates of occupational categories, but these were negligible (below 3.6%; Steinsbekk & Wichstrom, 2018).

Attrition rate in TESS is regarded low and analysis show very little selective attrition (Steinsbekk & Wichstrom, 2018), which strengthens internal validity. Notably though, in the studies included in this thesis, especially Paper III, several variables were linked to attrition (revisit Table 2 and chapter 2.1.6). Attrition was measured as not participating at the specific measurement in question and does therefore not account for when a participant fully dropped out of the study. That may have biased the attrition analyses, for instance emotion regulation age 6 years predicted attrition not only at age 8 years, but all following time-points as well. However, this is likely influenced by that most children dropped out between age 4 to age 8 and the following predictions may be trailing due to who those dropped between age 6 and age 8 years. Nevertheless, attrition data may indicate that boys more frequently missed measurements from age 10 years, and that symptoms of ADHD may have contributed to non-participation two years later. Insomnia at age 12 years may also have affected participation at age 14 years, however this was not found in the first paper indicating inconsistency in data. Nevertheless, these attrition estimates explained a small part of attrition variance (below 5%). All these attrition results point toward a possible underestimation of insomnia prevalence (Paper I). However, although the *level* of insomnia in the population might deviate somewhat from that reported here, prospective *associations* are less likely to be affected by differences in prevalence.

Lastly, the oversampling of those with high SDQ scores are likely to have increased sample variability and thus statistical power, which is important when addressing infrequent features and may have increased the accuracy of such estimates. However, as the children age one can challenge the concept of weighting the data based on how problem scores were at age 4 years. The impression after hours of analysis of the TESS data is, however, that the use of weighting has negligible influence on results. In all, considerable efforts have been made to limit and assess selection bias, yet there may be minor systematic error involved in non-participation (those declining) and attrition (by study variables).

4.5.1.2 Confounding

Confounding may be considered a confusion of effect and is an important issue in observational studies aiming to inform causality (Gerhard, 2008; Rothman et al., 2008). Especially in Paper III, the apparent impact of predictors of insomnia may have been distorted because other factors potentially influencing insomnia are mistaken for, or mixed with, the included predictors. Confounding is hard to counter as there is only so many variables that can be included when predicting infrequent outcomes. Given the likely presence of such omitted variables that may be of importance for insomnia, a unique strength of Paper III is the ability of the applied statistical model (RI-CLPM) to adjust for all time-invariant between-person confounders. Please note that although the time-invariant part of the variable is adjusted for (i.e., stable across time), the time-variant part (i.e., that changes values across time) is not accounted for as long as the variable is not measured and included in the model. Parenting for instance, may include stable components in parents' personality and perhaps overall parenting style. However, parenting may change when experiencing life stressors or positively through parenting programs. Therefore, although time-invariant aspects were accounted for, the time-varying factors not included in Paper III may have influenced the coefficients. Even though time-invariant factors do not change over time, their potential *impact* on insomnia may change (e.g., parenting to be more important in younger ages, etc.). By using RI-CLPM in Paper III we were able to examine whether the stability of insomnia observed in Paper I and in the literature was due to time-invariant factors (perhaps most notable genes) or time-variant reinforcing factors. That is, as the time-invariant components of insomnia were accounted for, it was the time-invariant aspects of previous insomnia that contributed to later insomnia risk. Possible contributors here may be the development of a negative association with the sleep setting or changed compensatory behaviours (e.g., consuming energy drinks to cope with daytime sleepiness). In sum, potential bias from confounding in Paper III are likely to be low compared to most developmental observational research investigating predictors of an outcome but may still potentially bias the results.

4.5.1.3 Information bias

Information bias occurs because our efforts to capture real world concepts are imperfect. Information bias, or measurement error, arises as the instruments used, the participants and the researchers all contribute to more or less accurate attempts to apprehend reality. Focusing on the data collection in Paper I and III, the use of semi-structured

psychiatric interviews to capture insomnia and symptoms of ADHD is both uncommon and a strength, as questionnaires are more typically used. Although there is some subjectivity involved when interviewers consider the presence of symptoms, they are trained and experienced interviewers with possibilities to probe for more information when uncertain. Further, in Paper III validated questionnaires were applied, but some of the measures did display moderate internal consistency by means of Cronbach's alpha, a method with several weaknesses despite its widespread use (Cho & Kim, 2015). Also, the weakest alpha estimates in Paper III were in the measures with fewest items, which probably is a statistical artifact because Cronbach alpha is item-sensitive (Pallant, 2011). Moreover, the overweight of maternal informants is typical in developmental research but may contribute to systematic error if parents differ in their reporting. However, research indicates that parents are in agreement when reporting on their children's sleep (Becker et al., 2017). Lastly, several biases may threaten the validity if the respondent providing all the data is the same (e.g., social desirability, mood states, leniency; Podsakoff et al., 2003). In Paper III the RI-CLPM adjusts for the time-invariant components of potential rater biases. Hopefully, we have countered some of these by using an interviewer as the scorer of symptoms and by using several informants both for insomnia (i.e., parent and child) and its predictors (i.e., parent, child, teacher).

Another aspect that may influence the attempted conceptualization of real-world phenomena is the processing of data, and several considerations regarding the applied insomnia measure should be mentioned. Overall, not strictly complying to the DSM-IV criteria may have hampered the validity of our insomnia measure. The intentions were good, (i) to make the criteria more applicable to children and adolescents, (ii) to conform to the updated DSM-5 and ICSD-3 criteria when possible, and (iii) contribute to diagnostic consistency within research by operationalizing the criteria. However, the resulting criteria may be criticized to fall in between validated diagnostic criteria. Although the prevalence, stability and predictors of the insomnia definition used in Paper I and III are largely in line with insomnia research presented by others, several of the data processing choices may have contributed to both false-positive and false-negative cases of insomnia. For instance, the use of 20-30 minutes of sleep onset latency and wake after sleep onset occurring three times a week may be too simplistic or rigid, as research on symptom thresholds in adults indicate that these criteria depend on each other: as the severity increases (e.g., 60 min sleep onset latency) the frequency required decreases (e.g., once a week; Lineberger et al., 2006). Further, the

questions in the interviews used to assess sleep onset latency and time awake after sleep asks about “normally” or “usually”. The responses are thus likely to represent mean durations. This may constitute a concern given that a child with a mean sleep onset latency of 25 minutes (our criteria was ≥ 30 minutes) may very well have more than 30 minutes sleep onset latency three nights a week, which may have led to an underestimation of insomnia in the current thesis. Further, we used a 3-month period of insomnia symptoms because our measures did not give exact symptom duration at all measurement points. However, this may not have influenced our data much as studies of adolescents have found mean length of insomnia to be two to three years (Chung et al., 2014; Johnson et al., 2006b). The above-mentioned conservative insomnia measure at age 8 years may have led to an underestimation of insomnia and biased the predictor analyses in Paper III. A regarded strength of the insomnia measure is the joint parent- and child-scores. Nevertheless, the possibility of one informant reporting symptoms but not impairment and the other the opposite may lead to false positives. Especially as the impairment measure was not insomnia specific and constituted a separate part of the interview, which increases the risk of including cases where sleep is affected (e.g., awakenings due to nightmares) but due to another condition (e.g., traumatic life event). However, it may be difficult to evaluate which problems that causes impairment, especially given the vast consequences of problematic sleep. Additionally, recall that the inability to exclude insomnia cases due to substance use, other sleep or mental disorders respectively, may have led to a minor overestimation of insomnia cases (methods section 2.1.7.1 Insomnia).

An important adjustment made in DSM-5 was the replacement of the DSM-IV symptom of ‘nonrestorative sleep’ with early morning awakenings with inability to return to sleep. This adjustment was made because nonrestorative sleep can be a consequence of other sleep disturbances and is often seen in addition to the other symptoms of insomnia, and seldom alone. Therefore, the use of this symptom in the current thesis may threaten the validity of the insomnia measure. Further, there is also a concern regarding the new symptom of early morning awakenings used in Paper I. The results indicated that no children had this symptom (age 10, 12 and 14 years) and although this may indicate that our measure fail to capture these problems, it should be noted that estimates elsewhere are also low (< 3%; Roberts et al., 2008; Shanahan et al., 2014). Our lack of cases may be due to the use of a maximum sleep duration of 8 hours in the applied criteria. Because this criterion was based on mean weekly sleep duration it may have excluded children that thrice a week slept shorter

than 8 hours and who struggled with early morning awakening. In contrast, it is also possible that this symptom is nearly non-existent in this age group, as a later circadian preference typically arises in early adolescence (Russo et al., 2007). This may lead to later bedtimes and accumulated sleep pressure that precludes early morning awakenings. In all, we did not find the prevalence of insomnia to differ between DSM-5 and DSM-IV criteria in Paper I, but we have no analyses confirming that it was the same individuals who met the criteria for the disorder. Nevertheless, the applied DSM-IV criteria do resemble the core characteristics of diagnostic criteria and concur with principal research recommendations and is arguably a strong measure of insomnia compared to previous research attempting to capture clinically defined insomnia in childhood and early adolescence.

Our ability to accurately apprehend reality may also have been affected by the way our statistical analyses were conducted. Both papers did not have exact predetermined hypotheses but were formed according to existing theory and literature on insomnia. We further aimed to include potential predictors that we had measured across most of the 10-year period captured. Finally, if possible, we aimed to use measures with different informants to counter common-method bias. In Paper III, we decided to model the predictors so that they had equal importance across the study period, because analyses indicated that the model did not deteriorate. A notable upside with this modelling is that it reduces the number of estimates and thereby lower the risk of false positives (i.e., borderline significant p-values by chance). One potential limitation in the design of TESS, however, is that biennial measurements may be a longer than ideal period when trying to address the effect of predictors and an outcome. Two years is quite a long time when it comes to capture changes in children's development. We may therefore have missed the immediate or short-term effects of predictors on insomnia.

4.5.1.4 External validity

External validity refers to whether conclusions can be applied beyond the study and source populations, to different settings and cultures (Rothman et al., 2008). In this case, whether the results in Paper I and II can be generalized to other populations of 4 to 14-year-old children. One threat to generalization is characteristics of the study and source population. In the TESS study, the sample is largely representable of the Norwegian population (Steinsbekk & Wichstrom, 2018) but contained significantly more divorced parents (7.6%) compared to the national population (2.1%; Statistics Norway, 2017).

Generalization of the findings may also be limited by the fact that the TESS is carried out in Norway, which is a country with low rates of psychiatric disorders in childhood (Wichstrøm et al., 2012). Furthermore, in Norway, housing and children's sleeping environment holds a generally high standard, and in line with evolutionary theory (Dahl, 1996, 1998) one would expect more vigilance and sleep disturbance if perceived as less safe (physically and socio-emotionally). Also, the findings from the TESS may not apply to more ethnically diverse populations or other cultures.

4.5.2 Validity in Paper II

Paper II is not an outright systematic-review and may be categorized as a systematized review according to the typology provided by Grant and Booth (2009). Thus, the paper possesses a greater likelihood of bias compared to those that adhere more strictly to guidelines for systematic reviews (Centre for Reviews and Dissemination, 2016; Higgins et al., 2019).

4.5.2.1 Internal validity

The strengths of Paper II include a comprehensive systematic search and screening procedure with clearly defined inclusion criteria. However, as only one reviewer performed the selection of studies after the initial screening process, the subjectivity involved may have led to selection bias. Although the review only reported results from multivariate analyses which may have reduced the risk of confounding, such reporting is still dependent on the individual study's ability to control for confounding. Therefore, direct comparisons of estimates (i.e., effects) between these studies should be done cautiously. Moreover, comparisons of effects are further complicated by that fact that the magnitude of odds ratios depends on the scaling of predictor. Paper II are likely to have countered some confounding through only including studies that adjusted for previous insomnia, which is important due to its persistent nature shown in Paper I. Lastly, despite efforts to only include studies applying favorable diagnostic criteria for insomnia, the review's effort to capture real world insomnia is no better than its components. It was difficult to pin-point an exact cut-off for inclusion versus exclusion of studies and the results may have been different with other included studies. Some may argue that certain questionnaires are validated to capture diagnoses of insomnia, and as no available research are able to draw the exact lines of the fine-tuned thresholds of insomnia, we relied on studies evaluating the core symptoms of insomnia without summing these.

4.5.2.2 External validity

The few included studies in Paper III may suggest that the generalizability is somewhat uncertain and highly dependent on whether one has succeeded in including the "right" studies. When considering the included studies though, it should be noted that they represent five countries and different cultures and span the ages 6 to 18 years at follow-up. Further, they are all community studies with differences in time between measurements and informants used. Thus, relative to the few included studies, the reported results in the paper may be an acceptable representation of children and adolescents of the world.

5 Conclusions and suggestions for future research

Findings in this thesis suggest that insomnia is prevalent in nearly one in ten children which greatly surpasses those diagnosed with insomnia in health care settings. Moreover, the results suggest insomnia to be moderately stable across the 10-year period investigated (age 4 to 14 years). The present work therefore indicates that insomnia warrants clinical attention. Additionally, as the included literature review illustrated, research on which factors that might contribute to insomnia development in childhood and early adolescence are sparse and inconclusive. The last study in this thesis addressed this gap in knowledge, and indicates a role for child, rather than family, factors. More specifically, increases in emotional reactivity and symptoms of ADHD and decreases in emotion regulation skills predicted later insomnia from ages 4 to 14 years. Although these results should be considered modest in terms of effect size, they may inform involved processes in insomnia development and targets of interventions.

As discussed, there is some uncertainty regarding the validity of the results in the present thesis. The findings should therefore be replicated to determine the reliability of the results. Future research of prevalence and stability of insomnia should aim to apply updated diagnostic criteria. For the validity of the current work, it would further be informative if future research examined prevalence and stability applying different diagnostic criteria, including, but not exclusively, (i) the use of several different thresholds for sleep onset latency, time awake after sleep onset and early morning awakenings, (ii) with and without insomnia specific daytime distress or impairment, and (iii) with an evaluation of comorbid disorders and to the degree insomnia is a predominant complain. Moreover, more high-quality prospective studies are vital for our understanding of predictors of insomnia. As highlighted in this thesis, there is currently gaps both between theoretical models and empirical research, and between empirical research and clinical practice. These gaps need to be further illuminated. Research on potential predictors should arise from theory, and the current thesis strengthens the case for indicators of hyperarousal. Future work should thrive to measure insomnia more in line with clinical practice.

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Papers I-III

Appendices