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Child and Family Predictors of Insomnia from Early Childhood to Adolescence

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Abstract

Background: Insomnia is prevalent among children and adolescents and is associated with a wide range of negative outcomes. Knowledge about its determinants is therefore important, but due to the lack of longitudinal studies, such knowledge is limited. The aim of the present inquiry is to identify child and family predictors of future pediatric insomnia within a psycho-bio-behavioral framework.

Methods: A representative community sample (*n*=1,037) was followed biennially from 4 to 14 years of age (2007–2017). Insomnia was defined based on the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV) criteria and was diagnosed by a semistructured clinical interview of children (from age 8 years of age) and parents (all ages). Predictors included parent ratings of child emotional reactivity, family functioning, and marital conflict; self-reports of personality; and teacher-rated emotion regulation skills.

Results: Random intercept cross-lagged analyses revealed that within-person increases (i.e., relative to the child's typical levels across childhood) in emotional reactivity and decreases in emotion regulation skills predicted insomnia diagnosis two years later from ages 4 to 14 after adjusting for previous insomnia and all unmeasured time-invariant factors. Previous insomnia was the strongest predictor of later insomnia, whereas family functioning and marital conflict did not predict insomnia.

Conclusions: Increases in emotional reactivity and decreases in emotion regulation skills predicted insomnia above and beyond all unmeasured time-invariant factors and could be targets for interventions. Previous insomnia predicted later insomnia, thereby underscoring the importance of detecting, preventing, and treating insomnia at an early age.

Keywords: Sleep Initiation and Maintenance Disorders; Temperament; Emotion Regulation; Longitudinal Studies; Neuroticism

Abbreviations

CAPA	Child and Adolescent Psychiatric Assessment
CFI	Comparative Fit Index
CI	Confidence Interval
DF	Degrees of Freedom
DSM	Diagnostic and Statistical Manual of Mental Disorders
ICC	Intraclass Correlation
M	Mean
OR	Odds Ratio
PAPA	Preschool Age Psychiatric Assessment
RI-CLPM	Random Intercept Cross-lagged Panel Model
RMSEA	Root Mean Square Error of Approximation
SD	Standard Deviation
SDQ	Strengths and Difficulties Questionnaire
SRMR	Standardized Root Mean Square Residual
TESS	Trondheim Early Secure Study
TLI	Tucker-Lewis Fit Index

1 Introduction

Insomnia is the most common sleep disorder in childhood [1-6]; it evinces some continuity over time [2-4] and is associated with impaired health-related quality of life [2] and a risk of developing psychiatric symptoms [4, 7]. To aid preventive and treatment efforts, the determinants of insomnia need to be identified. Predictors of general sleep problems and symptoms of insomnia in childhood have been examined [8], but no previous study has investigated more comprehensive etiological models of diagnosable insomnia that include factors from different levels of influence across childhood. Accordingly, the aim of the present inquiry was to identify child- and family-level predictors of insomnia from age 4 to 14 within a psycho-bio-behavioral framework.

1.1 A Psycho-Bio-Behavioral Model of Insomnia

Harvey et al. [9] proposed a psycho-bio-behavioral model of insomnia in adults that provides empirical support for a vulnerable phenotype of hyperarousal. Specifically, the model suggests that individuals with certain genotypes and personality traits (e.g., neuroticism) have stronger psychobiological responses to stress (i.e., stress reactivity) and consequently experience more cognitive, emotional and/or physiological arousal than others. Because arousal is incompatible with sleep [10], such a phenotype is prone to the learned negative associations that characterize individuals with insomnia [11] (e.g., between the sleep setting and pre-sleep arousal). Difficulties with arousal regulation has been proposed by Dahl [10] as a contributor to problematic sleep in children. According to this evolutionary perspective, sleep and vigilance are conceptualized as opponent processes in a larger system of arousal regulation that on a moment-to-moment basis is influenced by attentional and emotional threats, demands or experiences. As it has been adaptive

for sleep to occur with minimal need for vigilance, human safety from predators was organized through protective social groups [12]. The social-emotional context, that is feelings of social connectedness and its related emotions, have therefore been connected to the sense of safety required for sleep.

Notably, however, the above-cited research does not discuss whether these potential mechanisms also apply to childhood insomnia. Furthermore, individual differences in the ability to downregulate arousal are not accounted for. We argue that insomnia is affected not only by the sensitivity to stress that produces arousal but also by the ability to deal with stress-induced arousal once it occurs. In the present work, we add to the existing research by examining how factors potentially increasing and decreasing arousal impact the development of childhood insomnia.

1.2 Developmental Considerations

In adapting Harvey et al.'s [9] model to childhood insomnia, some developmental considerations should be noted. Temperament is viewed as a prototype of personality, and the emotional reactivity dimension of temperament is highly similar to neuroticism [12]. Children high in *emotional reactivity* are predisposed to react to stressful stimuli with intense and enduring negative emotions (e.g., fear, anger, sadness). Such children are also at increased risk of bedtime resistance and poor sleep hygiene [13]. Although some research reports emotional reactivity to be associated with problematic sleep in childhood [5, 14, 15], other studies have revealed mixed results [16, 17]. Further, research on the impact of reactivity on diagnosable insomnia is lacking, and we therefore aim to fill this gap.

Emotion regulation denotes the ability to (down)regulate emotions, and thereby arousal, once they occur. Here, we aim to investigate whether this skill may protect against childhood insomnia. Preliminary findings among children (from infancy to 9 years) and adolescents (13-18 years) have suggested that poor emotion regulation is associated with general sleep problems [18, 19]. Again, whether this holds for insomnia remains to be investigated.

Children sleep in their homes. The home environment, especially the emotional climate within the family, may help children downregulate, thus facilitating sleep initiation when there is a sense of safety. However, a sense of threat increases arousal [10]; hence, a dysfunctional or conflictual family environment may act as a stressor and negatively impact sleep. Research does indicate that marital conflict [20, 21] and a lack of family routines [22] predict global sleep problems in children and that family conflicts forecast insomnia in late adolescence [23]. We extend this research by investigating whether *family functioning* and *marital conflict* predict diagnostically defined insomnia across childhood.

In sum, there is a lack of longitudinal studies on diagnosable insomnia in childhood that examine predictors at the child and family levels. We aim to remedy these shortcomings by using data from a representative community sample of children biennially assessed for insomnia through clinical interviews from age 4 to 14.

1.3 Methodological Considerations

Most research on the etiology of childhood insomnia, including that reported herein, is observational and is thus subject to unobserved confounding. Recent advances in within-person analysis of panel data provide the opportunity to adjust for one class of confounders whether they are known or not, namely, time-invariant confounders (i.e., those that do not change their

value over the observation period, e.g., sex, genetics) [24]. Even though time-varying factors may still influence the results, it should be noted that most time-varying variables (e.g., parenting, life events) show some stability over time, and these time-invariant aspects of unmeasured time-varying variables are also adjusted for in within-person analyses. Of note, when adjusting for all unmeasured time-invariant factors in within-person analysis, between-person effects are separated from within-person effects [25]. Between-person effects are stable differences between individuals over time (i.e., the 'rank order' of their average level), such as the variability in the overall *level* of emotion regulation skills or insomnia *across individuals* over time. Within-person effects, on the other hand, denote interindividual *changes* (temporal deviations) in a person's own average score over time, such as his/her variability in emotion regulation, thereby using the person as his or her own control. Common methods of analysis (e.g., cross-lagged panel models or multiple regressions) typically mix between- and within-person information, resulting in an uninterpretable blend of effects [26] and making it difficult to derive clinical implications. Between-person effects are helpful for detecting who is at risk (e.g., those with poorer emotion regulation relative to peers), whereas within-person effects may identify modifiable targets for interventions (e.g., individual increases in emotional regulation reduce insomnia risk) and suggest etiological implications. As our theoretical framework suggests a vulnerable phenotype of hyperarousal, we are especially interested in the individual processes (i.e., within-person effects) that may increase an individual's risk of insomnia.

As others have pointed out [27, 28], the stability of disorders may—in principle—stem from two different processes with differing implications for treatment and prevention. In the present case, (i) insomnia may contribute to future insomnia, for example through learning principles (e.g., rises up after going to bed, watches TV with parents – positive reinforcement) or

management efforts which aim to reduce insomnia, but on the contrary contribute to maintain it (e.g., going to or being put to bed early) or (ii) the stability of insomnia may be attributable to stable risk factors operative across development (e.g., genes [29]). If the previously reported partial stability of insomnia across childhood [3] diminishes or vanishes when time-invariant factors are adjusted for, the second explanation will be supported. However, if the association remains, this will support the view that insomnia may contribute to further insomnia [3] and that early intervention may be warranted to prevent insomnia from becoming persistent.

1.4 Hypotheses

We hypothesize that (i) highly emotionally reactive children and those who display low emotional regulation ability will experience more insomnia during the 10-year period than their peers, as will children whose family situation is characterized by poorer family functioning and high marital conflict (i.e., the between-person level, indicating groups of children that may be at insomnia risk). Further, (ii) increased emotional reactivity, decreased emotion regulation, worse family functioning and more detrimental marital conflict will predict an increased risk of later insomnia at the within-person level (i.e., individual fluctuations in the scores of predictors forecast individual changes in insomnia risk) when adjusting for previous insomnia and all unmeasured time-invariant factors. Moreover, (iii) it is unknown whether previously reported stability in insomnia will persist when controlling for time-invariant confounding variables.

2 Material and Methods

2.1 Participants and Procedure

All children born in 2003 and 2004 in Trondheim (N=3,456), Norway, were invited to participate through a letter of invitation sent prior to an ordinary community health check-up at age 4. This invitation included a screening assessment for emotional and behavioral problems, the parent-reported Strengths and Difficulties Questionnaire (SDQ) for 4- to 16-year-olds [30], which has excellent screening efficiency in 4-year old's [32]. Health nurses informed parent(s) about the study and obtained written consent to participate (82.2% of those invited agreed to participate). Children were divided into four strata based on their SDQ scores (0-4, 5-8, 9-11, 12-40) to oversample for mental health problems and thus increase variability and statistical power. The probability of being selected increased as SDQ scores increased (37%, 48%, 70% and 89% in the respective strata). Of the 1,250 families drawn and invited, 1,007 (80.6%) participated at baseline (T1), whereas some did not participate until later time points. Parents and children participated biennially in an assessment day, and the mean ages at these assessment times were 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; and T6: M_{age}=14.35, *SD*=.16. The assessment day involved questionnaires, interviews, tasks, and tests. Completed questionnaires from the child's main schoolteacher were obtained within a few weeks after the time of assessment. Further details can be found in Table S1 (descriptives), Fig. S1 (procedure and participation rates) and the cohort profile paper of the main study [31]. We had diagnostic insomnia information from at least one time point for 1,037 participants (of the 1,053 participants in the main study), thus forming the analytical sample. Adjusted for stratification, the sample is representative of the Norwegian population in terms of the parents' level of education [32] and family variables [33], except for a higher divorce rate (7.6% vs 2.1%; [34]). Attrition was predicted by poor emotion regulation skills at age 6, male sex from age 10 onwards, and insomnia and family functioning at age 12

(Table S2). However, all R^2 values were <5%, indicating low explained variance in attrition. The attrition in the main study is described elsewhere [31]. The Regional Committee for Medical and Health Research Ethics Mid-Norway approved the study.

2.2 Measures

Because the Trondheim Early Secure Study (TESS) started in 2007, *insomnia* was defined by the Diagnostic and Statistical Manual of Mental Disorders (DSM)-IV criteria and measured at ages 4 and 6 by the Preschool Age Psychiatric Assessment (PAPA; [35]), a clinical parent interview that follows a structured protocol. The PAPA is a revised preschool version of the Child and Adolescent Psychiatric Assessment (CAPA; [36]), which was applied to children and parents separately for children aged 8-14 years.

According to the DSM-IV [37], insomnia is characterized by difficulty initiating and/or maintaining sleep and/or a subjective experience of nonrestorative sleep that lasts for a considerable amount of time and causes clinically significant distress or impairment in important areas of functioning. However, in the absence of a cutoff for the required frequency or time in minutes of insomnia symptoms, epidemiological research on insomnia has been challenging. No child-specific insomnia criteria exist in the DSM-IV. We therefore relied on the DSM-5 [38] suggested cutoffs for sleep latency and time awake after sleep onset (20-30 minutes) and expert consensus recommendations from the Sleep Quality Consensus Panel assembled by the National Sleep Foundation [39].

DSM-IV insomnia (coded 1 or 0) was therefore based on the following symptoms: (1) ≥ 30 minutes to fall asleep or the use of sleep medication; (2) ≥ 20 minutes awake after sleep onset; or (3) nonrestorative sleep (insufficiently rested after sleep). A symptom was considered present

if reported by either the child or the parent (i.e., cross-reporting allowed, e.g., child reporting symptom only and parent reporting impairment only). The symptom(s) had to be perceived as problematic at least three times a week over the previous three months and be accompanied by reports of clinically significant distress or impairment in one of 11 important areas of functioning (e.g., parent relations, school, play). At age 8, a ≥ 1-hour cutoff was used for time to fall asleep and time awake after sleep onset because the CAPA probes for duration of symptoms in hours rather than minutes (i.e., more or less than 1 hour). Insomnia data from age 8 years of age should therefore be viewed as yielding a conservative, proxy insomnia measure. We corrected this from age 10 years and onwards by collecting information regarding the abovementioned insomnia symptoms in minutes. In terms of interrater reliability, power analysis revealed that having a projected 7.5% average insomnia rate would require 233 cases to be recoded if the expected interrater agreement (kappa value) was .80 and estimated with .15 precision [40]. To achieve at least this power and precision blinded raters (n = 7) recoded 270 audio-recorded interviews, which yielded an interrater reliability for a diagnosis of insomnia of *k*=.75. Coders had at least a bachelor's degree in a relevant field and extensive experience in working with children and families. They underwent a two-week intensive training course in PAPA and CAPA interviews in addition to extended training conducted by the team who developed the CAPA/PAPA at Duke University.

Emotional reactivity was captured by the negative affectivity dimension of the parent-reported Children's Behavior Questionnaire short form (CBQ-SF; [41]) at ages 4 and 6 years (31 items; [α]= .78-.82). Because the CBQ is not applicable to children aged 8, emotional reactivity was not measured at this time point, whereas from age 10 onwards, the self-reported Big Five Inventory (BFI) dimension of neuroticism [42] was applied (8 items; [α]= .60-.80). Neuroticism

is highly similar to the negative affectivity temperament dimension [12]. Both the CBQ-SF and BFI have acceptable validity for assessing temperament and personality, respectively [43, 44]. Because the CBQ-SF and BFI use different Likert scales (7- and 5-point, respectively), we used conversion to z-scores to make the metrics of the two measures comparable across time.

Emotion regulation was captured by the teacher-reported emotion regulation subscale of the Emotion Regulation Checklist (ERC; [45]) from age 6 onwards (8 items; [α]= .71-.74). The ERC measures how frequently children display socially appropriate emotion regulation on a 4-point Likert scale (from 1= *never* to 4= *almost always*). Mean scores were computed, and higher scores indicate better regulation skills. The ERC correlates with observers' ratings of children's regulation abilities, and it can be reliably differentiated from other emotion-related constructs [45].

The overall level of *family functioning* was measured at all time points by the parent-reported General Family Functioning scale [46] of the Family Assessment Device (FAD) [47] to capture problem solving, communication, roles, affective responsiveness, affective involvement, and behavior control within the family. Statements were rated on a 4-point Likert scale (strongly agree, agree, disagree, strongly disagree), and a mean score was calculated; higher scores indicate worse family functioning (12 items, [α]= .87-.90). The General Family Functioning scale has previously been used independently as a measure of overall family functioning and shows good reliability and validity [46].

Marital conflict was measured from age 6 onwards by the parent-reported Conflicts and Problem-Solving Scales, which capture the strategies used in conflict by oneself and the partner [48]. The mean scores of the following self (43 items; [α]= .88) and partner strategies (43 items; [α]= .90-.91) were used: involving the child in marital conflicts (e.g., "argue in front of the

child(ren)"), stonewalling (e.g., "withdraw love or affection"), avoidance/capitulation (e.g., "change the subject"), verbal aggression (e.g., "make accusations"), physical aggression (e.g., "throw something") and cooperation reversed (e.g., "express thoughts and feelings openly"). Strategies were rated on a 4-point Likert scale (from 1= *never* to 4= *often*); a higher score represents more frequent use of detrimental marital conflict strategies. The CPS has shown good convergent validity with other measures of family conflict [48].

2.3 Statistical Analyses

We used a robust maximum likelihood estimator and a full information maximum likelihood (FIML) approach to handle missing data under the assumption that data were missing at random, as indicated by our attrition analysis (Table S2). FIML is asymptotically equivalent to multiple imputation and provides less biased estimates than complete case analysis [49]. As the sample was screen stratified, the number of children in the stratum was divided by the number of participants in the same stratum to calculate probability weights to arrive at corrected population estimates.

A random intercept cross-lagged panel model (RI-CLPM) was applied to investigate the between- and within-person effects of the four predictors and previous insomnia on later insomnia across the 6 biennial measurements (Fig. S2) [25]. Recall that between-person effects concern stable differences in the level of variables between individuals (i.e., 'rank-order') across time (i.e., the whole study period). In this case, they reflect the extent to which a child's overall level on a risk factor (e.g., emotion regulation), over the study period, relative to that of other children is related to that child's overall rate of insomnia diagnoses across the period relative to that of peers (i.e., a correlation). Such correlations are helpful for detecting who is most at risk

for insomnia in a population compared to others. However, causality only operates at the within-person level: The overall group mean level of a risk factor (i.e., between-effects) does not impact a specific child's risk for insomnia. In RI-CLPM, within-person effects denote time-point-specific fluctuations from the abovementioned mean of each child's score across the whole study period, thus the child acts as his/her own control. In other words, our main focus, the within-person change in insomnia is the difference between a child's tendency towards insomnia diagnosis across the 10-year period and whether or not the child has insomnia at a specific time point. Thus, the within-person effect describes whether a change in one's overall level of the risk factor (e.g., decreased emotion regulation at age 8) predicts whether one is at greater or reduced risk of insomnia two years later relative to one's own average insomnia risk.

The RI-CLPM captures between-person time-invariant effects by correlated latent factors, one for each construct, with the factor loadings of the observed scores at all time points set to 1. In the within-person cross-lagged part, one latent variable is created for each of the variables involved at each measurement point using the observed score as a single indicator with a factor loading of 1. The variance in the observed scores is set to 0, and as a result, the variance is transferred to the latent indicators that capture deviations from the person's average level for the variable at hand. For a more detailed technical description and an illustration of the model, see the online supplement.

3 Results

The bivariate correlations and descriptive statistics for all variables, including insomnia prevalence, are displayed in Table S3. The prevalence and stability of insomnia have been

presented in detail in a previous paper; nearly one in five participants had insomnia at least once between the ages of 4 and 14 years, and one in three retained their diagnosis 2 years later [3].

Recall that we had no hypotheses suggesting that the included predictors were important for certain developmental periods only. To examine whether the strength of the relationship between any of the predictors and insomnia differed across time, we compared the fit of an RI-CLPM model where cross-lagged paths were constrained to be of equal strength across ages 4 to 14 (i.e., equal magnitude of predictor-outcome relationships) to that of a model where the strength of the predictor-outcome relationships was allowed to vary across time. Because the model fit did not significantly worsen in the model with constrained paths (ΔX^2=13.55, df=18, p=.76) and the model fit was good (χ^2 (180)=199.96, p=0.15, RMSEA=0.010, SRMR=0.031, CFI=0.996, TLI=0.992), this model was preferred for parsimony reasons. Please note that the commonly used criteria of good fit between the model and the observed data are RMSEA values ≤ 0.06, SRMR values ≤ 0.08, and TLI and CFI values ≥ 0.95 [50].

3.1 Between-person correlations

Our first hypothesis was that the proposed predictors would be associated with insomnia at the between-person level, which was the case for emotional reactivity and regulation but not family functioning and marital conflict (Table 1). These results indicate that children with higher scores on emotional reactivity and lower scores on emotion regulation reported more insomnia than children with more favorable scores on these predictors across the 10-year period.

Insomnia	Ustd. correlation (95% CI)	p-value	Std. correlation
Emotional reactivity[a]	.021 (.011, .032)	<.001	.573
Emotion regulation[b]	-.005 (-.009, .000)	.026	-.379
Family functioning[c]	.005 (-.001, .010)	.108	.244
Marital conflict[b]	.003 (-.001, .007)	.137	.216

Table 1. Between-person correlations with insomnia from age 4 to 14

Note. Measured biennially at [a]age 4-6 and 10-14; [b]age 6-14; [c]age 4-14. Ustd. = unstandardized. Std. = standardized. These are correlations between factors tapping the constructs across the whole study period.

3.2 Within-person predictions

Our second hypothesis was also partially confirmed. At the within-person level (Table 2), significant cross-lagged effects from emotional reactivity and emotion regulation skills to insomnia indicated that a child's deviation from his or her average predictor score (i.e., 'individual mean') was followed by deviations from his or her average insomnia score two years later. Thus, children who scored higher than they typically did on emotional reactivity and lower than their average on emotion regulation skills were more at risk of insomnia at the following assessment, adjusted for previous insomnia and all unmeasured time-invariant factors. Contrary to our hypothesis, family functioning and marital conflict did not predict later insomnia at the within-person level. Last, insomnia proved stable across childhood and was, as indicated by the standardized values, the strongest of the predictors in the model; increased insomnia at one time point increased the risk of increased insomnia two years later by .20 points.

Insomnia	B (95% CI)	p-value	β
Insomnia[a] (0-1)	.195 (.111, .279)	<.001	.179
Emotional reactivity[b] (z-score)	.017 (.004, .031)	.010	.070
Emotion regulation[c] (1-4)	-.044 (-.089, .000)	.050	-.060
Family functioning[a] (1-4)	.042 (-.008, .093)	.101	.051
Marital conflict[c] (1-4)	-.005 (-.169, .160)	.954	-.003

Table 2. Within-person predictors (range of scale) of insomnia from age 4 to 14

Note. Measured biennially at [a]age 4-14; [b]age 4-6 and 10-14; [c]age 6-14. B= Unstandardized path coefficient. An increase of 1 in the predictor (i.e., a change in score from one's average level that is scale dependent) forecasts an increase in the change in insomnia risk relative to one's average level of insomnia. β = standardized path coefficient. These values are standardized (i.e., z-scores)

and thus comparable across different measures (i.e., scale independent), which allows for comparison of the strength of the associations. The standardized value refers to the SD change in insomnia that will appear when the predictor changes one SD (e.g., .07 SD increase in insomnia risk deviation per 1 SD increase in emotional reactivity deviation).

4 Discussion

In the search for determinants of diagnosable insomnia in childhood, we investigated the between- and within-person effects of predictors from a psycho-bio-behavioral model of insomnia and hyperarousal theory. We found that individuals' deviations in emotional reactivity and emotion regulation skills predicted their risk of insomnia two years later (i.e., within-person effects), whereas family factors (marital conflict and poor family functioning) did not. Moreover, previous insomnia forecasted later insomnia, even when controlling for all unmeasured time-invariant factors (e.g., stable effects of genes [30]). Only emotional reactivity and emotion regulation skills were correlated with insomnia at the between-person level, indicating that children with higher levels of emotional reactivity and poorer emotion regulation skills also tended to report more insomnia than other children across the 10-year period investigated.

Our results suggest that emotional reactivity contributes to the development and maintenance of diagnosable insomnia from preschool to early adolescence (age 4-14). Previous research reported emotional reactivity to be longitudinally associated with more general sleep problems [5, 14, 15], a finding we extend by showing that it also applies to DSM-IV-defined insomnia when controlling for unmeasured time-invariant factors. These within-person results indicated that if a child's emotional reactivity increased from his or her own mean level of emotional reactivity, the child was at increased risk of insomnia two years later. It is possible that

emotional reactivity causes the child to react more strongly to arousing stimuli in proximity to sleep onset (e.g., social media, disturbing noises or thoughts), which requires the child to have a greater capacity to downregulate. The child may not possess this capacity, thus resulting in aroused states that preclude sleep. Furthermore, one might speculate that increased emotional reactivity heightens the risk of problematic parent-child interactions, which may also occur in proximity to bedtime. Consequently, the child may become even more emotionally aroused and bring more difficult emotions to bed, which in turn prolongs sleep onset latency. Individuals with insomnia tend to be hyperaroused not only at bedtime but also throughout the day [51]. Accordingly, a proclivity to arousal might also have effects throughout the night and contribute to nocturnal awakenings—another symptom of insomnia.

As hypothesized, decreased emotion regulation skills at the within-person level predicted insomnia two years later, as defined in the DSM-IV, and children with poor emotional regulation skills reported more insomnia across the 10-year period (i.e., between-person effects). These results expand previous findings in children and adolescents that emotional regulation is longitudinally associated with more general sleep problems [18, 19]. Because such downregulation skills predicted insomnia in addition to emotional reactivity in the present study, arousal running counter to sleep initiation may stem from both higher emotional reactivity and lack of ability to downregulate emotional arousal once it occurs.

Although previous research has found insomnia to predict later insomnia across childhood [3], this does not necessarily imply that previous insomnia is involved in the etiology of later insomnia. It might as well be that what appears to be stability is simply due to stable confounding factors (e.g., persistent vulnerability to insomnia across childhood). Importantly, we here demonstrate that insomnia at one time point increases the risk of later insomnia even when

all time-invariant confounds were adjusted for; a finding consistent with the view that previous insomnia may be part of the etiology of later insomnia. It is possible, as first reported in adults [52], that adolescent insomnia may provoke behavior (e.g., going to bed earlier, daytime napping) or mental processes (e.g., exaggerating the importance of sleep, increased sleep-related worry) that consolidate or further escalate insomnia. For younger children, it is more likely that these behaviors and mental processes are enforced by parents (e.g., earlier bedtimes, exaggerated focus on the importance of sleep).

Neither family functioning nor marital conflict was associated with insomnia at the within- or between-person level. It is possible that because the present study captured a strictly DSM-IV-defined insomnia diagnosis, family distress may contribute to the development of sleep problems [20-23] but not diagnosable *insomnia*. Notably, other parental or family factors not examined herein could be of importance (e.g., parental education [5], stress levels [53], parental presence at sleep onset [54]).

Subject to replication, the presented results support the psycho-bio-behavioral model of insomnia suggested by Harvey et al. [9] and suggest that children who are more emotionally reactive and display poorer emotion regulation skills than their peers might be at risk of insomnia. Furthermore, the results indicate that interventions aimed at improving emotional reactivity and emotional regulation skills in children may protect against or prompt insomnia remittance. Emotional reactivity may initially seem biological and trait-like and thus may seem to be a less likely effective target for interventions. However, research chronicles considerable change at the between-person level in childhood [55], and our within-person findings imply that these child-related factors fluctuate from their mean values over time. Moreover, school-based interventions have proven promising for teaching children temperament-differentiated strategies

for coping with stressors [56]. Finally, the fact that insomnia was the strongest predictor of later insomnia attests to the importance of early identification, and effective treatment is certainly available [57]. These insomnia treatments include strategies that enhance emotional regulation and positive parent-child interactions in proximity to bedtime.

In addition to the strengths of the current study, including the use of a large representative community sample followed up biennially with clinical interviews for ten years and statistical procedures that account for all unmeasured time-invariant factors, there are some notable limitations. First, the PAPA and CAPA apply a three-month period for insomnia symptoms, in contrast to the one-month period used in the DSM-IV criteria for insomnia, which might have led to underestimations of prevalence. The underestimation of prevalence may have been especially prominent at 8 years of age, with our conservative proxy insomnia diagnosis (a 1-hour threshold for sleep latency and night awakenings). Also, the third edition of the International classification of sleep disorders highlights bedtime resistance as a symptom of insomnia [1]. This symptom is not captured in the present inquiry, which is another likely contributor to underestimation, perhaps especially at younger ages where bedtime resistance might be more prevalent [61,62]. Also, the use of parent-report only at ages 4 and 6, in contrast to also relying on child report from ages 8 onwards may have further lowered the prevalence at the younger ages [3]. Conversely, because we were not positioned to exclude other sleep disorders (e.g., circadian disorders, sleep-disordered breathing) that are likely to lead to reports of being insufficiently rested after sleep, insomnia prevalence may have been overestimated. However, although the true *level* of DSM-IV-defined insomnia in the population might deviate somewhat from that reported here, our focus was on prospective *associations*, which are less likely to be affected by differences in prevalence than prevalence estimates themselves. Second, emotion regulation and marital

conflict were collected from age 6, and we did not capture emotional reactivity at age 8. Thus, we may have missed information that could have affected the results. Third, to accommodate developmental changes that occur from age 4 to 14 we changed the emotional reactivity measure. Even though developmentally appropriate, the change of measures from the parent-reported CBQ-SF to the child-reported BFI may have implied, as indicated by the low correlation between the measures (Table S3), that time-invariant factors may to a lesser extent been adjusted for. Fourth, our findings may not apply to more ethnically diverse populations or other cultures, including those with different sleep routines. Finally, other factors not examined here could be of importance for insomnia (e.g., bedtime routines, sleep arrangements and sleep hygiene). Perhaps most notable of these are parent-child interactions preceding bedtime and parental presence at sleep onset, which are specific targets of the treatment of insomnia in young children [57-59]. The lack of direct measurement of behavioral factors that may cause or maintain insomnia should also be considered a limitation of the present study.

5 Conclusions

Within-person increases in emotional reactivity and decreases in emotion regulation skills predicted insomnia (as defined by the DSM-IV) two years later when adjusting for all unmeasured time-invariant factors, whereas marital conflict and family functioning did not predict insomnia. Hence, the aforementioned child-related factors may be involved in the etiology of pediatric insomnia and could be considered in both preventive and treatment efforts. Moreover, the fact that concurrent insomnia was the strongest predictor of later insomnia underscores the importance of early identification and treatment to prevent chronic insomnia.

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Declarations of Interest

None.

Data Availability

The participants did not consent to data availability.

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Table 1. Between-person correlations with insomnia from age 4 to 14

Insomnia	Ustd. correlation (95% CI)	p-value	Std. correlation
Emotional reactivity[a]	.021 (.011, .032)	<.001	.573
Emotion regulation[b]	-.005 (-.009, .000)	.026	-.379
Family functioning[c]	.005 (-.001, .010)	.108	.244
Marital conflict[b]	.003 (-.001, .007)	.137	.216

Note. Measured biennially at [a]age 4-6 and 10-14; [b]age 6-14; [c]age 4-14. Ustd. = unstandardized. Std. = standardized. These are correlations between factors tapping the constructs across the whole study period.

Table 2. Within-person predictors (range of scale) of insomnia from age 4 to 14

Insomnia	B (95% CI)	p-value	β
Insomnia[a] (0-1)	.195 (.111, .279)	<.001	.179
Emotional reactivity[b] (z-score)	.017 (.004, .031)	.010	.070
Emotion regulation[c] (1-4)	-.044 (-.089, .000)	.050	-.060
Family functioning[a] (1-4)	.042 (-.008, .093)	.101	.051
Marital conflict[c] (1-4)	-.005 (-.169, .160)	.954	-.003

Note. Measured biennially at [a]age 4-14; [b]age 4-6 and 10-14; [c]age 6-14. B= Unstandardized path coefficient. An increase of 1 in the predictor (i.e., a change in score from one's average level that is scale dependent) forecasts an increase in the change in insomnia risk relative to one's average level of insomnia. β = standardized path coefficient. These values are standardized (i.e., z-scores) and thus comparable across different measures (i.e., scale independent), which allows for comparison of the strength of the associations. The standardized value refers to the SD change in insomnia that will appear when the predictor changes one SD (e.g., .07 SD increase in insomnia risk deviation per 1 SD increase in emotional reactivity deviation).

Table S1. Sample characteristics at T1 (age 4 years) of the Trondheim Early Secure Study (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 country	3.3	32
Other country	4.3	42
Ethnic origin of biological father		
Norwegian	90.5	887
Western country	6.2	61
Other country	3.3	32
Biological parent marital status		
Married	54.7	536
Cohabitating	33.6	330
Divorced/separated	9.8	96
Other	1.9	19
Informant parent socioeconomic status		
Leader	5.7	54
Higher professional	25.5	242
Lower professional	39.3	374
Skilled worker	25.8	245
Farmer/fisherman	0.5	5
Unskilled worker	3.2	30
Household gross annual income		
0–225’ NOK (0–20’ USD)	3.4	33
225’–525’ NOK (20’–47’ USD)	18.3	180
525’–900’ NOK (47’–81’ USD)	51.8	509
> 900’ NOK (> 81’ USD)	26.5	260
Childcare		
Official day care center	94.8	942
Other	5.2	52

Note. Unweighted sample characteristics of the 1007 children assessed at T1 in the TESS. Missing data not included in percentages. NOK to USD exchange rate from 24 March 2020.

Table S2. Significant attrition predicted by study variables.

Attrition	OR	95% CI
Age 8 years		
Emotional regulation (age 6 years)	0.57	0.35, 0.94
Age 10 years		
Emotional regulation (age 6 years)	0.47	0.29, 0.76
Male sex	1.39	1.07, 1.81
Age 12 years		
Emotional regulation (age 6 years)	0.46	0.29, 0.73
Male sex	1.31	1.01, 1.69
Age 14 years		
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 was defined as not participating at that timepoint. The R^2 values were all <5%.

Table S3. Means (% for insomnia), standard deviations (95% CI for insomnia) and bivariate correlations of the study variables.

	M	SD	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27
1 Ins[4]	2.6%	(1.6, 3.5)	-																										
2 Ins[6]	2.4%	(1.4, 3.5)	.22[a]	-																									
3 Ins[8]	8.3%[a]	(6.2, 10.5)	.02	.18[b]	-																								
4 Ins[10]	10.2%	(7.8, 12.6)	.12[a]	.10	.21[c]	-																							
5 Ins[12]	7.9%	(5.6, 10.1)	.11	.11[a]	.23[c]	.32[c]	-																						
6 Ins[14]	10.5%	(7.8, 13.1)	-.02	.00	.10	.16[a]	.26[c]	-																					
7 Er[4]	-0.134	± 0.990	.14[c]	.10[b]	.10[a]	.07	.08	.05	-																				
8 Er[6]	-0.118	± 0.973	.09[c]	.14[c]	.15[b]	.13[c]	.13[b]	.15[b]	.65[c]	-																			
9 Er[10]	-0.034	± 1.003	.04	.07	.02	.13[b]	.14[c]	.04	.05	.14[b]	-																		
10 Er[12]	-0.029	± 1.014	.05	.04	.07	.13[b]	.21[c]	.20[c]	.07	.18[c]	.45[c]	-																	
11 Er[14]	-0.036	± 1.000	.07	.06	.03	.19[c]	.27[c]	.31[c]	.09[a]	.19[c]	.37[c]	.55[c]	-																
12 Ers[6]	3.138	± 0.415	-.02	-.09	-.09[a]	-.11[a]	-.15[b]	-.06	.00	-.05	.02	.02	.03	-															
13 Ers[8]	3.184	± 0.395	-.07	-.03	-.15[b]	-.19[c]	-.09[a]	-.06	.04	-.04	-.03	-.12[b]	-.07	.40[c]	-														
14 Ers[10]	3.097	± 0.416	-.08	-.05	-.12[b]	-.12[b]	-.11[a]	-.08	-.05	-.10[a]	-.12[b]	-.12[b]	-.10[a]	.21[c]	.34[c]	-													
15 Ers[12]	3.029	± 0.442	-.06	-.03	-.04	-.12[b]	-.15[b]	-.08	.04	-.04	-.04	-.06	-.03	.21[c]	.30[c]	.43[c]	-												
16 Ers[14]	2.874	± 0.448	.01	-.03	-.04	-.10[a]	-.10	-.16[b]	.05	.05	-.11[a]	-.07	-.01	.10[a]	.21[c]	.26[c]	.34[c]	-											
17 Ff[4]	1.633	± 0.400	.10[c]	.05	.01	.04	.04	-.07	.15[c]	.14[c]	.04	.04	.03	-.01	-.09[a]	-.05	-.08	-.07	-										
18 Ff[6]	1.671	± 0.407	.09[b]	.05	.06	.07	.03	-.02	.23[c]	.21[c]	.06	.03	.04	-.04	-.03	-.08[a]	-.07	-.05	.57[c]	-									
19 Ff[8]	1.610	± 0.383	.09[b]	.07	.00	.11[a]	.05	.06	.15[c]	.16[c]	.02	.00	-.01	-.09[a]	-.10[a]	-.04	-.07	-.05	.54[c]	.59[c]	-								
20 Ff[10]	1.592	± 0.394	.11[b]	.07[a]	-.04	.14[b]	.11[a]	.05	.17[c]	.21[c]	.08[a]	.08	.04	-.03	-.10[a]	-.08[a]	-.14[c]	-.06	.51[c]	.53[c]	.69[c]	-							
21 Ff[12]	1.596	± 0.387	.09[a]	.06	.06	.14[c]	.15[b]	.11[b]	.17[c]	.19[c]	.04	.09[a]	.06	-.02	-.09[a]	-.11[b]	-.15[b]	-.06	.46[c]	.52[c]	.62[c]	.70[c]	-						
22 Ff[14]	1.626	± 0.392	.06	.05	.05	.14[b]	.07	.11[a]	.13[b]	.19[c]	.07	.09[a]	.09[a]	-.02	-.06	-.08	-.13[b]	-.09[a]	.46[c]	.44[c]	.52[c]	.60[c]	.66[c]	-					
23 Mc[6]	1.752	± 0.263	.06	.06	.04	.03	.04	-.04	.19[c]	.19[c]	.05	.12[b]	.06	-.07	-.08	-.05	-.01	.05	.44[c]	.53[c]	.43[c]	.38[c]	.36[c]	.30[c]	-				
24 Mc[8]	1.719	± 0.255	.08[a]	.06	.08	.11[b]	.08	-.02	.15[c]	.18[c]	.05	.07	.04	-.08	-.07	-.03	.00	.09	.42[c]	.44[c]	.51[c]	.44[c]	.39[c]	.35[c]	.79[c]	-			
25 Mc[10]	1.698	± 0.259	.13[b]	.11[a]	.07	.12[b]	.07	.02	.15[c]	.20[c]	.10[a]	.13[b]	.10[a]	-.05	-.10[a]	-.06	-.04	.03	.36[c]	.38[c]	.46[c]	.55[c]	.45[c]	.38[c]	.73[c]	.80[c]	-		
26 Mc[12]	1.702	± 0.263	.12[b]	.12[b]	.08	.14[b]	.13[a]	.02	.13[b]	.15[b]	.09[a]	.11[a]	.05	-.06	-.09	-.05	-.04	.04	.35[c]	.34[c]	.40[c]	.48[c]	.51[c]	.40[c]	.68[c]	.73[c]	.81[c]	-	
27 Mc[14]	1.687	± 0.255	.09	.09[a]	.09[a]	.12[b]	.11[a]	.00	.14[b]	.16[c]	.06	.14[b]	.07	.02	-.03	-.04	-.03	.06	.29[c]	.26[c]	.32[c]	.41[c]	.42[c]	.45[c]	.62[c]	.68[c]	.72[c]	.79[c]	-

Note. Ins, DSM-IV defined insomnia (range is 0-100% prevalence in the population, insomnia coded at the individual level as 0 or 1) measured by the Preschool Age Psychiatric Assessment (PAPA; age 4-6) and the Child and Adolescent Psychiatric Assessment (CAPA; age 8-14); Er, emotional reactivity (z-score) measured by the negative affectivity dimension of the Children's Behavior Questionnaire short form (CBQ-SF; age 4-6) and Big Five Inventory (BFI; age 10-14) dimension of neuroticism; Ers, emotion regulation skills (range 1-4) measured by the emotion regulation subscale of the Emotion Regulation Checklist (ERC); Ff, family functioning (range 1-4) measured by the General Family Functioning scale of the Family Assessment Device (FAD); Mc, marital conflict (range 1-4) measured by the Conflicts and Problem-Solving Scales; M, mean; CI, confidence interval; SD, standard deviation; [4]Age 4; [6]Age 6; [8]Age 8; [10]Age 10; [12]Age 12; [14]Age 14. [a] $p<.05$; [b] $p<.01$; [c] $p<.001$.

Highlights

- Emotional reactivity and regulation predicted two-year later insomnia
- Insomnia risk was analyzed with a random intercept cross-lagged panel model
- The results presented here support a possible vulnerable phenotype of insomnia
- The strongest predictor of insomnia was previous insomnia
- Both preventive and treatment efforts may be guided by these results