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1. Introduction

Eleven to thirty-eight percent of adolescents have chronic pain, with onset of musculoskeletal pain peaking during early adolescence [46], and up to sixty-four percent continue having pain into adulthood [5]. The prevalence of insomnia (DSM-IV-criteria) among adolescents can approach 23.8 percent [19,41], and more than half of youth with chronic pain experience disturbed sleep [65]. Research has consistently demonstrated that pain and sleep problems are related [7]. Based on both human and animal studies, a bidirectional relationship has been proposed [2,50,80,86,88], with more recent longitudinal and micro-longitudinal studies (primarily) indicating a stronger influence of sleep problems on pain than vice versa [3,7,9,20,25,38,83]. Still, many questions remain regarding the relationship’s underlying mechanisms [7,9,25,55].

There is growing interest in explaining the associations between sleep and pain by mood changes, motivated by the prospect of enhancing pain treatments with elements from sleep- and mood interventions [9]. Yet, studies investigating moods as potential mediators in the sleep-pain association, are still too few to produce conclusive findings. Also, evidence of such associations mostly comes from cross-sectional studies [10,13,23,36,58,61,68,69,73], three of which focused on youth [10,23,69]. Indeed, only longitudinal designs with multiple measurement occasions can capture the temporal dimension inherent in mediation [52,90]. A handful studies have been longitudinal, all finding moods to mediate the sleep-pain relationship [12,30,48,89]. However, no study has examined the mediating role of moods using a longitudinal design that allows for a strict test of mediation; with predictor, mediator and outcome variables all measured at separate occasions.

To our knowledge, only one longitudinal study and no studies including a non-clinical sample have examined the mediating role of mood in the sleep-pain relationship in adolescents. So far, only one model has addressed this relationship across this age-span, the mutual
maintenance model of pediatric pain and sleep [88]. It highlights a bidirectional influence between pediatric pain perception and sleep characteristics mediated by mood, since mood disturbance often co-occurs both with sleep and pain [88]. There is initial support to the model [69], suggesting depressed mood, anxious mood, and (potentially) positive affect as mediators in a pediatric chronic pain sample.

Potential mediation effects in the sleep-pain relationship have not yet been fully specified. Common neurological pathways, such as the dopaminergic system, have been proposed to explain the co-morbidity of sleep disturbance, pain and disturbed mood [27], where symptom changes co-occur in a reciprocal triad [31]. In contrast, temporal relationships between these constructs have been hypothesized; that sleep disturbance precedes mood disturbance, which precedes pain [48]. Simultaneously examining the bidirectional mediation of mood in the sleep-pain relationship is critical for addressing this question. Such an analysis has not yet been published.

The purpose of this study was to examine the bidirectional, temporal relationship of insomnia symptoms and pain via depressed mood, anxious mood and positive affect as mediators, using longitudinal data spanning from early, mid, to later adolescence. We hypothesized that: 1) there is a bidirectional relationship between insomnia symptoms and pain, 2) depressed mood, anxious mood, and positive affect mediate the relationship bidirectionally.

2. Methods

2.1 Participants and procedure

The sample consisted of participants in the “Three Cities Study”, a 5-year longitudinal project (2014-2018) in three communities in mid-Sweden. A general aim of this project was to identify factors that are associated with psychosocial problems among adolescents. Data were collected at five yearly measurement occasions. The self-report questionnaires were filled out
in the students’ classrooms. Test leaders, but not teachers, were present for assistance. Every
class received 300 SEK for each data collection. To reduce sampling bias, the study used
passive consent from the students’ parents and active consent from the students [79]. The study
has been approved by the regional Ethical Board in Uppsala (No 2013/384).

The target sample was 3336 7th and 8th grade students. Of these, 2766 students (82.91
%) participated in the first wave of data collection (T1) and form the baseline sample. Of the
baseline sample, 2521 participated in wave 2 (T2), 1982 participated in wave 3 (T3), 1666
participated in wave 4 (T4), and 1568 participated in wave 5 (T5). In total, 1132 students
participated in all 5 measurement occasions (40.9 % of the baseline sample); 1753 students
(63.4 %) participated in at least four occasions, 2230 (80.6 %) in at least three, 2622 (94.8 %)
in two or more, and 144 (5.2 %) only participated at T1. At T1, the mean age was 13.65 years
(\textit{SD} = 0.65 years), and at T5, the mean age was 17.68 years (\textit{SD} = 0.66 years). Thus, the current
study used longitudinal data spanning from early adolescence, through mid-adolescence, to
later adolescence.

An ordinal logistic regression analysis showed that rate of attrition was predicted by
being a boy (- 0.226, \textit{P} = .007), having divorced parents (0.379, \textit{P} < .001), being born outside
of Sweden or having parents born outside of Sweden (- 0.227, \textit{P} < .001), and having higher
levels of insomnia symptoms (- 0.037, \textit{P} = .001).

2.2 Self-report measures

2.2.1. Descriptive measures and covariates

Socio-demographic measures at T1: \textit{Age} was measured by the respondent indicating their age
in years. \textit{Gender} was assessed with one dichotomous item – “boy” or “girl”. The respondents
reported whether their parents were divorced or not in one dichotomous item of \textit{Parents’
marital status}. \textit{Immigrant status} was assessed with three items, asking respondents if they were
born in Sweden, where their mother was born, and where their father was born. As a covariate, immigrant status was coded into three values: 1) born in Sweden and both parents born in Sweden, 2) born outside of Sweden but in Europe, or having at least one parent born outside of Sweden but in Europe, or 3) born outside of Europe or having at least one parent born outside of Europe. As a descriptive measure, immigrant status dichotomized as being born outside of Sweden or having both parents born outside of Sweden, following the official Swedish definition of immigrant background [62]. Socio-economic status (SES) was assessed using the Family Affluence Scale, version 2 (FAS-II), from WHO’s Health Behavior in School-aged Children (HBSC) – survey [4], which includes four items where the respondent assesses their family’s affluence. The cut-off score for low SES was set to 4.61, which is one standard deviation below the mean \(M = 6.28\) (\(SD = 1.67\)) of FAS-II in the Swedish HBSC-survey (\(N = 23088\)) [4].

[40,77]2.2.2. Pain

The current study focuses on musculoskeletal pain, which was defined as “pain in the back, neck or shoulders”. Three aspects of the pain experience were measured. Pain intensity was measured with a 0-9 numeric rating scale [92], asking respondents to rate their average back, neck or shoulder ache during the last six months, on a 0-9 scale, ranging from “not painful at all” to “very painful”. This measure was originally intended to be a 11-point NRS-11 scale but was administered as a 10-point scale in the Three Cities Study. Importantly, the labels were the same as in the NRS-11 (“not painful at all” and “very painful”). Moreover, different versions of pain intensity scales, with different number of response alternatives, has shown similar results [91]. Pain frequency was assessed via one item from the Health Behavior in School-Aged Children Checklist (HBSC) [37]. Respondents were to rate how often they had experienced pain in the back, neck or shoulders during the last six months, on a five-point scale, ranging from 0 (“rarely or never”), 1 (“about every month”), 2 (“about every week”), 3 (“more
than once a week”), to 4 (“about every day”). Pain interference was measured using three items, adopted from the Social Phobia Screening Questionnaire [28] for the purposes of the Three Cities Study, where a short measure was required. The rationale for using the interference items from the Social Phobia Screening Questionnaire was that its format has been administered to a large sample of Swedish adolescents, successfully measuring functional interference due to social anxiety [32]. Respondents were to rate whether, during the last 6 months, their pain in back, neck or shoulders was of such nature that it severely interfered with 1) school work 2) leisure activities and 3) peer relationships. Each item had three responses: 2 (“no”), 1 (“yes, a bit”) and 0 (“yes, definitely”). The three items were reversed and averaged into a total score ranging 0 to 2. The measure has been used in one previously published study [94].

Further on, in the methods and results section, musculoskeletal pain will be referred to as “pain”.

2.2.4. Insomnia symptoms

The Insomnia Severity Index (ISI) [11] is a well-established self-report instrument for measuring insomnia symptoms and clinical insomnia, also in adolescents, and is suitable for measuring change over time. It consists of seven items regarding sleep problems during the last two weeks, each with ratings from 0 (“no problems at all”) to 4 (“very much”). A total score of 9 has been suggested as an appropriate cut-off for insomnia in adolescents [17]. In adolescents, the ISI has been shown to consist of three subscales: A subscale of interference and distress due to sleep problems (items 5, 6, and 7), a subscale of sleep satisfaction and difficulties falling asleep (items 1 and 4), and a subscale of difficulties maintaining sleep (items 2 and 3; Jansson-Fröjmark & Bauducco, [Forthcoming]). The ISI has demonstrated high internal consistency in a sample of Swedish youths with chronic pain (α = 0.83) [45]. It also correlates significantly with other subjective measures of sleep quality, such as the Pittsburg Sleep Quality Index and sleep diaries, as well as objective measures of sleep quality such as polysomnography [56] and
actigraphy [93]. The internal consistency in the current sample was $\alpha = .85$ for T1, $\alpha = .84$ for T2, $\alpha = .86$ for T3, $\alpha = .87$ for T4, and $\alpha = .88$ for T5.

2.2.5. Depressed mood and Positive affect

The Center for Epidemiology Studies Depression Scale for Children (CES-DC) [72] was used to assess depressed mood and positive affect. This questionnaire contains 20 items of which each is rated from 0 (“not at all”) to 3 (“a lot”) and relates to symptoms perceived during the last week. It has been validated in Swedish adolescents [63]. The cut-off for clinical depression is a total score of 24, on a range from 0 to 60. It has been used in studies on somatic illness due to low overlap with somatic symptoms [58]. At T1, CES-DC was measured on a 1- to 5- point scale, whereas at the other time points the original 0– to 3- point scale was used. Therefore, each item at T1 was first rescaled to 0 - 4, and then adjusted (by multiplying the item value by 0.75).

CES-DC contains four subscales: depressed mood, positive affect, somatic activity and interpersonal functioning. The subscale “Depressed mood”, consisting of items 1, 3, 6, 10, 17 and 18, was used as a measure of depressed mood in the current study. The internal consistency of the subscale in the current sample was $\alpha = .92$ for T1, $\alpha = .89$ for T2, $\alpha = .89$ for T3, $\alpha = .90$ for T4, and $\alpha = .90$ for T5. The subscale “Positive affect”, consisting of item 4, 8 and 12, was used as a measure of positive affect. The internal consistency of the subscale in the current sample was $\alpha = .89$ for T1, $\alpha = .68$ for T2, $\alpha = .71$ for T3, $\alpha = .71$ for T4, and $\alpha = .67$ for T5.

2.2.6. Anxious mood

Anxious mood was assessed with the five item Overall Anxiety and Impairment Scale (OASIS) [60]. Respondent were asked to rate frequency, intensity of anxiety and fear, as well as functional impairment due to anxiety and fear they have experienced during the last week on Likert scales, ranging from 0 (“none/no”) to 4 (“constant/extreme/all the time”). The summed
total score ranges from 0 - 20, with higher scores indicating more anxiety. The cut-off for clinical anxiety problems is 8 [14], and the scale has previously shown good psychometric properties. The internal consistency in the current sample was $\alpha = .87$ at T1, $\alpha = .89$ at T2, $\alpha = .89$ at T3, $\alpha = .90$ at T4, and $\alpha = .91$ at T5.

2.3 Statistical analyses

Data preparation was performed using IBM SPSS Statistics for Windows, version 26.0 [42]. Descriptive statistics and subsequent structural equations modeling (SEM) were performed in Mplus, version 8.2 [57].

2.3.1 Data preparation

To facilitate model convergence and interpretation of covariance matrices, all model indicators were rescaled to matched scales via proportion of maximum scoring (POMS)-transformation [52]. When subscales were not applicable, items were parceled together based on a balancing approach, where the item with the highest item-scale correlation was paired with the item with the lowest item-scale correlation [52]. Item parcels were averaged.

Latent constructs were subsequently built of the observed subscales or parcels. A latent construct of pain was made by three indicators: pain intensity, pain frequency and pain interference. A latent construct of insomnia symptoms was constructed by the ISI’s three subscales. A latent construct of depressed mood was constructed from three CES-DC item parcels, consisting of items 1 and 6, items 3 and 17, and items 10 and 18, respectively. A latent construct of positive affect was constructed from the three items in the “Positive affect” – subscale of the CES-DC. A latent construct of anxious mood was constructed from OASIS item 4, as well as two OASIS item parcels, one consisting of items 1 and 3, the other of items 2 and 5.
Missing data was handled with full maximum likelihood estimation. This is a model-based approach to handling missing data, where all available information in the data is used to estimate the parameters and standard errors of the statistical model [22]. We assumed a missing at random – mechanism as explaining missingness in the dataset and included the entire baseline sample of n = 2766 in all analyses, regardless of degree of attrition on subsequent measurement occasions. Since Mplus treats missing values in covariates with listwise deletion, missing data on SES and immigrant status were imputed via the EM-algorithm in SPSS. The amount of missing data on the SES – variable was 2.9 percent and 1.1 percent on the immigrant status – variable.

2.3.2 Structural equation modeling

First, longitudinal measurement models for each of the constructs (pain, insomnia symptoms, depressed mood, positive affect and anxious mood) were constructed and separately tested for measurement invariance. That is, (1) configural invariance (i.e. model form equal across time), (2) weak/metric invariance (i.e. factor loadings for each indicator equal across time), and (3) strong/scalar invariance (i.e. indicator intercepts equal across time).

Subsequently, the four separate measurement models were combined into four extended measurement models – one model with only insomnia symptoms and pain, and three models that also included each of the mediators. Measurement invariance was tested for each of the four models. Model fit assessments was based on a composition of previous guidelines [34,52,71]: \( \chi^2 \) with associated degrees of freedom is reported, as well as the Comparative Fit Index (CFI), which is an incremental fit index, and the Root Mean Square Error of Approximation (RMSEA), which is an absolute fit index. Given the sample size and number of observed variables in the current study, we expected the \( \chi^2 \) – values to be significant. An RMSEA of .07 - .05 represents an acceptable fit, and a value of less than .05 represents a good
fit of the data. A CFI of .90 - .95 represents an acceptable model fit, and a CFI of more than .95 represents a good fit of the data.

Having determined strong measurement invariance in all measurement models, a cross-lagged panel model with the latent variables pain and insomnia symptoms was constructed in order to assess the bidirectional relationship between insomnia symptoms and pain. This model was subsequently expanded into three cross-lagged longitudinal mediation models examining the overall total effect, overall direct and overall indirect effects, as well as time-specific direct and indirect effects [18,52].

Since mediation and total effects are unfolding over the course of time in longitudinal studies, our primary focus was on calculating the overall effects from predictor at T1 to outcome at T5, via all six possible routes passing through the mediator at some point (See Text and Figures, Supplemental Digital Content 1, for more details). The product effects of these six routes were summed into an overall indirect effect reflecting the amount change in the outcome at T5 due to the predictor at T1 that was mediated. The overall total effect is the sum of all path effects from the predictor at T1 to the outcome at T5, and the overall direct effects represent all paths not passing through the mediator [18,78]. The proportion of the total effect explained by the mediator was calculated by dividing the overall indirect effect by the overall total effect, as recommended by MacKinnon & Dwyer [54]. Since the product of coefficients constituting the indirect effect rarely is normally distributed, we used bias-corrected confidence intervals based on 10 000 bootstrap samples to test the indirect effects for significance [29]. We opted for a strict criterion of 99 percent confidence intervals because of the large sample size. Two additional tests were conducted to explore potentially confounding effects in the mediation analyses [18,52]: (1) Test of equilibrium or test for homogeneity of within-time variance-covariance matrices. A significant test indicates that external factors have influenced the relationships among the constructs differently over time. (2) Test for omitted variables or test
for nonzero residual correlations among the constructs. A significant test indicates that omitted variables have significantly influenced the mediation models (See Text and Tables, Supplemental Digital Content 2, for more details on the tests for omitted variables). Note that a significant result on either test gives information on the extent of potential unobserved influence in the model, but does not in itself contradict mediation [52].

Gender, age, immigrant status and SES, measured at baseline, were included as time-invariant covariates in all analyses.

3. Results

3.1 Sample characteristics

Sociodemographic data are summarized in Table 1. The sample was balanced regarding gender. 11.7 percent of participants reported a pain frequency of at least once per week at baseline, which is comparable to the 12.5 percent prevalence reported in the general Swedish population of 13-year-olds [85]. As can be seen in Table 2, there is a tendency for all key variables to increase over time. Two exceptions are pain intensity, which peaked at T4, and positive affect, which seems stable over time.

At baseline, girls scored significantly higher on all key variables as compared to boys, the exception being positive affect, which showed the inverted relationship. Age was not significantly associated with any of the key variables. Native Swedish participants reported significantly lower levels of pain interference compared to participants from an immigrant background. Lower SES was associated with higher levels of pain interference and lower levels of positive affect (See Text and Tables, Supplemental Digital Content 2, for detailed descriptions of these analyses).
3.2 Preliminary analysis: Model evaluations

3.2.1 Measurement models

In an unrestricted, configural longitudinal measurement model, which showed good fit to the data ($\chi^2_{2295} = 5052.4$, 90% CI $RMSEA = 0.020 – 0.022$, $CFI = 0.975$), all latent variables significantly correlated with each other, at all time-points. The correlations followed a simplex-like pattern [52]. The lowest correlations were found between positive affect and pain, ranging from $r = .078$ (positive affect at T2 and pain at T4) to $r = .300$ (between positive affect and pain at T1), and the highest correlations between depressed mood and anxious mood, ranging from $r = .833$ (between depressed mood and anxious mood at T5) to $r = .418$ (anxious mood at T1 and depressed mood at T5).

Four measurement models were fitted, and all models included insomnia symptoms and pain at all 5 time-points. One model only included insomnia symptoms and pain; the other three models also included one mediator (depressed mood, positive affect and anxious mood) at all five time-points. The indicators to each latent construct were allowed to correlate with each other on subsequent and every other time-points (See Text and Figures, Supplemental Digital Content 3, for detailed information on the measurement models). All models showed a good fit of the data and held up to the assumption of strong measurement invariance (Table 3). They furthermore did not violate the assumption of equilibrium, as can be seen in Table 3. This indicates that external factors have not significantly influenced the relationships among the constructs in the model differently over time. A significant test for omitted variables indicated that omitted variables may have influenced the relationships between the constructs in the model.
3.2.2 Longitudinal structural models

First, we established that an autoregressive model including insomnia symptoms and pain, with cross-lagged paths both from insomnia symptoms and from pain, demonstrated the best fit of the data, compared to competing models (See Text and Figures, Supplemental Digital Content 4, for more details). Three latent longitudinal full mediation models were then fitted. Each model included a mediator at all five measurement occasions: one model included depressed mood as mediator, one included positive affect, and one had anxious mood as mediator. Each model included direct bidirectional cross-lagged paths between insomnia symptoms and pain at every other time-point (T1 to T3, T2 to T4, T3 to T5), as well as indirect paths passing through the mediator (at T2, T3 and T4). The three models showed good fit to the data, as can be seen in Table 3, and held up to the assumption of strong measurement invariance.

3.2.3 Covariate associations with latent constructs at T1

Regarding the covariates, which the T1 latent constructs were regressed on, gender had a significant effect on all variables ([-0.120 to -0.422], SE = [0.018 - 0.020]), with girls showing higher symptom levels than boys. Immigrant status consistently had a significant effect on depressed mood and positive affect ([0.074 to -0.053], SE = [0.020 - 0.022]), where participants with immigrant background rated lower on depressed mood but higher on positive affect. SES had a significant effect positive affect and anxious mood ([0.122 to -0.051], SE = [0.021 - 0.021]), where participants with lower SES demonstrated lower positive affect and higher anxious mood. Inclusion of covariates did not cause any meaningful changes in the models and the main direct- and indirect effects were not affected. Including the covariates in the models, however, appeared to slightly attenuate the effects from insomnia symptoms to pain, and to increase the effects from pain to insomnia symptoms (See Text and Figures, Supplemental Digital Content 4, for more detailed information).
3.3 Primary analysis: Cross-lagged models for bidirectional associations and mediation

3.3.1 Bidirectional relationship between sleep and pain

There was a significant overall effect of insomnia symptoms from T1 on pain at T5 (0.244, 99% CI [0.161 - 0.326]), as well as a significant overall effect of pain from T1 on insomnia symptoms at T5 (0.087, 99% CI [0.047 - 0.128]). A bidirectional relationship between insomnia symptoms and pain was confirmed, where an increase in insomnia symptoms leads to future increases in pain, and vice versa. The effect of insomnia symptoms on pain was larger than the effect of pain on insomnia symptoms; the difference between the overall effects was significant (0.157, 99% CI [0.061 – 0.246]). There was a tendency for the effect of pain on insomnia symptoms to decrease over the measured time-period (standardized range: a significant 0.110 for the first cross-lagged effect, to a non-significant 0.035 for the last; difference test: 0.038, 99% CI [0.001 - 0.076]), as can be seen in Figure 1. The effect of insomnia symptoms on pain fluctuated but remained significant across the entire time-period.

3.3.2 Depressed mood as a mediator in the relationship between insomnia symptoms and pain.

Depressed mood mediated the effect from insomnia symptoms at T1 to pain at T5, with an overall indirect effect of 0.029 (99% CI [0.011 - 0.054]), the mediating effect representing 16.9% of the total effect. Depressed mood did not mediate the overall total effect from pain at T1 to insomnia symptoms at T5, (0.004, 99% CI [0.000 - 0.011]), with the mediation effect representing 5.9% of the total effect. For more information, see Table 4. The full model, with path coefficients, is depicted in Figure 2.
3.3.3 Positive affect as a mediator in the relationship between insomnia symptoms and pain.

Positive affect did not mediate the effect from insomnia symptoms to pain (0.005, 99% CI [-0.007 to 0.017]) nor the effect from pain to insomnia symptoms (0.000, 99% CI [-0.004 to 0.003]). The overall direct effects were significant, as can be seen in Table 4. The full structural model can be seen in Figure 3.

3.3.4 Anxious mood as a mediator in the relationship between insomnia symptoms and pain.

Anxious mood mediated the overall indirect effect from insomnia symptoms at T1 to pain at T5 (0.031, 99% CI [0.010 - 0.061]), explaining 15.5% of the overall total effect. The overall indirect effect from pain at T1 to insomnia symptoms at T5 was non-significant (0.004, 99% CI [0.000 - 0.012]), where the overall indirect explained 5.3% of the overall total effect. All direct effects were significant (see Table 4). The full structural model can be seen in Figure 4.

4. Discussion

This longitudinal study, spanning adolescence, confirmed a bidirectional relationship between insomnia symptoms and pain, the effect of insomnia symptoms on pain being the stronger effect. Depressed and anxious mood mediated the effect of insomnia symptoms on pain, but not vice versa. Positive affect did not mediate the relationship in either direction. Our longitudinal design and large sample of adolescents allows us to confirm mood as a mediator in the effect of sleep on pain, and allows for generalization of our results in adolescents.

The finding that insomnia symptoms and pain bidirectionally affect each other, with the effect of insomnia symptoms on pain being stronger than vice versa (Fig. 1), confirms our first hypothesis and is consistent with previous research [7,25]. Moreover, the current results suggest
that the effect of pain on insomnia symptoms decreases across adolescence. One idea is that the self-regulatory abilities required to cope with pain develop during adolescence, allowing older adolescents to handle negative effects of pain more efficiently and thereby avoiding its negative impact on sleep. Indeed, previous research has demonstrated that maturation of the prefrontal cortex during adolescence leads to improved cognitive control and emotion regulation [74]. However, pain predicts sleep problems in adults [5,7], sometimes even stronger than the effect of sleep on pain [6,44]. Future longitudinal studies, spanning from childhood throughout adulthood, could provide further insights into whether the effect of pain on sleep changes across development into adulthood. Additionally, due to differences between acute and chronic pain [16], future studies may want to examine these patterns also in chronic pain populations.

Our second hypothesis was partially supported. Depressed and anxious mood mediated 16.5 and 15.5 percent of the effect of insomnia symptoms on pain, respectively, but neither mediated the effect of pain on insomnia symptoms. This echoes previous cross-sectional studies, two of which included adolescents [23,69]. All four longitudinal studies found evidence for depressive and/or anxious symptoms as mediator(-s) in the sleep-pain relationship [12,30,48,89]. A common limitation of these studies is that the mediator temporally overlapped with either the predictor or the outcome. A few studies found evidence for mediating effects in the relationship from pain to sleep [58,73,89], possibly due to their cross-sectional design or temporal overlap in their mediation analyses, which may have confounded the directionality of the effects.

The current results partly support the mutual maintenance model of pediatric pain and sleep [88], and so we propose an update of the model (Figure 5). Here, mood unidirectionally mediates the path from insomnia symptoms to pain rather than bidirectionally. However, further investigation and replication of the results are needed, encompassing longitudinal studies that include both clinical and normal population samples. Future studies may extend the present
study by exploring whether the model can be expanded throughout the life-span. We also indicated a stronger effect from insomnia symptoms to pain than vice versa.

[Figure 5]

The temporal distance between measurement occasions is important in longitudinal mediation, since the effects captured depend on the time-lag. [18]. Longer temporal distances, such as in the current study, are suitable for trait-like constructs [52]. Although we acknowledge significant state-elements in the key constructs, insomnia includes chronic elements [75], particularly when comorbid with pain [9], chronic pain is a stable condition that has been conceptualized as a trait [81], and moods are associated with traits [8,49].

Most conceptual research on mechanisms affecting the sleep-pain relationship has focused on depression. However, previous research links depression and anxiety together via a common, underlying construct; \textit{trait negative affect} [8], which is distinct from both sleep problems and pain [48]. Positive affect was not a significant mediator in the current study, which is congruent with studies on youth[90]. In adults, sleep problems attenuate pain inhibition by reducing positive affect [26], an effect hypothesized to increase in the transition from childhood to adulthood [23]. Our results suggest that this change may happen after adolescence.

Can sleep, pain and mood be temporally separated, or do they covary due to a common, underlying factor? They have been described as a critical triad, where a change in one construct changes the others [31], with the mesolimbic dopamine system being the underlying neurological pathway [27]. Our finding that mood only mediates the effect of insomnia symptoms on pain, and previous findings that mood is a stronger mediator between sleep and pain than pain is between sleep and mood, are in line with the idea that sleep problems, by leading to emotional dysregulation, may impact pain [48]. This hypothesis is consistent with research showing that sleep influences both emotion generation and emotion regulation [67],
and that maladaptive emotion regulation indirectly impacts pain via negative affect [47]. Taken together, these results suggest that sleep, pain and mood are more intricately temporally related than the critical triad – theory implies. Future longitudinal studies could explore the mediating role of sleep in the pain-mood relationship, and thereby expand on cross-sectional evidence of this relationship [45]. Recent studies have also found compelling evidence of sleep as a mediator between PTSD and pain [1,59,70], adding clarity to the complex relationship between pain and PTSD [39]. Examining the mediating role of PTSD in the sleep-pain relationship would be an interesting extension of the current study.

The current study addresses common methodological limitations in studies on mediation in the sleep-pain relationship [90]: First, it is based on an explicit theoretical framework, which facilitates a more unified progress in this research field. Second, it examines mediation longitudinally; the current study is the first to temporally separate predictor, mediator and outcome. We also measured the mediator at multiple time-points, which is a robust method of assessing mediation across a developmental stage [52]. Third, the study implemented latent constructs, thereby controlling for measurement error that might otherwise bias the estimates of associations among variables.

The following limitations warrant more discussion. First, a priori collected data were used. This population-based dataset allowed us to test the mutual maintenance model with valid measures across adolescence, but we were limited in assessing additional potential mediators, such as repetitive negative thinking, which is associated with both sleep problems and pain [21]. Sleep disturbance can also increase pain via inflammation and endogenous opioid-mediated pain inhibition [16,25]. Future studies should include a wider array of potential mediators, to improve understanding of the mechanisms affecting the sleep-pain relationship. Second, our reliance on self-report measures, which increases the risk of common method bias. However, a dataset of this magnitude can hardly include other types of measures, and the modeling of latent
constructs controls for a large proportion of shared error variance among variables. In addition, the ISI and objective measures of sleep correlate highly [56,93], indicating that the ISI is a robust measure of sleep problems. Moreover, pain is affected more by sleep quality than sleep time [16], suggesting that subjective sleep measures may be a preferred method in relation to pain. A third limitation is the rate of missing data, which may bias the estimates. However, the rate was within the normal range for prospective studies [33], and maximum likelihood estimation, which we used here, produces unbiased parameter estimates under a fairly unrestricted assumption [22,52]. Also, the statistical models used in this study do not distinguish between potentially important within-person and between-person effects[35]. Finally, although we controlled for the confounding effect of key covariates, our results may be affected by omitted variables, a problem inherent in all observational studies. Several unobserved influences may have been of importance, such as developmental influences on the examined relationships, or potentially moderating effects of, for example, gender or cultural background. Future studies could apply a counterfactual framework for causal mediation analysis, which allows for a more thorough examination of confounders [43,53]. Notwithstanding these limitations, the current study adds valuable information on how complex processes unfolds over time in a naturalistic setting.

In conclusion, this study found a bidirectional influence between insomnia symptoms and pain across adolescence, and that the effect of insomnia symptoms on pain is mediated by depressed and anxious mood. A complex interplay between biological (e.g., puberty), psychological (e.g., intensively perceived emotions) and social (e.g., autonomy) developmental changes across adolescence contributes to the interactive increases of insomnia, pain, and mood disturbance [9,15,66,76]. Often, these conditions become chronic and persist into adulthood, where they also predict additional comorbidities and disability [9,16,87]. One potential contribution to this is that critical brain circuits may become altered due to the experienced
sleep- and pain disturbance, facilitated by high neuroplasticity in adolescence, to become permanent when transitioning into adulthood [16,64]. Adolescence therefore appears a pivotal developmental period for implementing interventions to prevent insomnia and pain from developing and becoming chronic [64]. Hybrid interventions, combining elements from both Cognitive behavior therapy (CBT) for insomnia and CBT for pain, have shown promising results, both in adults [24,82] and in adolescents [51,64]. The current results indicate that it may be critical to specifically integrate depressed and anxious mood as clinical components in future hybrid interventions, to further improve treatment effects, especially regarding pain.

5. Acknowledgements

This study was made possible by access to data from the Three Cities Study, a longitudinal research program at the department of Law, Psychology and Social work at Örebro University, Sweden. The research program was supported by a grant from the Swedish research council for sustainable development (Formas), the Swedish research council for health, work life and welfare (Forte), The Swedish Research Council (VR) and Sweden’s innovation agency (Vinnova) (grant number 2012-65). The program was approved by the regional ethics board of Uppsala (nr. 2013/384). Special thanks the schools and adolescents who participated in the project. Additional thanks to Katja Boersma, PhD, PI of the Three Cities Study, for allowing us to use and publish data in the current study, for helping with queries related to the data set, and for initial input on the study’s focus. Additional thanks also to Lauree Tilton-Weaver, PhD, for assisting with statistical queries.

We provide the program syntax for our analyses, both for SPSS (See Text, Supplemental Digital Content 5, SPSS syntax) and for Mplus (See Text, Supplemental Digital Content 6, Mplus syntax). We also provide the variance-covariance matrix that our analyses were based upon (See Text, Supplemental Digital Content 7, variance-covariance matrix).
matrix of data), which allows for replication of our analyses. However, the individual data may only be accessed after permission from Katja Boersma, PI of the Three Cities Study.

6. Conflict of interest statement

The authors have no conflict of interests to declare.
7. References


Table 1

Descriptive statistics of the baseline sample; T1 (N = 2766).

<table>
<thead>
<tr>
<th>Variable</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender, girls</td>
<td>47.6</td>
</tr>
<tr>
<td>Immigrant background</td>
<td>24.0</td>
</tr>
<tr>
<td>Low SES</td>
<td>18.3</td>
</tr>
<tr>
<td>Divorced parents</td>
<td>33.6</td>
</tr>
<tr>
<td>Pain frequency &gt; once per week(^a)</td>
<td>11.7</td>
</tr>
<tr>
<td>Clinical depression</td>
<td>13.6</td>
</tr>
<tr>
<td>Clinical insomnia</td>
<td>21.0</td>
</tr>
<tr>
<td>Clinical anxiety</td>
<td>11.9</td>
</tr>
</tbody>
</table>

Note: The clinical items refer to proportion ≥ the cutoffs mentioned in the methods-section.

\(^a\) During the last 6 months.
Table 2

Descriptive statistics for key variables at T1 to T5, as well as proportion of missing data on each variable.

<table>
<thead>
<tr>
<th>Measure</th>
<th>M (SD)</th>
<th>Missing data %</th>
<th>M (SD)</th>
<th>Missing data %</th>
<th>M (SD)</th>
<th>Missing data %</th>
<th>M (SD)</th>
<th>Missing data %</th>
<th>M (SD)</th>
<th>Missing data %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pain intensity [0-9]</td>
<td>1.87 (2.37)</td>
<td>2.6 (2.49)</td>
<td>2.30 (2.58)</td>
<td>10.1 (2.55)</td>
<td>2.42 (2.48)</td>
<td>29.2 (2.55)</td>
<td>2.48 (2.55)</td>
<td>40.5 (2.55)</td>
<td>2.28 (2.55)</td>
<td>44.4 (2.55)</td>
</tr>
<tr>
<td>Pain frequency [0-4]</td>
<td>0.91 (1.16)</td>
<td>2.8 (1.26)</td>
<td>1.09 (1.31)</td>
<td>10.3 (1.33)</td>
<td>1.18 (1.33)</td>
<td>29.3 (1.33)</td>
<td>1.20 (1.33)</td>
<td>40.6 (1.33)</td>
<td>1.22 (1.33)</td>
<td>43.8 (1.33)</td>
</tr>
<tr>
<td>Pain interference [0-2]</td>
<td>0.21 (0.46)</td>
<td>2.9 (0.49)</td>
<td>0.22 (0.49)</td>
<td>10.4 (0.49)</td>
<td>0.23 (0.48)</td>
<td>29.3 (0.48)</td>
<td>0.23 (0.49)</td>
<td>40.6 (0.48)</td>
<td>0.24 (0.49)</td>
<td>44.2 (0.49)</td>
</tr>
<tr>
<td>Insomnia symptoms [0-28]</td>
<td>5.58 (4.93)</td>
<td>7.9 (5.16)</td>
<td>5.95 (5.64)</td>
<td>12.2 (6.00)</td>
<td>6.81 (6.00)</td>
<td>29.9 (6.15)</td>
<td>7.41 (6.00)</td>
<td>41.3 (6.00)</td>
<td>7.57 (6.00)</td>
<td>44.1 (6.00)</td>
</tr>
<tr>
<td>Depressed mood [0-18]</td>
<td>3.79 (4.29)</td>
<td>5.1 (4.26)</td>
<td>3.80 (4.27)</td>
<td>12.5 (4.63)</td>
<td>4.04 (4.63)</td>
<td>30.5 (4.63)</td>
<td>4.64 (4.63)</td>
<td>41.5 (4.63)</td>
<td>5.02 (4.63)</td>
<td>44.8 (4.63)</td>
</tr>
<tr>
<td>Positive affect [0-9]</td>
<td>6.67</td>
<td>4.9</td>
<td>5.96</td>
<td>12.4</td>
<td>6.09</td>
<td>30.8</td>
<td>6.04</td>
<td>41.6</td>
<td>6.07</td>
<td>44.5</td>
</tr>
<tr>
<td>----------------------</td>
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<td>------</td>
</tr>
<tr>
<td></td>
<td>(1.94)</td>
<td>(2.46)</td>
<td>(2.49)</td>
<td>(2.43)</td>
<td>(2.35)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anxiety [0-20]</td>
<td>3.03</td>
<td>3.3</td>
<td>3.49</td>
<td>10.8</td>
<td>3.97</td>
<td>29.5</td>
<td>4.22</td>
<td>40.8</td>
<td>4.28</td>
<td>43.8</td>
</tr>
<tr>
<td></td>
<td>(3.54)</td>
<td>(3.97)</td>
<td>(4.18)</td>
<td>(4.42)</td>
<td>(4.53)</td>
<td></td>
<td></td>
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</tr>
</tbody>
</table>

Note: All the means and standard deviations were estimated using full information maximum likelihood estimation (FIML).
### Table 3

Model fit statistics for the tests of invariance in the four cross-lagged models, tests for equilibrium and omitted variables, model fit indices after adjusting for covariate-effects, as well as for the final structural models.

<table>
<thead>
<tr>
<th>Model tested</th>
<th>$\chi^2$</th>
<th>df</th>
<th>$P$</th>
<th>RMSEA</th>
<th>RMSEA 90% CI</th>
<th>CFI</th>
<th>ΔCFI</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Measurement model estimates</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td><strong>IS and pain</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Null model</td>
<td>39626.52</td>
<td>435</td>
<td>&lt;.001</td>
<td>---</td>
<td>---</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td>Configural invariance</td>
<td>1256.18</td>
<td>318</td>
<td>&lt;.001</td>
<td>.033</td>
<td>.031 - .035</td>
<td>.976</td>
<td>---</td>
</tr>
<tr>
<td>Metric (weak) invariance</td>
<td>1311.94</td>
<td>334</td>
<td>&lt;.001</td>
<td>.033</td>
<td>.031 - .034</td>
<td>.975</td>
<td>.001</td>
</tr>
<tr>
<td>Scalar (strong) invariance</td>
<td>1424.77</td>
<td>350</td>
<td>&lt;.001</td>
<td>.033</td>
<td>.032 - .035</td>
<td>.973</td>
<td>.002</td>
</tr>
<tr>
<td>- With covariate controls</td>
<td>2053.44</td>
<td>462</td>
<td>&lt;.001</td>
<td>.035</td>
<td>.034 - .037</td>
<td>.960</td>
<td>---</td>
</tr>
<tr>
<td>Test of equilibrium</td>
<td>2256.66</td>
<td>502</td>
<td>&lt;.001</td>
<td>.036</td>
<td>.034 - .037</td>
<td>.955</td>
<td>.005</td>
</tr>
<tr>
<td><strong>IS, pain and depressed mood</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
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<tr>
<td>Null model</td>
<td>70653.40</td>
<td>1170</td>
<td>&lt;.001</td>
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<tr>
<td>Configural invariance</td>
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<td>&lt;.001</td>
<td>.026</td>
<td>.024 - .027</td>
<td>.979</td>
<td>---</td>
</tr>
<tr>
<td>Model</td>
<td>Chi-Square</td>
<td>DF</td>
<td>p-value</td>
<td>CFI</td>
<td>RMSEA</td>
<td>90% CI for RMSEA</td>
<td>p-value for RMSEA</td>
</tr>
<tr>
<td>------------------------------------------</td>
<td>------------</td>
<td>------</td>
<td>---------</td>
<td>------</td>
<td>-------</td>
<td>------------------</td>
<td>------------------</td>
</tr>
<tr>
<td>Metric (weak) invariance</td>
<td>2361.11</td>
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<td>&lt;.001</td>
<td>.027</td>
<td>.025 - .028</td>
<td>.977</td>
<td>.002</td>
</tr>
<tr>
<td>Scalar (strong) invariance</td>
<td>2639.54</td>
<td>825</td>
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<td>.027 - .029</td>
<td>.973</td>
<td>.004</td>
</tr>
<tr>
<td>- With covariate controls</td>
<td>4069.56</td>
<td>993</td>
<td>&lt;.001</td>
<td>.034</td>
<td>.032 - .035</td>
<td>.956</td>
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</tr>
<tr>
<td>Test of equilibrium</td>
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<td>1059</td>
<td>&lt;.001</td>
<td>.034</td>
<td>.033 - .035</td>
<td>.952</td>
<td>.004</td>
</tr>
<tr>
<td>IS, pain and positive affect</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Null model</td>
<td>51486.01</td>
<td>990</td>
<td>&lt;.001</td>
<td>---</td>
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<tr>
<td>Configural invariance</td>
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<td>&lt;.001</td>
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<td>.023 - .025</td>
<td>.975</td>
<td>---</td>
</tr>
<tr>
<td>Metric (weak) invariance</td>
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<td>801</td>
<td>&lt;.001</td>
<td>.024</td>
<td>.023 - .026</td>
<td>.974</td>
<td>.001</td>
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<td>.025 - .028</td>
<td>.968</td>
<td>.006</td>
</tr>
<tr>
<td>- With covariate controls</td>
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<td>993</td>
<td>&lt;.001</td>
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<td>.027 - .029</td>
<td>.957</td>
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</tr>
<tr>
<td>Test of equilibrium</td>
<td>3467.18</td>
<td>1059</td>
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<td>.028 - .030</td>
<td>.952</td>
<td>.005</td>
</tr>
<tr>
<td>IS, pain and anxious mood</td>
<td></td>
<td></td>
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<td></td>
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</tr>
<tr>
<td>Null model</td>
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<td>990</td>
<td>&lt;.001</td>
<td>---</td>
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<td>---</td>
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<tr>
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<td>&lt;.001</td>
<td>.025</td>
<td>.023 - .026</td>
<td>.980</td>
<td>---</td>
</tr>
<tr>
<td>Metric (weak) invariance</td>
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<td>801</td>
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<td>.025</td>
<td>.024 - .026</td>
<td>.979</td>
<td>.001</td>
</tr>
<tr>
<td>Scalar (strong) invariance</td>
<td>2365.93</td>
<td>825</td>
<td>&lt;.001</td>
<td>.026</td>
<td>.025 - .027</td>
<td>.976</td>
<td>.003</td>
</tr>
<tr>
<td>Model Description</td>
<td>Chi-Sq.</td>
<td>DF</td>
<td>p</td>
<td>Lower 95%</td>
<td>Upper 95%</td>
<td>RMSEA</td>
<td>SRMR</td>
</tr>
<tr>
<td>-------------------------------------------</td>
<td>---------</td>
<td>----</td>
<td>-------</td>
<td>-----------</td>
<td>-----------</td>
<td>--------</td>
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</tr>
<tr>
<td>With covariate controls</td>
<td>3447.17</td>
<td>993</td>
<td>&lt;.001</td>
<td>.030</td>
<td>.029 - .031</td>
<td>.963</td>
<td>---</td>
</tr>
<tr>
<td>Test of equilibrium</td>
<td>3815.01</td>
<td>1059</td>
<td>&lt;.001</td>
<td>.031</td>
<td>.030 - .032</td>
<td>.958</td>
<td>.005</td>
</tr>
</tbody>
</table>

**Longitudinal structural model estimates**

<table>
<thead>
<tr>
<th>Model Description</th>
<th>Chi-Sq.</th>
<th>DF</th>
<th>p</th>
<th>Lower 95%</th>
<th>Upper 95%</th>
<th>RMSEA</th>
<th>SRMR</th>
</tr>
</thead>
<tbody>
<tr>
<td>IS and pain</td>
<td>2287.12</td>
<td>488</td>
<td>&lt;.001</td>
<td>.037</td>
<td>.035 - .038</td>
<td>.955</td>
<td>.005</td>
</tr>
<tr>
<td>IS, pain and depressed mood</td>
<td>4335.15</td>
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<td>.034</td>
<td>.033 - .035</td>
<td>.953</td>
<td>.003</td>
</tr>
<tr>
<td>IS, pain and positive affect</td>
<td>3579.35</td>
<td>1053</td>
<td>&lt;.001</td>
<td>.029</td>
<td>.028 - .031</td>
<td>.951</td>
<td>.006</td>
</tr>
<tr>
<td>IS, pain and anxious mood</td>
<td>3843.66</td>
<td>1053</td>
<td>&lt;.001</td>
<td>.031</td>
<td>.030 - .032</td>
<td>.958</td>
<td>.005</td>
</tr>
</tbody>
</table>

Note. All structural models include the longitudinal CFA’s of insomnia symptoms and pain. IS = insomnia symptoms. Covariate controls were gender, age, immigrant status and SES.
Table 4

Unstandardized overall effects – indirect, direct and total – in each of the four SEM-models.

<table>
<thead>
<tr>
<th>Model / Effect</th>
<th>Indirect</th>
<th>99% CI</th>
<th>Direct</th>
<th>Total</th>
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</thead>
<tbody>
<tr>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td><strong>IS and pain</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Overall IS 1 → Pain 5</td>
<td>---</td>
<td>---</td>
<td>0.244⁹</td>
<td>0.244⁹</td>
</tr>
<tr>
<td>Overall Pain 1 → IS 5</td>
<td>---</td>
<td>---</td>
<td>0.087⁹</td>
<td>0.087⁹</td>
</tr>
<tr>
<td><strong>IS, pain and depressed mood</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Overall IS 1 → Pain 5</td>
<td>0.029</td>
<td>0.011 to 0.054</td>
<td>0.143⁹</td>
<td>0.172⁹</td>
</tr>
<tr>
<td>Overall Pain 1 → IS 5</td>
<td>0.004</td>
<td>0.000 to 0.011</td>
<td>0.064⁹</td>
<td>0.068⁹</td>
</tr>
<tr>
<td><strong>IS, pain and positive affect</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Overall IS 1 → Pain 5</td>
<td>0.005</td>
<td>- 0.007 to 0.017</td>
<td>0.231⁹</td>
<td>0.236⁹</td>
</tr>
<tr>
<td>Overall Pain 1 → IS 5</td>
<td>0.000</td>
<td>- 0.004 to 0.003</td>
<td>0.085⁹</td>
<td>0.085⁹</td>
</tr>
<tr>
<td><strong>IS, pain and anxious mood</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Overall IS 1 → Pain 5</td>
<td>0.031</td>
<td>0.010 to 0.061</td>
<td>0.169⁹</td>
<td>0.200⁹</td>
</tr>
<tr>
<td>Overall Pain 1 → IS 5</td>
<td>0.004</td>
<td>0.000 to 0.012</td>
<td>0.071⁹</td>
<td>0.075⁹</td>
</tr>
</tbody>
</table>

Note. IS = Insomnia symptoms.

⁹ Confidence interval does not contain zero.
Figure 1. Depiction of the latent cross-lagged panel model between insomnia symptoms and pain. For simplicity, only the structural portion of the model is displayed, and the underlying measurement model with multiple indicators is omitted, as well as cross-sectional correlations. All latent variables are correlated cross-sectionally. The estimates are with covariate controls included. Dotted arrows represent non-significant paths. IS=insomnia symptoms. All path coefficients are standardized.

Figure 2. Depiction of the latent autoregressive longitudinal full mediation analysis, with depressed mood as mediator. For simplicity, only the structural portion of the model is displayed, the underlying measurement models as well as cross-sectional indicators are omitted.
The estimates are with covariate controls included. Dotted arrows represent non-significant paths. IS=insomnia symptoms, DM = depressed mood. All path coefficients are standardized.

**Figure 3.** Depiction of the latent autoregressive longitudinal full mediation analysis, with positive affect as mediator. For simplicity, the representation of the measurement model, as well as cross-sectional correlations, are omitted. The estimates are with covariate controls included. Dotted arrows represent non-significant paths. IS=insomnia symptoms, PA=positive affect. All path coefficients are standardized.
Figure 4. Depiction of the latent autoregressive longitudinal full mediation analysis, with AM as mediator. For simplicity, only the structural portion of the model is displayed, and the underlying measurement model with multiple indicators is omitted, as well as cross-sectional correlations. The estimates are with covariate controls included. Dotted arrows represent non-significant paths. IS=insomnia symptoms, AM = anxious mood. All path coefficients are standardized.
Figure 5. A model of the sleep-pain relationship in pediatric persistent pain populations. Modified from Valrie and colleagues [62], after permission from the author. The greyed areas were not a focus in the current study. Contrary to the original model, the arrows are unidirectional from insomnia symptoms to mood, and from mood to pain. The arrow from insomnia symptoms to pain is larger than the arrow from pain to insomnia symptoms.
**Supplementary Digital Content 1.** Text and Figures describing the specific indirect mediation effects and the analyses of overall indirect effects. pdf

**Supplementary Digital Content 2.** Text that describes analyses of baseline data. pdf

**Supplementary Digital Content 3.** Text and Figures describing the configural longitudinal measurement models of pain, insomnia symptoms, depressed mood, positive affect and anxious mood. pdf

**Supplementary Digital Content 4.** Text and Figures showing details of the structural model building, as well as the fully specified mediation models. pdf

**Supplementary Digital Content 5.** Text of syntax of the analyses conducted in Mplus, version 8. Docx

**Supplementary Digital Content 6.** Text of the variance-covariance matrix that the study’s analyses were based on. Docx