

Trajectories of sleep problems from childhood to adolescence: a population-based longitudinal study from Norway

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SUMMARY

The aim of the current study was to assess the development and stability of sleep problems from childhood to late adolescence. This was a longitudinal cohort study of 2026 children, who completed three comprehensive health surveys, at age 7–9, 11–13 and 16–19 years. Data on difficulties with initiating and/or maintaining sleep (DIMS: assessed using a single item) and time in bed (TIB) were collected at all three waves, while insomnia assessed in line with the DSM-5 criteria and sleep duration were also assessed in the last wave. Negative binomial regression analyses were used to examine prospective associations. Sleep problems in 7–9-year-old children were found to persist into late adolescence for approximately one-third of the participants, both with regard to DIMS and short TIB. Children having chronic DIMS at the first two waves had nearly twice the risk of fulfilling the DSM-5 criteria later for insomnia in late adolescence [adjusted relative risk RR: 1.91]. Short TIB at age 11–13 was also associated with increased risk of subsequent short sleep duration (adjusted RR: 1.32) and TIB (adjusted RR: 1.40). These findings have important implications for practitioners and families. Although the majority of children will outgrow their problems once they reach late adolescence, the results also demonstrate that sleep problems are likely to become chronic for one in every third child with a sleep problem early in life. Given the many negative consequences of insomnia in adulthood, these findings call for increased awareness of childhood sleep problems as a public health concern.

INTRODUCTION

Sleep problems are common across the lifespan, including childhood and adolescence. Reports have shown that as many as three in four 11–15-year-olds have some degree of sleeping difficulty (Ipsiroglu *et al.*, 2002), and approximately 20% of 16–19-year-old adolescents fulfil the diagnostic criteria for an insomnia disorder diagnosed in accordance with the latest revision of the DSM classification system (Hysing *et al.*, 2013). Difficulties initiating or maintaining sleep (DIMS) are among the most common sleep problems (Hysing *et al.*, 2013; Morrison *et al.*, 1992), and a recent longitudinal study from Norway showed that DIMS in young

adolescents (aged 13) typically persist well into adulthood (Hayley *et al.*, 2015b). A related report found that short sleep duration in 15-year-old adolescents predicted short sleep duration significantly at age 30 years (Hayley *et al.*, 2015a). Unfortunately, sleep problems in children and adolescents seem to be rising (Pallesen *et al.*, 2008).

The stability and developmental course of sleep problems and sleep duration among young children throughout early adolescence are more uncertain (Jenni *et al.*, 2005, 2007; Krueger and Friedman, 2009). To the best of our knowledge, no studies have investigated previously the extent to which sleep problems in childhood extend into late adolescence. Most of the previous literature is based on cross-sectional

research that compare different age groups. Due to cohort effects and sampling differences, these studies may not depict developmental patterns correctly.

There are longitudinal studies that have investigated sleep patterns in the same individuals over time, covering parts of the age-span from childhood to late adolescence. Based on parent report, the DIMS rates were reduced from 11 to 13-year-olds in one longitudinal study (Laberge *et al.*, 2001), and a twin study found a decreasing trend for insomnia symptoms from 8 to 15 years (Barclay *et al.*, 2015). Less is known about the development into later adolescence. Late adolescence is an important period as changes in a wide range of biological (e.g. delayed circadian rhythms, slower buildup of the sleep homeostatic factor), psychological (e.g. identity formation), social (greater autonomy) and educational (e.g. starting higher education) processes co-occur, culminating in complex interactions, often resulting in significantly impaired sleep (Carskadon, 2011).

Gender differences in sleep problems develop typically during puberty, and due to gender-specific timing of pubertal development girls have an earlier change towards shorter sleep duration and a delayed sleep phase than boys (Sadeh *et al.*, 2009). While sleep problems are equal for boys and girls in prepubertal children, girls have a higher rate of insomnia in adolescence (Hysing *et al.*, 2013). Thus, it is likely that the development of trajectories for sleep problems is different for boys and girls.

Based on these considerations, the aims of the current study were to (1) assess the natural development and stability of sleep problems across three waves, from ages 7–9, 11–13 and 16–19 years, and (2) examine if sleep problems in the first two waves predicted later sleep problems, including DSM-5 insomnia disorder in late adolescence, while adjusting for potential confounders. Additionally, gender differences was examined on an exploratory basis.

METHODS

Participants

Data stem from the first, second and fourth waves of the Bergen Child Study (BCS), carried out in autumn 2002, spring 2006 and winter/spring 2012, respectively. The BCS represents a longitudinal total population study of children in all public and private schools in the city of Bergen, Norway. The fourth wave of the BCS is also called the youth@hordaland-survey, and this wave included all adolescents in Hordaland County, in which the city of Bergen is the largest metropolitan area. The protocol and the population of the BCS are described in detail elsewhere (Heiervang *et al.*, 2007). In short, in the first wave, the target population consisted of 9430 primary school children aged 7–9 years, for whom 7007 (74.3%) parents provided informed consent to participate. The second wave was performed 4 years later, and 5683 children, now aged 11–13 years, participated. The fourth wave was conducted again 6 years later, in which

10 254 of the 19 439 invited adolescents participated (participation rate of 53%). The longitudinal sample in the current study with complete data on all three waves included a total of 2026 individuals of the original study population, making the participation rate 29% of the 7007 children in the first wave and 35.7% of the children who completed the second wave. Of note, data from an additional wave conducted when the children were aged 13–15 years, were not included in the current study, due to a very low response (18%) rate at that wave. For ease of reading, the three waves used in the current study will be labelled T1, T2 and T3, respectively.

Instruments

For the present study, parent-reported information is used for T1 and T2, whereas the adolescents' self-report is used for T3.

Difficulties with initiating and/or maintaining sleep (DIMS) – all waves

Sleep problems at all three waves were assessed by reports with one question encompassing difficulties with initiating and/or maintaining sleep, rated on a three-point Likert scale ('not agree', 'partly agree' and 'agree'). To avoid data cells that are too small, especially in the longitudinal sample, dichotomous variables were used for the purpose of the present study, in which responding either 'agree' or 'partly agree' was defined as having sleep problems. This operationalization has been applied previously in the BCS (Sivertsen *et al.*, 2009). No data on the severity or duration of the DIMS were available.

Insomnia – T3

Insomnia at T3 was operationalized according to the DSM-5 criteria for insomnia (American Psychiatric Association, 2013): DIMS at least three times a week, with a duration of 3 months or more, as well as tiredness or sleepiness on at least 3 days per week. Detailed information on the sleep variables in T3 has been published elsewhere (Hysing *et al.*, 2013).

Time in bed (T2 and T3) and sleep duration (T3)

At T2, the parents reported time spent in bed (TIB), operationalized as the difference between the usual bedtime and rising time. No data were available on sleep onset latency (SOL) and wake after sleep onset (WASO). For the purposes of the present study, TIB was analysed both continuously and categorically by quartiles: lowest quartile: <9:30, second quartile: 9:30–9:45, third quartile 9:45–10:00 and highest quartile >10:00.

At T3, self-reported bedtime and rise time were indicated in hours and minutes using a scroll-down menu with 5-min intervals and were reported separately for weekends and

weekdays. SOL and WASO were reported in hours and minutes using a scroll-down menu with 5-min intervals, and sleep duration was defined as TIB minus (SOL + WASO). For the purposes of the present study, sleep duration was used both continuously and categorically by quartiles: lowest quartile: <6:00, second quartile: 6:00–6:45, third quartile 6:45–7:30 and highest quartile >7:30. The corresponding quartile cutoffs for TIB at T3 were 7:00, 7:30 and 8:00.

Demographical information – T3

Gender and date of birth were identified through personal identity number in the Norwegian National Population Register. Socioeconomic status (SES) was assessed both by parental education and perceived family financial circumstances. Maternal and paternal education were reported separately with three response options; 'primary school', 'secondary school' and 'college or university'. Perceived family affluence (i.e. how well off the adolescent perceived their family to be) was assessed by asking the adolescents how their family affluence is compared to most others. The response alternatives were (1) 'better family affluence', (2) 'approximately like most others' and (3) 'poorer family affluence'. A recent report from the second wave of the Bergen Child Study, when the children were 11–13 years old, showed that the rating of family affluence correlated reasonably well ($r = 0.586$, $P < 0.001$) with data on taxable monetary income, which were available for a subsample of 642 participants (Bøe *et al.*, 2014). The adolescents also indicated if they lived with both their parents (yes/no).

Statistics

All analyses were performed using the SPSS statistical software package version 22 (SPSS Inc., Chicago, IL, USA). Negative binomial regression analyses were used to examine associations between sleep variables at waves 1 and 2 and subsequent sleep variables at wave 3. Rather than the more commonly used logistic regressions [producing odds ratio (OR)], we used negative binomial regressions [producing relative risk (RR)], which provides more correct estimates when the prevalence of the outcome of interest (in this case insomnia or short sleep duration) is relatively high. ORs can overestimate an effect size when the outcomes are frequent (Davies *et al.*, 1998). Both crude/unadjusted and adjusted analyses were conducted. Adjustment variables included gender, parental education, parents not living together and family financial circumstances. We also tested for interactions between gender and sleep variables by entering the product of these variables in separate blocks. Missing data were handled using listwise deletion.

Ethics

All three study waves were approved by the Regional Committee for Medical and Health Research Ethics in

Western Norway. For the first two waves, written informed consent was obtained from all parents included in this study. For T3, the adolescents' parents were informed about the study while the adolescents themselves consented to participate in the study, as Norwegian regulations state that individuals aged 16 years and older are required to consent themselves. Participants received no payment to participate.

RESULTS

Sample characteristics

The longitudinal sample included in the present study comprised 2226 individuals. Fifty-seven per cent of the children were girls, and the educational level of the parents was high compared with the national average (Statistics Norway, 2013). Specifically, 61% and 57% of the mothers and fathers, respectively, had an educational level beyond high school (see Table 1 for full demographic variables for the sample).

Adolescents who completed all three assessments (responders) were more likely to be female, as well as having parents with higher education, and came from a family with better financial circumstances compared to individuals who did not complete all three assessments (non-responders) (all P s < 0.001; data not shown). There were no significant differences in any of the sleep measures between responders and non-responders.

Sleep characteristics

As depicted in Fig. 1, 8.5% of the parents reported that their child had DIMS ('true' or 'partly true') at T1 (age 7–9) compared to 12.7% at T2 (age 11–13). In contrast, approximately one-third of the adolescents reported DIMS at T3 (age 16–19). At the first two waves, boys had marginally

Table 1 Demographic variables at age 16–19 (T3) in the longitudinal cohort ($n = 2226$)

Demographics at T3	%/mean
<i>n</i>	2226
Girls	56.5%
Maternal education	
Primary school	7.6%
High school	31.9%
University/college	60.5%
Paternal education	
Primary school	7.2%
High school	35.8%
University/college	57.1%
Parents not living together, %	28.1%
Family affluence, %	
Approximately like most others	67.1%
Better family affluence	28.5%
Poorer family affluence	4.3%

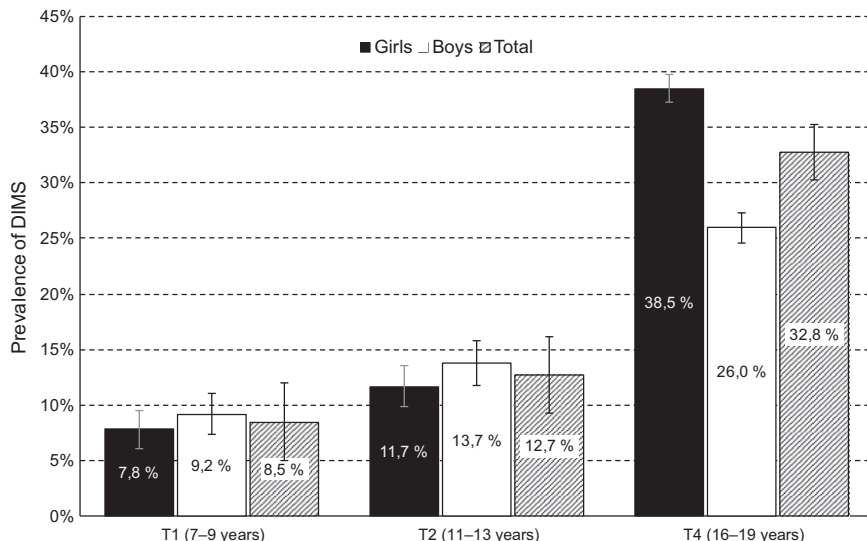


Figure 1. Prevalence of boys and girls reporting difficulties with initiating and/or maintaining sleep (DIMS) ('true' or partly 'true') across the three waves in the longitudinal sample of the Bergen Child Study. Error bars represent 95% confidence intervals.

more DIMS than girls, whereas the opposite was found at T3, 38.5% of the girls reporting DIMS compared to 26% of the boys.

Trajectories of DIMS

Figure 2a depicts the trajectories of DIMS from age 7-9 (T1) to age 11-13 (T2) and age 16-19 (T3). Of the 8.6% of the children who had DIMS at T1, approximately one-third also had sleep problems at both T2 and T3. In contrast, 26.2% of these children had DIMS at neither T2 nor T3 (see Fig. 1 for details). Figure 2B depicts the opposite direction: of the 35.8% of adolescents who had DIMS at T3, 7.4% had DIMS at all three assessment points. For this group, the majority (77.5%) reported no DIMS at the first two assessment points (Fig. 2b).

Trajectories of time in bed (TIB)

Figure 3a depicts the trajectories of TIB over the last two assessment points, from age 11-13 to age 16-19. Among

children in the lowest TIB quartile at T2, 29.5% were still in the lowest quartile 6 years later. In comparison, among those in the lowest TIB quartile at T3, 22.8% were classified as being in the lowest quartile also at T2 (see Fig. 3b for details).

Childhood DIMS as predictors of sleep problems in adolescence

A series of negative binomial regression analyses were conducted to examine to what extent different patterns of DIMS at T1 and T2 predicted subsequent sleep problems at T3 (age 16-19). As detailed in Table 2, compared to not having DIMS at T1 and T2, having DIMS at both these points was associated with a RR of 1.94 (95% CI: 1.63-2.32) for DIMS at T3. Adjusting for potential confounders (gender, parental education, parents not living together, family financial circumstances) did not reduce the association (RR: 2.00; 95% CI: 1.68-2.38; see Table 2 for details). Reporting DIMS at T1 only or T2 only was associated with a RR of 1.39 and 1.44, respectively, and also these associations were not reduced in the fully adjusted models. Adding the interaction

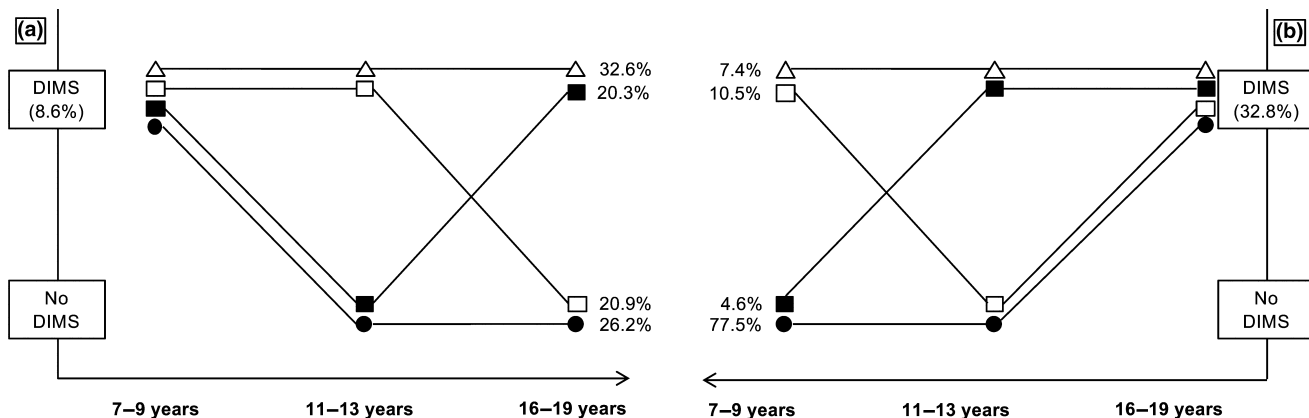


Figure 2. Trajectories of difficulties initiating and/or maintaining sleep (DIMS) at all three time-points in the Bergen Child Study.

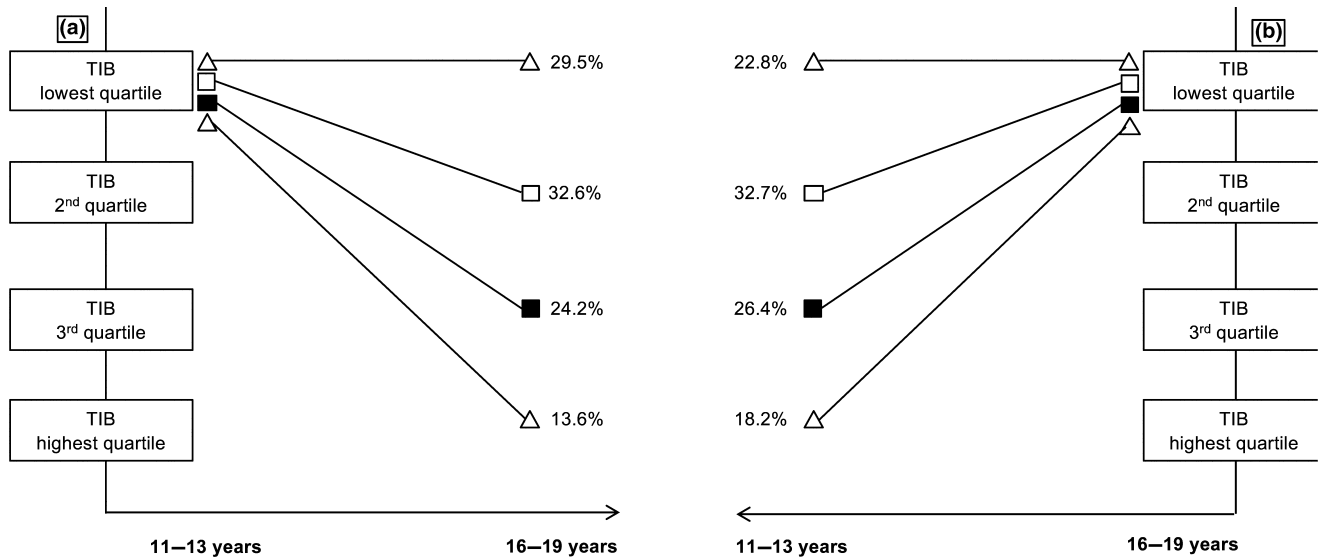


Figure 3. Trajectories of time in bed (by quartiles) at age 11–13 and 16–19 in the Bergen Child Study.

Table 2 Pattern of DIMS at age 7–9 (T1) and 11–13 (T2) as risk factors for DIMS and DSM-5 insomnia at age 16–19 (T3)

	DIMS at age 16–19 (T3)			
	Unadjusted model		Adjusted model*	
	RR	95% CI	RR	95% CI
DIMS pattern at T1 and T2				
No DIMS	1.00	–	1.00	–
DIMS T1 only	1.39	1.08–1.80	1.37	1.06–1.76
DIMS T2 only	1.44	1.21–1.72	1.46	1.22–1.75
Chronic insomnia	1.94	1.63–2.32	2.00	1.68–2.38
DSM-5 insomnia at age 16–19 (T3)				
	Unadjusted model		Adjusted model*	
	RR	95% CI	RR	95% CI
	DIMS pattern at T1 and T2			
No DIMS	1.00	–	1.00	–
DIMS T1 only	1.67	1.16–2.38	1.62	1.14–2.30
DIMS T2 only	1.54	1.18–2.00	1.58	1.20–2.07
Chronic insomnia	1.83	1.33–2.51	1.91	1.38–2.64

RR, relative risk; CI, confidence interval; DIMS, difficulties initiating and/or maintaining sleep
 *Adjusted for gender, parental education, parents not living together and family affluence.

term between gender and DIMS did not change the model significantly ($P = 0.67$).

Similar patterns were observed for DIMS at T1 and T2 predicting DSM-5 insomnia at T3. Chronic DIMS (DIMS at both T1 and T2) was associated with a relative risk of 1.83 (95% CI: 1.33–2.51) for subsequent insomnia at T3, and this association also remained unchanged when adjusting for gender, parental education, parents not living together and

family financial circumstances (RR: 1.91; 95% CI: 1.38–2.64). There was no significant interaction between gender and DIMS ($P = 0.22$).

TIB as a risk factor for short sleep duration and TIB in adolescence

As detailed in Table 3, short TIB at age 11–13 was a significant risk factor for both short TIB and short sleep duration at age 16–19. For example, being in the lowest TIB at T2 was associated with a RR of 1.35 (95% CI: 1.22–1.49) for subsequent short sleep duration (lowest quartile: <6 h) at T3 (age 16–19). Adjusting for gender, parental education, parents not living together and family financial circumstances did not attenuate the risk, and there was also no significant interaction term between gender and TIB ($P = 0.77$). In addition, all associations exhibited a dose–response pattern; for example, having a TIB at T2 in the second quartile still predicted sleep duration significantly in the second quartile at T3, but the effect-sizes were smaller (adjusted RR: 1.13) (see Table 3 for details).

DISCUSSION

In this population-based longitudinal study, childhood sleep problems in 7–9-year-old children were found to persist into late adolescence (aged 16–19) for approximately one-third of the participants. This pattern of findings held for both DIMS and short TIB. Children having chronic DIMS at the first two waves had nearly twice the risk of later fulfilling the DSM-5 criteria for insomnia in late adolescence. Adjusting for demographics did not attenuate these associations. Also, reporting short TIB in childhood was associated with increased risk of subsequent short sleep duration and TIB.

These results suggest a pattern of relative stability of sleep duration/TIB and sleep problems throughout childhood into

Table 3 Time in bed (TIB) at age 11–13 as risk factor for short TIB and short sleep duration at age 16–19

Exposure at wave 2 (age 11–13)	Outcome: TIB at age 16–19 (reference category: 4th quartile: > 8:00)							
	1st quartile (<7:00)				2nd quartile (7:00–7:30)			
	Unadjusted model		Adjusted model [#]		Unadjusted model		Adjusted model*	
	RR	95% CI	RR	95% CI	RR	95% CI	RR	95% CI
TIB pattern								
Lowest quartile (<9:30)	1.39	1.26–1.53	1.40	1.26–1.54	1.17	1.09–1.25	1.16	1.09–1.25
2nd quartile (9:30–9:45)	1.21	1.10–1.33	1.22	1.11–1.34	1.10	1.03–1.17	1.10	1.03–1.17
3rd quartile (9:45–10:00)	1.09	0.98–1.20	1.09	0.98–1.21	1.00	0.93–1.07	0.99	0.92–1.06
Highest quartile (>10:00)	1.00	–	1.00	–	1.00	–	1.00	–
Exposure at wave 2	Outcome: sleep duration at age 16–19 (T3; reference category: 4th quartile: > 7:30)							
	1st quartile (<6:00)				2nd quartile (6:00–6:45)			
	Unadjusted model		Adjusted model*		Unadjusted model		Adjusted model*	
	RR	95% CI	RR	95% CI	RR	95% CI	RR	95% CI
TIB pattern								
Lowest quartile (< 9:30)	1.35	1.22–1.49	1.32	1.19–1.47	1.25	1.16–1.36	1.24	1.15–1.35
2nd quartile (9:30–9:45)	1.16	1.05–1.28	1.15	1.04–1.27	1.13	1.05–1.22	1.13	1.05–1.22
3rd quartile (9:45–10:00)	1.04	0.94–1.16	1.02	0.92–1.14	1.08	0.99–1.16	1.07	0.99–1.16
Highest quartile (>10:00)	1.00	–	1.00	–	1.00	–	1.00	–

RR, relative risk; CI, confidence interval.
[#]Adjusted for gender, parental education, parents not living together and family affluence.

later adolescence. The relative long-term stability of sleep duration has been shown previously in younger children (Jenni *et al.*, 2007). The fact that this stability seems to expand to late adolescence raises the possibility that this might be a trait-like characteristic. However, the picture is complex, and while the risk of later sleep problems is increased in children with early sleep problems, most of the children with sleep problems at one time-point will not report this at a later time-point. Further, the sharp increase in sleep problems over time, especially from early to late adolescence, necessarily means that many children without early sleep problems will develop sleep problems over time.

We found a gradual increase in sleep problems from early childhood to adolescence, with the most marked increase towards late adolescence. This is in contrast to some of the few previous longitudinal studies reporting a decrease of sleep problems in early adolescence (Barclay *et al.*, 2015). While this could be due to methodological differences and sampling issues, cohort effects should also be considered (Keyes *et al.*, 2015). Further, the rise in sleep problems was most pronounced in late adolescence, a developmental period that is not covered typically in the previous studies. This finding raises the need for future research to determine the other pathways and contributors to the onset of sleep problems in the later adolescent years, in addition to childhood onset.

Early sleep problems were identified as an important predictor of insomnia in adolescence. Although beyond the

scope of the present study, an important next step is to explore the predictors of the developmental trajectories over and above earlier sleep problems. Genetic factors may account for more of the variance early in childhood, while environmental and psychological factors may be more pronounced in later adolescence (Barclay *et al.*, 2015). Identifying the environmental and psychological factors that account for the increase in insomnia symptoms, and further exploring if some early indicators may predict the development of later problems, may be especially important. It is noteworthy that while the rates of DIMS in boys was slightly higher than girls in the younger age groups, the rate of DIMS in girls was higher (38.5%) than in boys (26.0%) in the 16–19-year age group. Similar gender-dependent developmental patterns have also been observed regarding depression (Hankin, 2009). Studies have shown that puberty seems to be a risk factor for insomnia in girls but not in boys (Johnson *et al.*, 2006), which may explain some of these changes. A deeper understanding of the mechanisms that contribute to these patterns are important, given that adolescent sleep problems have been linked to a range of negative conditions and behaviours, including mental health problems (Roberts *et al.*, 1995; Sivertsen *et al.*, 2014), self-harm (Hysing *et al.*, 2015b; Wong and Brower, 2012) and alcohol problems (Hasler *et al.*, 2014; Sivertsen *et al.*, 2015), in addition to school non-attendance (Egger *et al.*, 2003; Hysing *et al.*, 2015a) and poor academic performance (Hysing *et al.*, 2016; Shochat *et al.*, 2014). It is also possible that early

mental health problems and depressive symptoms are candidates that account for the increase in insomnia symptoms, a possibility that needs further exploration (Patten *et al.*, 2000).

Some methodological limitations of the present study should be noted. Data stem from one county in Norway, and although the distribution of urban and rural areas reflects Norway as a whole, the possible limited generalizability must be taken into account. Also, attrition from the study could affect generalizability. In the present study, the longitudinal sample was substantially smaller than either of the individual waves. Unfortunately, the problem with non-participation in survey research seems to be rising (Morton *et al.*, 2012). Our non-response analyses showed that adolescents who completed all three assessments were more likely to be of female gender, and in general come from families of higher socioeconomic strata. Although previous research from the first wave of the Bergen Child Study (the same population as the current study) showed that non-participants had more psychological problems than participants (Stormark *et al.*, 2008), we found no significant differences in any of the sleep measures between those who completed all three waves versus those who did not. Another important limitation is the use of different informants across the waves: whereas sleep data (and other information) were provided by the parents for the first two waves, the adolescents themselves provided this information at age 16–19. Although it might seem ideal to retain the same informant across all waves, having parents assess their adolescent's sleep patterns and behaviour during their high school years is not likely to provide accurate data with regard to sleep in this age group. It should also be noted that previous research has shown a high correlation ($r = 0.90$) between parent-reported sleep duration and actigraphy-recorded sleep duration in young children, although with a slight overestimation of sleep duration from parents on the self-report (Sekine *et al.*, 2002). Nevertheless, the results should be interpreted with caution due to the combination of different informants. Along the same lines, a single item was used to assess difficulties with initiating and/or maintaining sleep, and no objective measures were used to assess sleep problems. Although self-reported sleep parameters, including SOL and WASO, differ typically from those obtained from objective assessments (Lauderdale *et al.*, 2008), recent studies have shown that such self-report sleep assessments can be recommended for the characterization of sleep parameters in both clinical and population-based research (Zinkhan *et al.*, 2014). Also, the accuracy of self-reported SOL and WASO is generally better among adolescents than in older adults (Dillon *et al.*, 2015), and a study of young adolescents in Hong Kong recently found good agreement between actigraphy-measured and questionnaire-reported sleep durations (Kong *et al.*, 2011). Moreover, it should be noted that the use of quartiles for categorization of sleep duration variables may yield cutoffs that do not necessarily correspond with clinical

recommendations. For example, the recommended sleep duration from the National Sleep Foundation for adolescents aged 16–19 is 8–10 h; the cutoff for the highest quartile was as low as 7.5 h. Finally, the presence of comorbid mental health symptoms and disorders were not examined in the current study. Future studies should examine the role of somatic and mental health symptoms as possible moderators and mediators of the course of insomnia.

Clinical implications

Although the findings show that the majority of children will outgrow their problems once they reach late adolescence, the current study also demonstrates that the sleep problem is likely to become chronic for approximately one in every third child who has a sleep problem early in life. Given the many negative consequences of insomnia in adulthood, and the pattern of relative stability in sleep duration/time in bed and sleep problems, the present findings call for increased awareness of childhood sleep problems as a public health concern. The findings also raise the possibility that early intervention efforts, targeting the 7–9-year-old age groups, may be a worthy investment for removing youth from the trajectory toward chronic sleep problems. The stability of both sleep duration/TIB and sleep problems may have several practical implications for practitioners. When understanding the individual's sleep need, a sleep history is of the essence to be able to combine the general expected sleep need for the age group with the individual's personal sleep needs. Further, the stability of sleep problems over time could indicate a proactive role for the practitioner, as many of the children will not merely outgrow their sleep problems and will need intervention.

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AUTHOR CONTRIBUTIONS

BS and MH were involved in acquisition of data. BS and MH were responsible for conception and design of the study, conducted the statistical analysis and drafted the manuscript. AH and SP gave critical revision of the manuscript for important intellectual content. BS and MH had full access to all the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis. All authors read and approved the final manuscript.

CONFLICT OF INTEREST

None reported.

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