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Why Can't My Child Sleep and Will There be Long-Term Consequences? Lessons from Prospective Community-Based Studies

Commentary on Zhang et al. Longitudinal course and outcome of chronic insomnia in Hong Kong Chinese children: a 5-year follow-up study of a community-based cohort. SLEEP 2011;34:1395-1402.

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In this edition of *SLEEP*, Jihui Zhang and colleagues[1] follow the development of over one thousand five hundred Chinese children for five years. The main focus of the study is the predictors and outcome of chronic insomnia—and the mixed pattern of results reported adds nicely to the growing body of data showing complex and likely bidirectional associations between sleep disturbances experienced in youth and many other difficulties. Amongst associations reported, the authors' note that chronic medical disorders at baseline were associated with the persistence of insomnia over time; conversely insomnia at baseline was associated with a health difficulty (specifically frequent laryngopharyngitis) assessed at follow-up.

In addition to complementing previous work, the report by Zhang and colleagues also reveals some, perhaps unexpected results, including the finding that baseline sleep disturbance did not predict "poor mental health" longitudinally. While this result differs from a host of other reports,[2] such discrepancies reveal the need to unpick associations in order to understand the driving mechanisms. For example, drawing distinctions within "poor mental health" could have been illuminating. This discrepancy could also be due to the specific sleep phenotype under investigation. The focus of the study by Zhang and colleagues[1] was on "chronic insomnia" assessed by parents during childhood, and self-report during adolescence. Different conceptualizations and measurements of sleep appear to be associated with other phenotypes in different ways.[2] Some methods of assessment are more accurate than others in assessing children's sleep (e.g., parents may have limited knowledge of periods during which their children are awake at night). Future work needs to use multi-methods to assess sleep in order to decipher exactly which components of sleep (e.g., length, quality, regularity) are key in driving associations with other phenotypes.

An interesting feature of the study by Zhang and colleagues is the focus on the transition from childhood to adolescence, a time of great change both socially and physiologically,[3] and a period during which sleep changes in many ways, including starting later at night and extending further into the morning.[4] During adolescence, sex differences in the prevalence of certain disorders emerge for the first time. Indeed, epidemiological data suggest that the female bias in the prevalence of depression (which is commonly associated with sleep disturbances, see DSM, IV)[5] emerges after the age of 13 years.[6] Interestingly, no sex differences in chronic insomnia were reported in the study by Zhang and colleagues who focused on participants aged 13.7 years at follow-up.[1] This suggests that the sex difference for chronic insomnia may appear later in life and further research needs to explicate exactly when insomnia-related sex differences become manifest.

A novel feature of the report by Zhang and colleagues is the focus on a non-Western population. This is particularly noteworthy as sleep in children may be influenced by cultural practices, social expectations, as well as geographical location (and associated levels of light, heat etc). Indeed, a recent report suggests that parents from predominantly Asian (e.g., Hong Kong) as compared to Caucasian (e.g., UK) countries are more likely to report sleep problems in their infants/toddlers.[7] Associations based on results from one study, may not apply to other populations, so further studies of non-Western populations are required.

An additional strength of the study by Zhang and colleagues is the use of prospective longitudinal data from a community sample, rather than the reliance on retrospective data. A recent, thought-provoking report compared estimates of a range of psychiatric disorders (anxiety, depression, alcohol dependence, and cannabis dependence) from 18-32 years of age, using prospective and retrospective data.[8] The prevalence of psychiatric disorders was approximately twice as high when focusing on prospective as compared to retrospective data. This supports the notion that retrospective data should be viewed with caution.

The aforementioned points emphasize the importance of the study by Zhang and colleagues in furthering knowledge of longitudinal associations between sleep and associated factors. Research now needs to focus on the mechanisms underlying these associations. Explanations for the associations between sleep and associated difficulties have thus far been diverse (e.g., focusing on issues relevant to nosology, rater bias, genetic and environmental influences, as well as pathways including hormonal and psychological processes; see elsewhere for review).[2] As such, it is clear that the only way to fully understand the mechanisms underlying longitudinal associations is to take a multidisciplinary approach.

Research is also needed to further investigate the possibility of causal links between sleep and subsequent difficulties. Scientists are rightly cautious when talking about causal associations, but it is possible that sleeping poorly during development could *cause* later difficulties. Various lines of enquiry support this notion. For example, sleep restriction and exten-

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sion in children has been shown to influence neurobehavioral functioning.[9] Indeed, amongst findings reported, children who extended their sleep by approximately 35 minutes over three nights, showed improvements in their performance on a test assessing digit forward memory.[9] At the neural level, in response to negative emotional stimuli, sleep deprived (as compared to non-sleep deprived) adults appear to show a greater amygdala response and weaker functional connectivity between the amygdala and the medial-prefrontal cortex (an area of the brain which helps regulate amygdala function).[10] This finding suggests that those who had been sleep deprived were less able to moderate emotional responses than others, which is consistent with a causal link between sleep and mood disorders. Such studies focus on short-term associations. Further investigations are needed to establish whether phenotypes may be causally linked longitudinally.

While establishing causation is no mean feat —when designing experiments and running analyses researchers could pay greater attention to the type of data that may be informative with regards to this issue. For example, in his seminal report, Hill and colleagues note that amongst other things, reporting dose-response associations as well as conducting experiments may be useful in establishing causal links.[11]

Regardless of whether childhood sleep disturbances *cause* later difficulties, the longitudinal data reported to date have implications for preventative techniques and the treatment of sleep problems in children. Indeed, these data suggest that sleep disturbances in childhood should not be dismissed or ignored as a typical part of development. Where help is sought for chronic sleep disturbances in children, other domains of functioning should be assessed. A focus on sleep may be useful clinically, as families may be more willing to seek help for and discuss sleep difficulties as compared to associated problems.

It is evocative to think that treating sleep disturbances in childhood will have long-term positive implications beyond improving sleep. This possibility needs to be tested empirically. Attempts to improve sleep in children are essential regardless of associated consequences since childhood sleep difficulties can cause suffering to children and their families.

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