

Does sleep mediate the effects of socioeconomic disadvantage on brain development?

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Socioeconomic disadvantage (SED) is associated with poorer neurocognitive outcomes and educational achievement in children. With as many as 40% of US children living in poverty or near poverty as of 2016,¹ this poses a major social and public health problem. SED is thought to affect children's development through several proximal risk factors, one of which is sleep disruption.

Insufficient or disrupted sleep is consistently linked to poorer neurocognitive development in children,² BUT...

Sleep disruption

Physiologic effects

Short-term

- Stress
- Somatic problems
- Psychosocial problems

Long-term

- CVD
- Obesity, T2 diabetes
- Cancer

Adapted from Medic et al²

2016 Consensus Statement of the American Academy of Sleep Medicine³

"Sleeping the number of recommended hours on a regular basis is associated with **better health outcomes** [...] Healthy sleep requires adequate **duration**, appropriate **timing**, good **quality**, **regularity**, and the **absence of sleep disturbances or disorders**"

... the causal link to SED is complex. Children and adolescents from SED backgrounds are at higher risk of having insufficient sleep due to disrupted sleep environments and family routines. There is evidence that this is one of the proximal risk factors through which SED affects neurological development, leading to worse cognitive, behavioural and educational outcomes.⁴ To date, however, few studies have focused specifically on primary school-aged children.

Socioeconomic disparities in sleep duration are associated with cortical thickness in children⁵

Objective

- ❖ To investigate the associations among socioeconomic factors, sleep duration, and brain morphometry in children

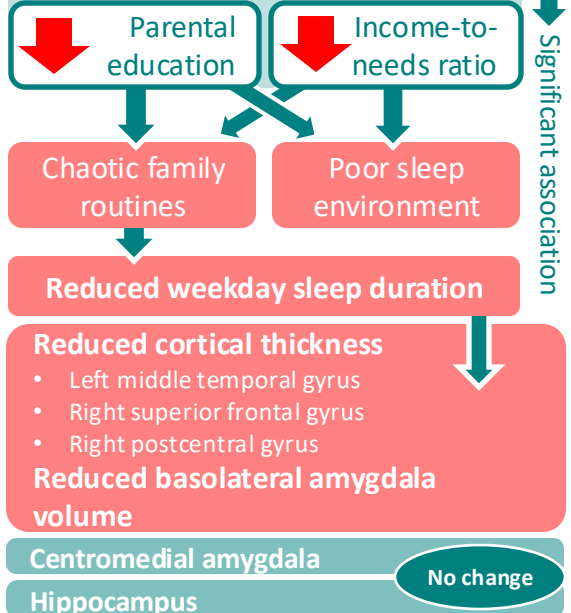
Methods

- 94 children (5-9 y), socioeconomically diverse families
- Socioeconomic status graded by family income-to-needs ratio and parental education
- Parents reported on children's **sleep durations, sleep environment, family routines**
- MRI-based **morphometric analysis** of children's cerebral anatomy (**cortex, amygdala and hippocampus**)

Results

- ✓ SED significantly associated with:
 - poorer sleep environments and family routines
 - **shorter weekday sleep duration** in children
- ✓ SED effect mediated by **family routines** only
- ✓ Shorter weekday sleep duration significantly associated with **reduced subregional cortical thickness** and **basolateral amygdala volume**

Socioeconomic determinants



Our thoughts:

- Children who slept less through the week exhibited reduced cortical thickness in the regions associated with **language processing, inhibitory control, and somatosensory processing**; involvement of the amygdala may also be associated with impaired **emotional processing**.
- This may contribute to explain the poorer behavioural and educational outcomes observed in sleep-deprived children and suggest new avenues for intervention.
- The results are consistent with the theory that sleep disruption associated with SED causes morphological changes in children's brains.
- However, the study design **did not allow confirmation** of the **causal directionality** between sleep duration and brain morphology, and the use of self-reported measures may have introduced systematic **bias**. Further investigations should focus on objective measurements and longitudinal data analysis.

1. Gitterman BA, et al. (2016) Poverty and Child Health in the United States. *Pediatrics* 137 (4): e20160339. 2. Medic G, et al (2017). Short- and long-term health consequences of sleep disruption. *Nat Sci Sleep* 19 (9): 151-161. 3. Paruthi S, et al (2016). Recommended amount of sleep for pediatric populations: A consensus statement of the American Academy of Sleep Medicine. *J Clin Sleep Med* 12 (06): 785-786 4. Simon EB et al (2020). Sleep loss and the socio-emotional brain. *Trends in Cognitive Sciences*, 24(6), 435-450 5. Hansen M, et al (2023). Socioeconomic disparities in sleep duration are associated with cortical thickness in children. *Brain Behav* 13(2):e2859.