



Disruptive Behavior in preschoolers: Connections to Sleep and the Medial Temporal Lobe

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Background

Preschool-age is a window of wide variability in behavioral and emotional regulation. Early challenges predict later outcomes (1) and poor regulation can negatively impact parental wellbeing (2).

That same developmental window is marked by substantial changes in both sleep and brain development (3-4).

Slow wave activity (SWA) may be a marker for cortical maturation (5): it predicts later whole-brain myelination (6) and shifts in SWA topography predict specific skill development (7).

SWA is also associated with emotional well-being in development (8-9). SWA is associated with regulation of emotional attention in young children (10) and disruption of SWA is related to youth psychopathology (11).

We expect that SWA may relate to maturation of the prefrontal circuitry that supports inhibitory and emotional control, which is disturbed in disruptive behavior (12). These behaviors are also influenced by underlying neural development, particularly development of the amygdala (13).

How sleep and the brain collectively contribute to disruptive behavior is unknown. **The amygdala is sensitive to disrupted sleep (14), so the region is a plausible link between the brain and sleep for individual differences in disruptive behavior.**

Methods

Participants were habitually napping children ($n=133$; $M_{\text{age}} = 3.76$ years; $SD_{\text{age}} = 0.55$; 50.5% male) from a broader longitudinal study.

Parental reports of disruptive behavior and parental stress were collected via the Multidimensional Assessment Profile of Disruptive Behavior (MAP-DB) and Parent Stress Index (PSI) parental distress subscale.

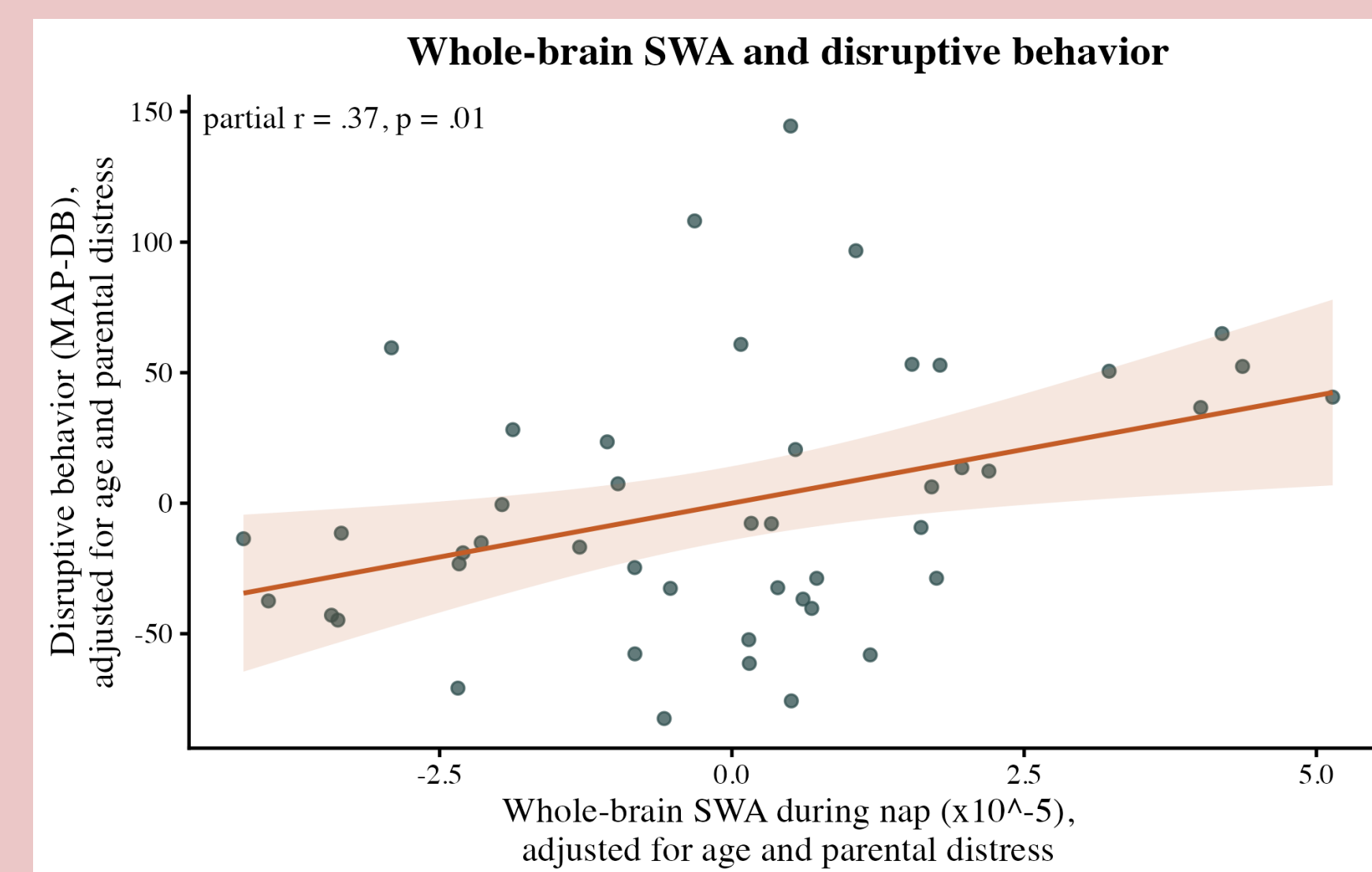
Polysomnography (EEG, EMG, EOG) during a nap in a subset of children ($n=89$) was used to assess SWA.

Average 24-hr sleep duration was obtained via 3-14 day actigraphy (GeneActiv; $n=64$).

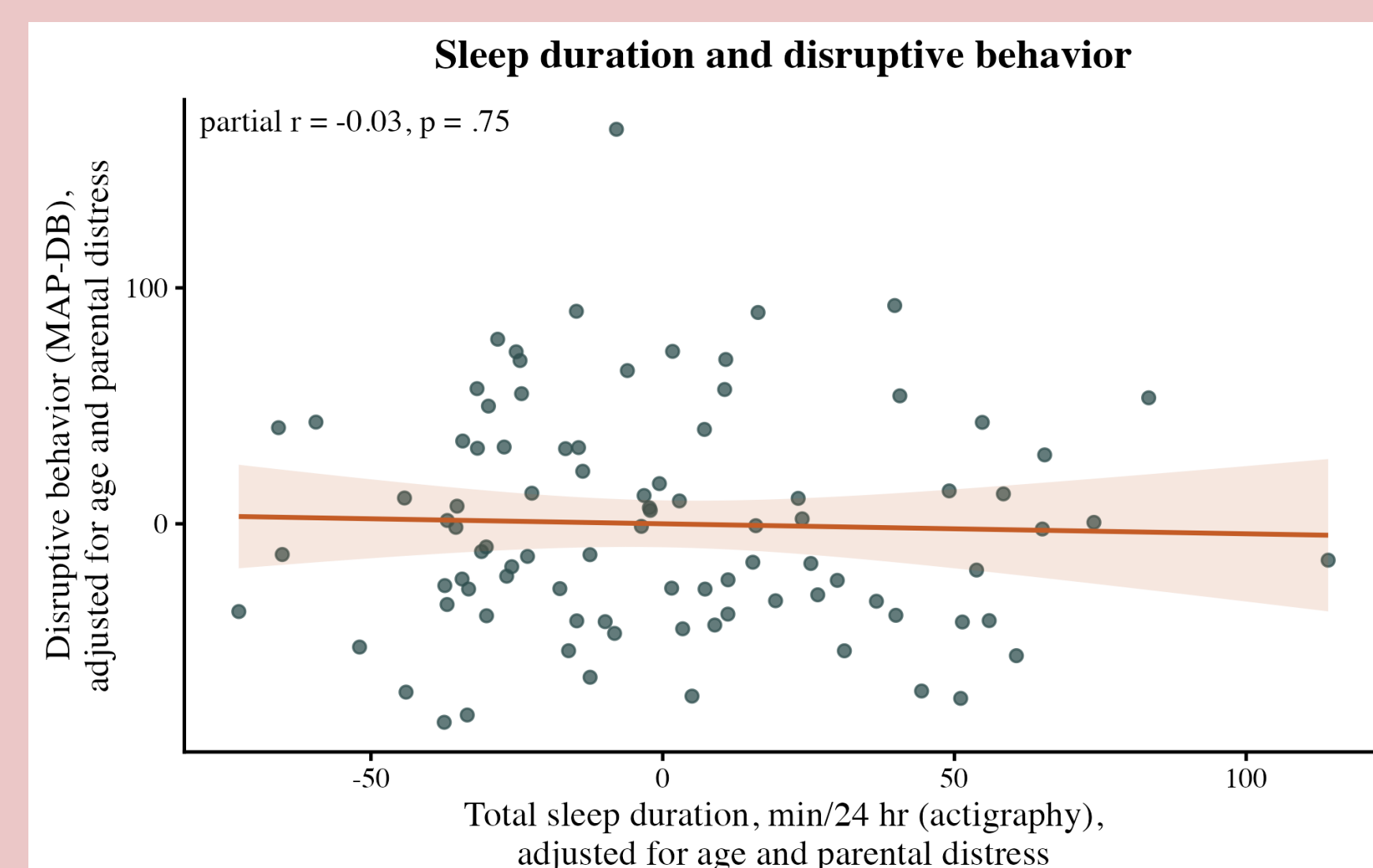
T1-weighted MRI scans were also collected from a subset of participants ($n=69$) and segmented via FreeSurfer; volumes were residualized considering total intracranial volume.

We tested individual relationships with disruptive behavior before inclusion in comprehensive models.

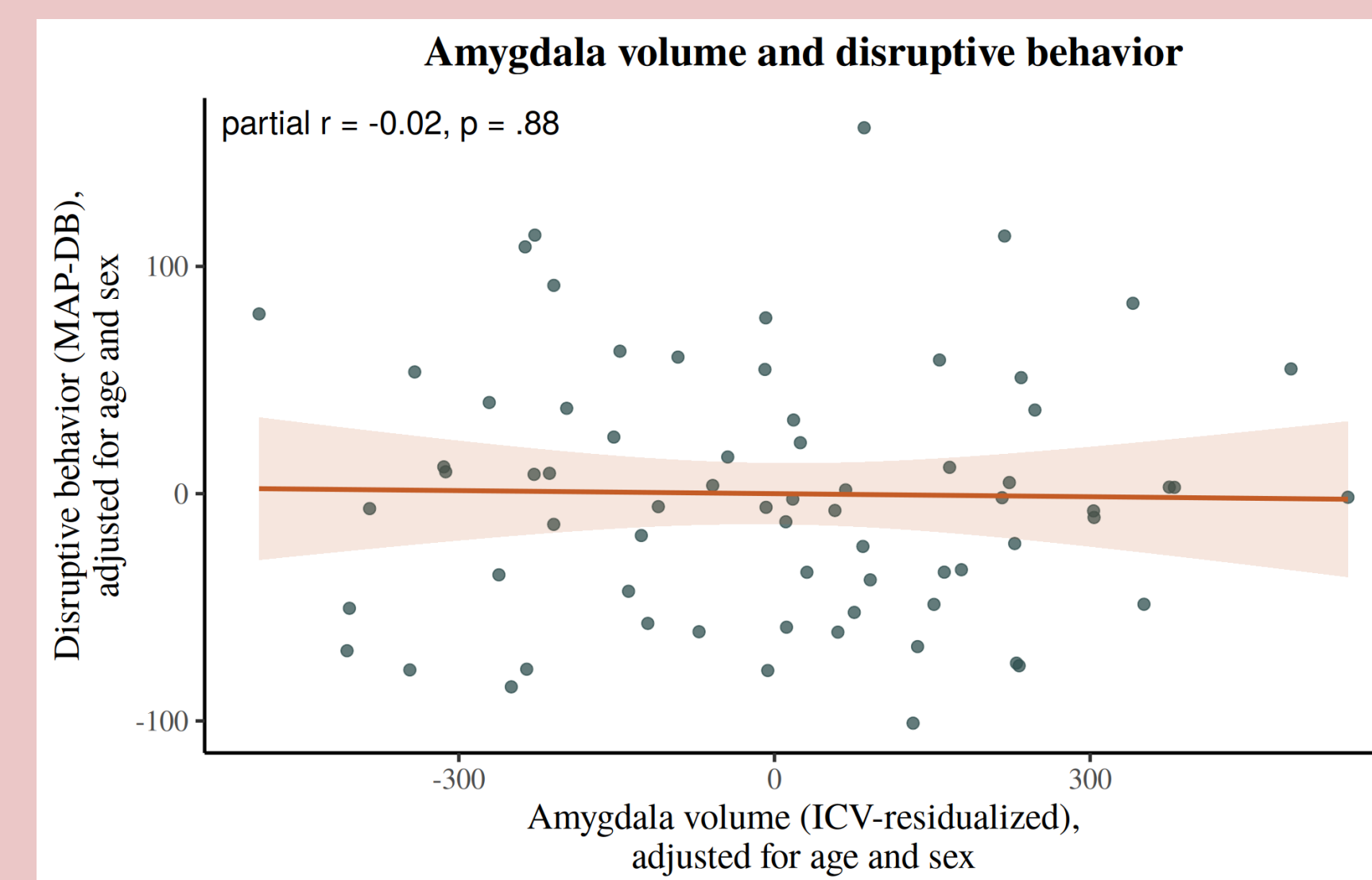
Results



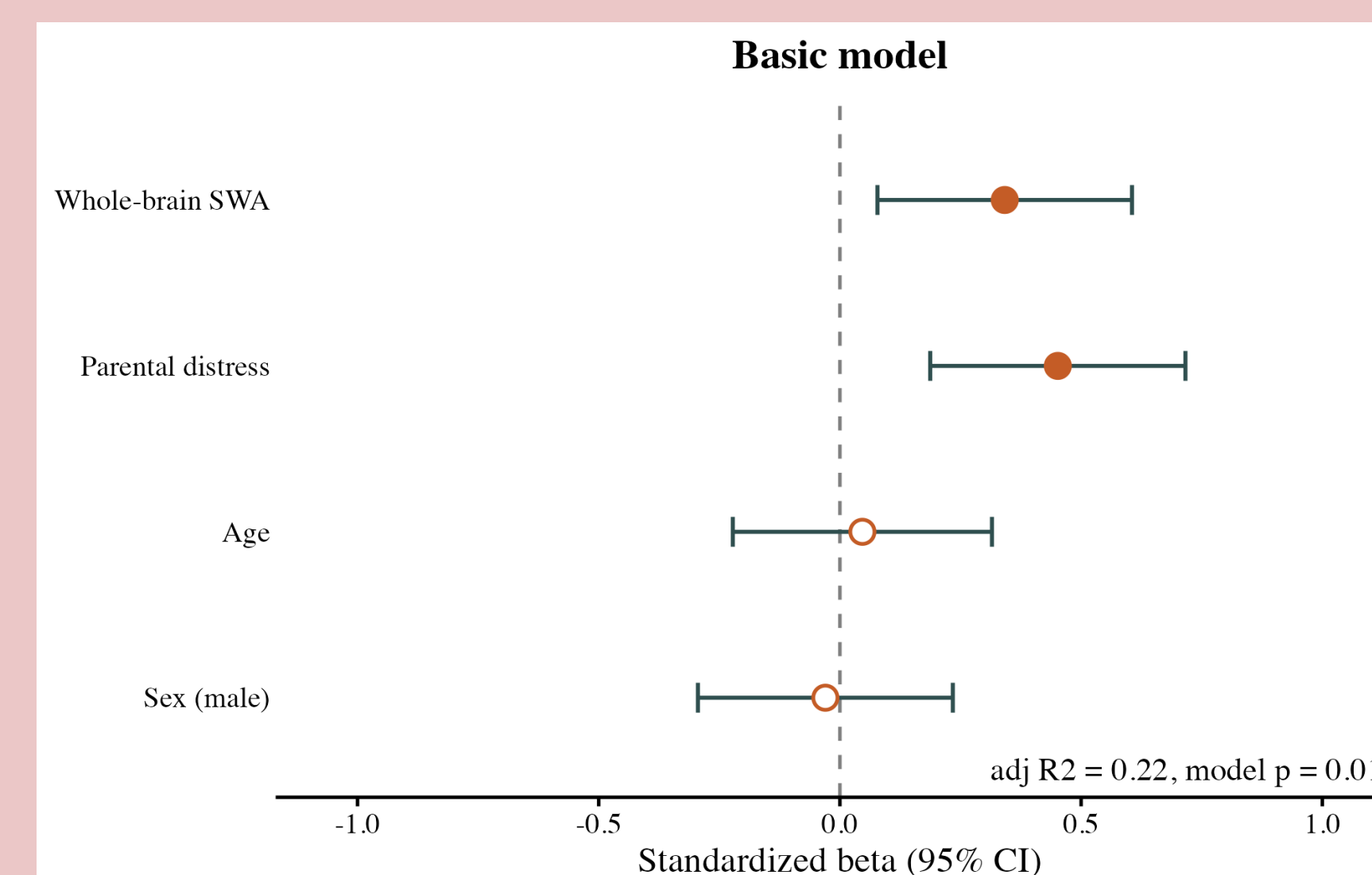
MAP-DB was significantly correlated with SWA, when controlling for age and parental distress, $\text{partial } r = .37$, $p = .01$. That was no longer true when also controlling for sleep duration, however, $\text{partial } r = .28$, $p = .13$.



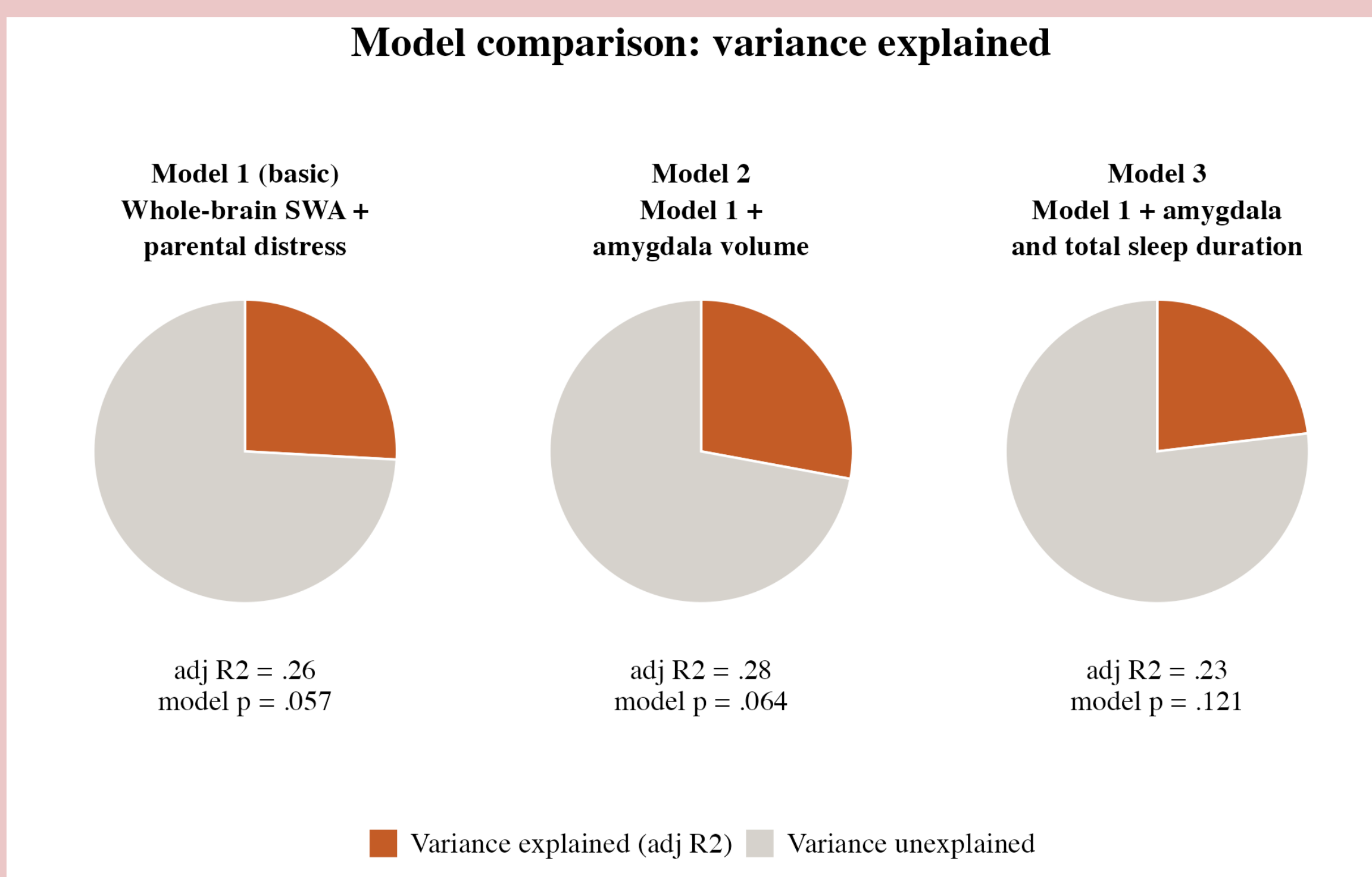
Habitual sleep duration was not directly related to disruptive behavior, $\text{partial } r = -.03$, $p = .75$, controlling for age and parental distress.



The amygdala was not directly related to MAP-DB, $\text{partial } r = .02$, $p = .90$, controlling for age and parental distress.



In a basic model ($n=89$), whole-brain SWA, $\beta = .34$, $p = .02$, and parental distress, $\beta = .45$, $p = .002$, each significantly predicted disruptive behavior; age and sex did not, $\text{adj. } R^2 = .22$, $\text{model } p = .007$.



We compared nested models on adjusted R².

A basic model consisting of just SWA, parental distress, age, and sex significantly fit the data (Basic Model).

That was no longer the case with the subset sample that includes MRI ($n=59$; Model 1). Other models additionally included amygdala volume (Model 2), and amygdala volume plus total habitual sleep duration (Model 3). None of those three models reached significance, but Models 1 and 2 were approaching significance with the smaller sample.



Conclusion

SWA was significantly related to disruptive behavior. However, amygdala volume was not.

In more comprehensive models (such as Model 2), amygdala volume combined with SWA did not significantly predict disruptive behavior, likely due to reduced power in this smaller subsample. That relationship weakened further once sleep duration was added. Perhaps because habitual sleep duration is negatively influenced by dysregulation and disruptive behavior while it is positively influenced by greater neural maturation which could feasibly be the cause of higher SWA.

The distinct contributions of SWA (relative to regional volumes) on disruptive behavior may reflect sleep's more proximal role in behavior while neural measures have a more distal impact, diluted by individual differences.

Future work should consider a larger complete sample and longitudinal changes in sleep, brain development, and disruptive behavior to track changes in maturity. Measures of cognitive performance would be valuable to assess the impact of disruptive behavior in preschoolers.

References

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