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Behavioral and neural concordance in parent-child dyadic sleep patterns

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ABSTRACT

Sleep habits developed in adolescence shape long-term trajectories of psychological, educational, and physiological well-being. Adolescents' sleep behaviors are shaped by their parents' sleep at both the behavioral and biological levels. In the current study, we sought to examine how neural concordance in resting-state functional connectivity between parent-child dyads is associated with dyadic concordance in sleep duration and adolescents' sleep quality. To this end, we scanned both parents and their child (N = 28 parent-child dyads; parent M_{age} = 42.8 years; adolescent M_{age} = 14.9 years; 14.3% father; 46.4% female adolescent) as they each underwent a resting-state scan. Using daily diaries, we also assessed dyadic concordance in sleep duration across two weeks. Our results show that greater daily concordance in sleep behavior is associated with greater neural concordance in default-mode network connectivity between parents and children. Moreover, greater neural and behavioral concordances in sleep is associated with more optimal sleep quality in adolescents. The current findings expand our understanding of dyadic concordance by providing a neurobiological mechanism by which parents and children share daily sleep behaviors.

1. Introduction

Adequate sleep is vital for developing youth. Inadequate sleep (e.g., high variability of day-to-day sleep duration, sleep deprivation) can result in long-term cognitive, behavioral and functional deficits in developing youth (Beebe, 2011). For example, less sleep time and lower sleep quality are related to maladjustment, including unstable mood, academic difficulties, poor cognitive and emotional control, and externalizing and internalizing symptoms (Beebe, 2011; Carskadon and Dement, 1987; Dahl et al., 1994; Dahl and Lewin, 2002; Roane and Taylor, 2008; Wolfson and Carskadon, 1998; Wong et al., 2010). Moreover, such poor sleep behavior can result in neural impairments in the developing brain associated with destructive alterations in neural systems (Jan et al., 2010), including reduced cerebral metabolism, neurogenesis due to increased circulation of the adrenal stress hormone, and gray and white matter alterations (Halbower et al., 2006; Horne, 1993; Kopp et al., 2006; Mirescu et al., 2006; Telzer et al., 2015).

Inadequate sleep is particularly endemic in adolescence, perhaps because adolescents do not receive the quality environmental context needed for restorative sleep and optimal brain development (Dahl and Lewin, 2002). The transition to middle and high school presents challenges to adolescents' ability to obtain adequate rest at night. For instance, early school start times conflict with their biologically driven circadian phase delay that leads adolescents to prefer later bedtimes (Carskadon et al., 1993; Dahl and Lewin, 2002). Academic demands require adolescents to stay up late completing school work, which disrupts their sleep time (Adam et al., 2007). Finally, increased access to technology (e.g., televisions, computers, internet, smart phones) creates evening distractions that result in later bedtime (Adam et al., 2007; Tazawa and Okada, 2001; Van den Bulck, 2004), while also altering the body's biological sleep patterns (e.g., disrupting melatonin release and circadian rhythm; Chang et al., 2015). Thus, understanding the environment by which sleep patterns are socialized to youth may increase our understanding of sleep behavior and brain development during this critical phase of development.

As children enter adolescence, parental influence on sleep declines, and adolescents tend to take more control over their own schedule, resulting in more inadequate and irregular sleep patterns. Although adolescence tends to be a developmental period when youth orient more towards their friends and seek more autonomy from their parents (Nelson et al., 2005), sleep largely occurs within the constraints of the family home such that the daily sleep routine of parents can significantly shape the daily sleep routine of adolescents (Fuligni et al., 2015). Recently, researchers have focused on social factors, particularly in the home environment, in shaping adolescents' sleep. For instance,

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the emotional climate, quality of family relationships, and parental monitoring of adolescents' sleep and wake times are key factors shaping adolescents' sleep behavior (Adam et al., 2007; El-Sheikh et al., 2006). An even more proximal contributor to adolescents' sleep is the sleep habits of parents themselves. For instance, correlational research has shown that irregular late bedtimes in parents is related to sleep problems and daytime sleepiness in children (Komada et al., 2009), parents who sleep less have children who sleep less (Li et al., 2010), and there tends to be strong similarities in parents' and children's sleep-wake cycles (Zhang et al., 2010). Moreover, using daily diary methods with independent daily ratings of sleep from adolescents and parents, adolescents' sleep behavior – when they go to bed, how much they sleep, and when they wake up – is closely synchronized with parents' sleep patterns (Fuligni et al., 2015). Importantly, this daily concordance in sleep patterns holds above and beyond factors such as study time, underscoring the unique and important role of parents' sleep for adolescents' sleep. In addition to research finding concordance in self-report measures, recent work has identified biological concordance in parent-child sleep patterns. For instance, using EEG measures, adolescents' sleep continuity and architecture were closely associated with parents' sleep continuity and architecture (Kalak et al., 2012). Together, this research underscores the important role of parents' sleep in shaping their children's sleep.

Parents and their children engage in high levels of dyadic coordination at the behavioral (e.g., shared affect) and biological level, such as the immediate coordination of ongoing physiological signals (e.g., heart rate; Feldman et al., 2011). Theoretical work suggests that harmonious interpersonal interactions including behavioral and psychological similarity are derived from more in-tuned neural states between two individuals (Wheatley et al., 2012), offering a likely neurobiological candidate underlying synchronized behavior in parent-child dyads, namely neural concordance. Indeed, we recently demonstrated that emotional concordance between parents and their adolescent children was associated with more in-tuned neural states within parent-child dyads, and greater neural concordance was associated with better adjustment (Lee et al., 2017). Therefore, neural concordance may represent a neurobiological marker by which parents and children share daily sleep behaviors.

In the current study, we aimed to examine neural level concordance associated with dyadic sleep concordance and how these two levels of dyadic concordance are associated with adolescents' sleep quality. To this end, we implemented a multimethod analytical approach combining resting state functional neuroimaging, in which both parents and their child underwent an fMRI scan to measure neural concordance and a daily diary method to estimate how sleep time of parent-child dyads fluctuate together across two weeks (i.e., behavioral concordance). We focused on total sleep time as an index of concordant sleep behavior because sleep time is more globally attributable to sleep pattern concordance (Fuligni et al., 2015). The benefit of this daily diary approach is two-fold. First, we had independent ratings from both the parent and adolescent child, moving beyond single-reporter studies. Secondly, by asking parents and adolescents to independently report on their sleep time every day for 14 days, we better captured daily sleep patterns than single time-point, self-report measures (Bolger et al., 2003). By asking parents and adolescents each day to report on their sleep time allowed us to estimate the concordance between parent and adolescent sleep at the daily level within dyads, reducing confounds that are present in a between-family approach (Fuligni et al., 2015).

To index neural level concordance, we quantified how functional connectivity patterns are similar in parent-child dyads. In particular, we focused on the default mode network (DMN), which represents an intrinsic regulation system that plays a pivotal function for segregating internally and externally directed cognitive processes (Buckner et al., 2008; Fox et al., 2005). Previous evidence indicates that the DMN is one of the main functional networks in the brain that regulates wakefulness including initializing, maintaining, and terminating sleep (Basner et al., 2013; Horovitz et al., 2009; McKenna and Eyler, 2012; Picchioni et al., 2013), by changing functional coupling with other independent functional networks (i.e., connectivity pattern of DMN to the other nodes; Larson-Prior et al., 2011; Laufs et al., 2003; Picchioni et al., 2013). Moreover, sleep deprivation is associated with reduced functional connectivity within the DMN as well as between the DMN and other networks (De Havas et al., 2012; Sämann et al., 2010), indicating that sleep is not based solely on the DMN *per se* but rather on how the DMN is wired to other networks. Such impaired downregulation of the DMN after sleep deprivation may result in the insufficient allocation of cognitive resources (Drummond et al., 2005), translating to behavioral changes following poor sleep (Sämann et al., 2010).

Hence, the current study examined how parent-child dyadic sleep concordance is related to functional dynamics of the DMN with other networks. To accomplish this, we identified all possible intrinsic networks in the brain including the DMN using independent component analysis (ICA), estimated functional connections between the DMN and all other intrinsic networks, and quantified how similarly the DMN is wired to other brain systems (DMN connectivity similarity) between parents and their child. We used ICA to identify DMN connectivity as opposed to seed-based connectivity patterns, because ICA can estimate each functional network independently based on the linear mixing assumption for temporal coherency (Beckmann and Smith, 2004), and thus provides metrics of both between- and within-network connectivity (Jafri et al., 2008; Joel et al., 2011), whereas seed-based methods only provide one single metric based on the user-specified temporal signal (i.e., seed).

We examined three key questions. First, is daily concordance in sleep time between parents and their adolescent child associated with neural concordance in DMN connectivity? Although prior research has investigated dyadic concordance in sleep behavior between parents and their child focusing on subjective (i.e., self-report daily report; Fuligni et al., 2015) and biological (e.g., EEG coherence; Kalak et al., 2012) aspects of sleep, no prior study has examined neurobiological concordance in relation to sleep behavior in parent-child dyads. Second, are daily concordance in sleep and neural concordance in DMN connectivity associated with adolescents' overall sleep quality? We have previously shown that adolescents who show greater affective synchrony and greater neural concordance with their parents report better emotional adjustment (Lee et al., 2017), suggesting that parent-child neural concordance may confer benefits for youth. Finally, we tested whether neural concordance in DMN connectivity is associated with sleep quality above and beyond the effects of daily concordance in sleep. Such effects would suggest a unique and important role of neural concordance that cannot be measured or explained by self-report measures of sleep.

2. Materials and method

2.1. Participants

28 parent-child dyads participated in the current study (n = 56; parent M_{age} = 42.79 years, range = 33–57, 14.29% father; child M_{age} = 14.93 years, range = 13–17, 46.43% female). Although no study has examined RSN connectivity concordance between dyads, we based our sample size on previous developmental resting-state fMRI studies (Gabard-Durnam et al., 2016; Gee et al., 2013). All parent-child dyads were biologically related and provided informed consent/assent, and no participants reported any mental health problems (e.g., current clinical diagnosis or pharmacological intervention for a mental illness).

2.2. Daily concordance in sleep-time

Adolescents and their parents each completed daily checklists indicating the amount of time they slept each night for two weeks (for a total of 14 checklists). Both parents and their adolescent child either

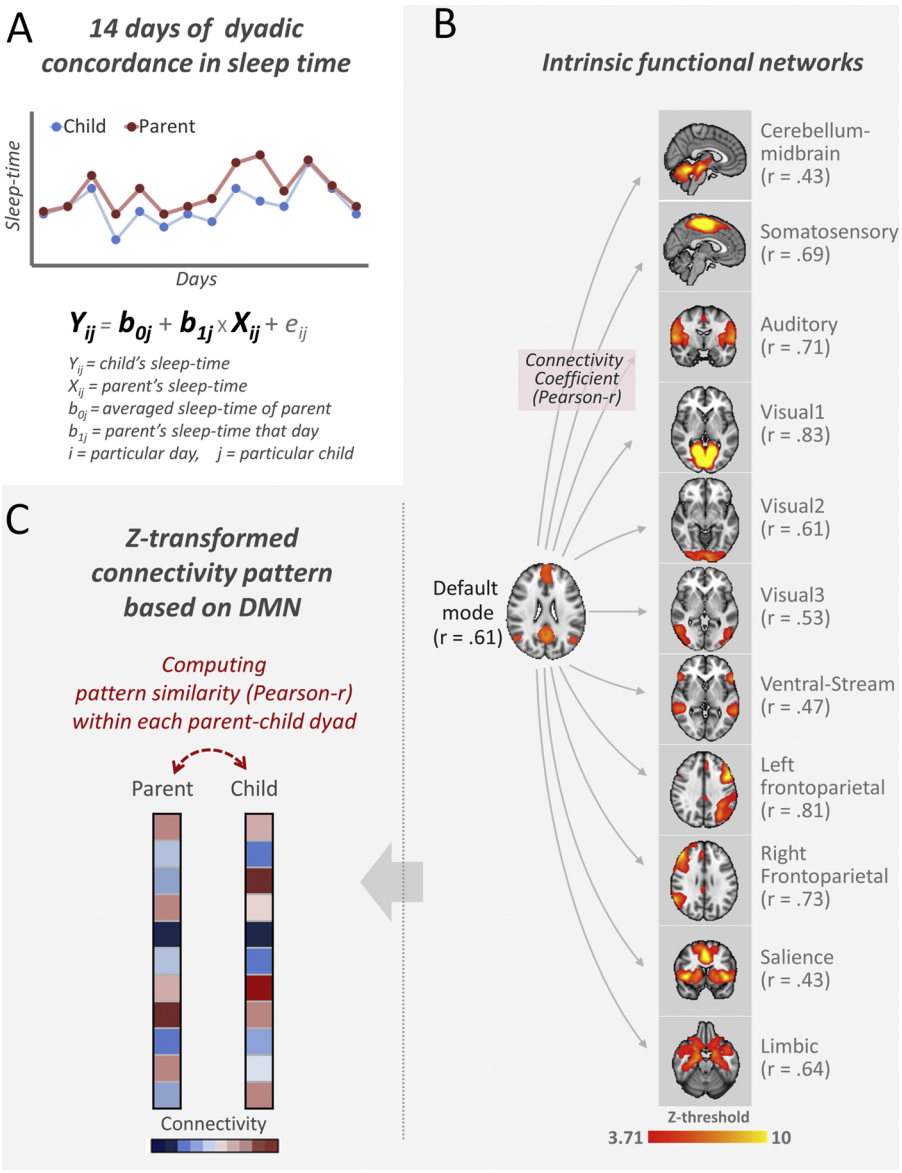

Fig. 1. (A) Hierarchical linear model to calculate dyadic concordance in sleep duration. Child's sleep time was predicted by parent's sleep-time on that day. (B) Intrinsic functional networks used in the current study and schematic representation of default-mode-network connectivity estimation. Each "*r*" indicates spatially cross-correlation coefficient value with canonical resting-state network templates acquired from a previous meta-analysis. In the current study, we used RSN maps above 0.4 as cross-correlation threshold. (C) The procedure of default-mode-network concordance calculation. *Fisher r-to-z*-transformed and vectorized connectivity patterns were correlated for each parent-child dyad.

completed the checklists by accessing a secure website or by using pencil and paper. For those completing with paper/pencil, we monitored completion of the checklists by providing participants with fourteen manila envelopes and an electronic time stamper. The time stamper is a small, hand-held device that imprints the current date and time and is programmed with a security code so that the correct date and time cannot be altered. Participants were instructed to place their completed checklists into a sealed envelope each night and to stamp the seal of the envelope with the time stamper. For those completing the surveys on the secure website, an email with the link to each daily survey was sent separately to the parent and child, and the time and date of completion were recorded via the website. Three adolescent children did not complete the daily checklists.

From these 14 daily reports, we calculated the daily concordance between adolescents' and parents' amount of time slept. The concordance in sleep duration for each dyad was estimated by predicting children's daily sleep time from parents' daily sleep time (Fig. 1A). Given the nested nature of the data, we used Hierarchical Linear Modeling (HLM) which was designed to analyze nested data of the type that were collected for this study (i.e., daily level data nested within individuals) using HLM 7.01 software (Raudenbush et al., 2013) as follows:

child's daily sleep time$_{ij}$ = b_{0j} + b_{1j}(parent's sleep time) + e_{ij}.

Sleep time on a particular day (*i*) for a particular child (*j*) was modeled as a function of the average amount of sleep time of the parent across days (b_{0j}) and the parent's amount of time slept that day (b_{1j}). The empirical Bayes estimate for each dyad over the 14 days was extracted from the statistical model. The empirical Bayes estimate is an optimally weighted average that combines the dyad's average slope and "shrinks" it towards the mean slope of the group (Diez, 2002), such that higher values indicate higher concordance of child's sleep-time based on parent's sleep-time. On average, we found that parent's sleep time does not predict children's sleep time (unstandardized coefficient = 0.072, SE = 0.071, p = 0.32). Instead, there was significant variability in the empirical Bayes estimate, ranging from −0.28 to 0.38, indicating that some families are desynchronous and others are highly synchronized. One of our primary goals was to test whether neural concordance is associated with this daily sleep concordance.

2.3. Adolescent's sleep quality

Adolescents completed the Pittsburgh Sleep Quality Index (PSQI; Buysse et al., 1989), a standard measure widely used to assess sleep quality for various ages and clinical populations. The 19 questions

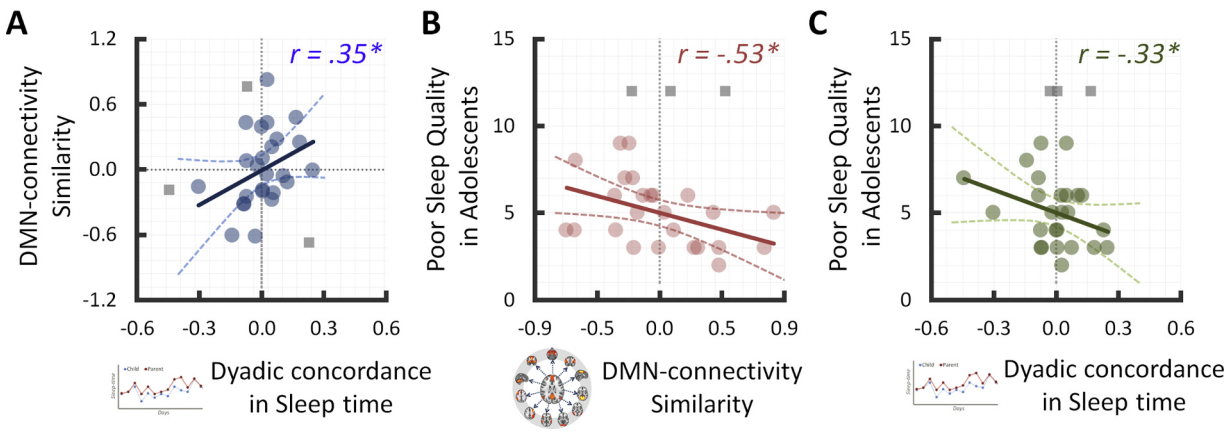

Fig. 2. The relationship between (A) Dyadic sleep concordance and neural concordance of default-mode network (DMN) connectivity, (B) Dyadic neural concordance and child's sleep quality and (C) Dyadic sleep-time concordance and Child's sleep quality in parent-child dyads. Note that lower scores indicate better sleep quality. The Colored-dash line indicates 95% confidence interval of regression lines. The gray dash lines indicate zero points at each axis. *p < 0.05 at a 95% CI after bootstrapping resampling (n = 50,000). The gray-colored squares were outliers identified and de-weighted by robust-method (Pernet et al., 2012).

generate seven clinically derived component scores: daytime dysfunction, sleep duration, sleep disturbances, sleep latency, sleep efficiency, use of sleep medications, and sleep quality. The 7 component scores are summed to obtain a global score ranging from 0 to 21, with higher scores indicating poorer sleep quality and scores less than 16 indicating good sleep quality as recommended by the scale developers. Average sleep quality in the current study was 5.79 (SD = 2.86), ranging from 2 to 12 (α = 0.74). All participants were below the cutoff of 16 for clinically impaired sleep.

2.4. Resting-state fMRI scan

2.4.1. Acquisition and preprocessing

Both parents and children completed a 6-min resting state brain scan on the same day, during which they were instructed to view a black screen with a white fixation cross. High-resolution structural images (T1-MPRAGE) were acquired first (repetition time or TR = 1.9 s, echo time or TE = 2.3 ms, matrix size = 256 × 256, field of view or FOV = 230 mm, flip angle or FA = 90°, 1 mm isotropic voxel). The resting-state data were acquired from a gradient-echo echo-planar image sequence. The resting-state scan parameters for 15 dyads were 180 volumes, 38 slices with no inter-slice gap, TR = 2 s, matrix = 92 × 92, FOV = 230 mm, FA = 90°, voxel size 2.5 × 2.5 × 3.3 mm^3, and 6 min duration, and the other 16 dyads were 120 volumes; 36 slices with no inter-slice gap, TR = 3 s, matrix = 64 × 64, FOV = 220 mm, FA = 90°, voxel size 3.5 × 3.5 × 4.0 mm^3 and 6 min duration. Data preprocessing were performed using FMRIB's Software Library (FSL; www.fmrib.ox.ac.uk/fsl), including skull stripping of structural images with BET, motion correction with MCFLIRT, smoothing with full-width half-maximum 6 mm, masking of non-brain voxels; 128 s high-pass, voxel-wise demeaning and normalization into 2 mm-MNI-standard via individual T1-weighted anatomical image with FLIRT. Noise signals were identified individually and removed using MELODIC ICA and an automated signal classification toolbox (Tohka et al., 2008; an average of 4.7 components (17.0%) were removed from each participant; mean framewise displacement, FD = 0.03 mm, range = 0.01–0.1 mm; no significant differences in motion between parents and adolescents, t(54) = 0.83, p = 0.41). Although there are several strategies suggested to rigorously correct for motion-related noise in resting state data, such as spike regression with 24-type of motion parameters (Lemieux et al., 2007; Satterthwaite et al., 2013) and individual high-motion contaminated volume scrubbing (Power et al., 2012), several drawbacks of these strategies also exist such that they can introduce overfitting by the use of a large set of nuisance regressors, loss of frequency information, and negative influences on the autocorrelation structure of data as well as a reproducibility issue (please see Pruim et al., 2015a,b). Therefore, we applied ICA denoising approach for the current analysis given the recent evidence that ICA denoising can effectively enhance the fidelity of data quality regarding controlling noise signal (Pruim et al., 2015b; Starck et al., 2013). It has been well demonstrated that RSNs estimated by ICA are less prone to artifactual effects from noise such as physiological signal, global signal fluctuation and motion due to the ability of ICA to account for the existence of such noise effects within additional non-RSN components (Boubela et al., 2013; Cole et al., 2010) with robustness of RSN identification in rs-fMRI (Poldrack et al., 2011). Furthermore, a recent study demonstrates that ICA-based denoising can diminish differences due to multi-center and protocol collection in rs-fMRI (Feis et al., 2015; Paolini et al., 2015).

2.4.2. Analysis for dyadic neural concordance of default mode network connectivity

Given our aim to evaluate how the DMN is wired to other independent functional networks, we applied an ICA approach again for group level RSN estimation in addition to motion-denoising, since the ICA approach can provide both metrics for within- and between-network connectivity without *a priori* hypotheses for a specific seed-region (Jafri et al., 2008; Joel et al., 2011).

To calculate between-network connectivity maps, we first estimated each individual-specific time-series of each resting state network (RSN) using the 12 group-level RSN maps previously identified from the group-level ICA analysis in which the current dataset was included (Lee and Telzer, 2016) at cross-validation threshold of 0.4 (i.e., template-matching procedure with the previous resting-state studies; Laird et al., 2011; Shirer et al., 2012; Smith et al., 2009) (Fig. 1B). Using these group-level RSN maps, we performed a linear model estimation against the separate individual data sets yielding subject-specific time-series for each RSN (i.e., temporal regression) (Filippini et al., 2009), yielding individual specific time-courses for each RSN. The estimated individual's time-courses for each RSN were then correlated between the DMN to those of all the other networks (between-RSN networks connectivity matrix around the DMN; Fig. 2B). After Fisher-Z transformation and vectorization of connectivity coefficients, connectivity pattern similarity as a form of multi-voxel pattern similarity analysis was calculated for each parent-child dyad by correlating the pattern vectors (Fig. 1C).

Note that when we reconstructed each RSN component using the current approach, each spatial map and time-course of a given RSN map

were estimated while regressing out the effect of other RSNs and unspecified signals including noise components and unspecified components. Therefore, despite the possibility that residual noise signals might exist even after the individual level ICA, given that in the multivariate regression approach, the global nature of residual motion signals that are represented/shared across multiple components can be controlled out within the analysis to a certain extent. This multiple linear regression approach (ICA/temporal regression) has a benefit to estimate and identify RSN specific signals without consideration about the presence of other signal effects which otherwise might negatively impact the valence of functional connectivity (Beckmann et al., 2009).

2.5. Analysis plan

Given the primary goal of the current study, we used correlational approach to elucidate the relationships between sleep concordance at the behavioral- and neural level and adolescents' sleep quality. In these analyses, we employed a robust method using Robust Correlation Toolbox (Pernet et al., 2012) to reduce possible impact of non-normal, skewed distribution of our sample, and statistical significance was tested by bootstrapping resampling (n = 50,000) at a 95% confidence interval ($p < 0.05$). In the analysis, adolescents' age and gender were controlled as possible confounds based on prior work indicating that age and sex can be influential for developmental trajectories of the brain (e.g., Herting et al., 2012; Tamnes et al., 2010). Finally, to test the independent associations of behavioral and neural concordance, we conducted multiple regression analyses in SPSS version 24 (IBM_Corp, 2013).

3. Results

3.1. The relationship between dyadic concordance in sleep behavior concordance and neural level concordance

To link dyadic concordance at both the behavioral and neural level (dyadic concordance in sleep behavior and dyadic neural concordance in DMN connectivity, respectively), we first correlated the association between sleep synchrony and neural concordance in parent-child dyads. We found a significant positive relationship, robust *Pearson* $r = 0.35$, $p < 0.05$, 95% CI = [0.07, 0.61] indicating that greater synchronized sleep-time is associated with more neural concordance in DMN connectivity in parent-child dyads (Fig. 2A).

3.2. The relationship between behavioral and neural concordance and adolescents' sleep quality

Next, we tested the real-life implications of behavioral and neural concordance in parent-child dyads by examining how sleep concordance and neural concordance are associated with adolescents' sleep quality. We found that neural concordance was significantly associated with better sleep quality, robust *Pearson* $r = -0.53$, $p < 0.05$, 95% CI = [−0.78, −0.25] (Fig. 2B). In the same way, behavioral sleep concordance was associated with better sleep quality, robust *Pearson* $r = -0.33$, $p < 0.05$ 95% CI = [−0.58, −0.02] (Fig. 2C).

Finally, we conducted a multiple regression analysis in which we examined whether neural concordance predicts adolescents' sleep quality above and beyond behavioral sleep concordance. Whereas neural concordance in DMN connectivity predicted better sleep quality (B = −2.66, SE = 0.895, $p < 0.05$, 95% CI = [−5.17, −1.14]), behavioral sleep concordance did not predict sleep quality (B = −3.83, SE = 2.22, $p > 0.05$, 95% CI = [−8.52, 0.661]).

4. Discussion

Growing public concern and understanding of how we can optimize sleep habits of adolescents is critical to improving their psychological, educational, and physiological well-being (Beebe, 2011; Carskadon and Dement, 1987; Dahl and Lewin, 2002; Roane and Taylor, 2008; Wong et al., 2010). The current findings provide insight into how adolescents' sleep quality may be shaped by their parents in terms of behavioral and neural level sleep concordance. We took a unique, multimethod approach and showed that behavioral level sleep concordance in parent-child dyads measured across 14 days is significantly associated with neural concordance in the DMN's between-network connectivity. Moreover, neural concordance in the DMN between parent-child dyads is associated with more optimal sleep quality in adolescents, above and beyond the effects of daily sleep concordance. Our findings expand previous work (Fuligni et al., 2015) by providing the first examination of dyadic neural correlates of sleep concordance with links to sleep quality in adolescents.

Sleep tends to largely occur within the constraints of the family home, and many studies have identified strong links between parents' and adolescents' sleep patterns, using self-report (Komada et al., 2009; Li et al., 2010; Zhang et al., 2010), daily concordance from multiple reporters (Fuligni et al., 2015), and biological similarity (i.e., EEG; Kalak et al., 2012). Yet, no prior study has examined dyadic similarity at the neural level as neurobiological markers that may explain such dyadic concordance in sleep patterns. The brain's functional architecture changes and is tuned gradually by individual's accumulating experiences (e.g., Gabard-Durnam et al., 2016). Previous studies have demonstrated that even a short-term behavioral manipulation, such as cognitive tasks, can change functional connectivity dynamics between intrinsic brain networks (e.g., Cohen et al., 2014), suggesting that experience tunes the functional architecture of the brain. Therefore, adolescents' DMN connectivity is likely shaped, in part, by accumulated experiences within the family context. Synchronizing sleep behavior between parents and their child may be one way the DMN is reconfigured and becomes neurally in-tuned. In other words, parent-child behavioral dynamics (i.e., similar sleep behaviors) may promote adolescents' well-being by reconfiguring adolescents' functional system to be more similar to their parents' functional system. Alternatively, it is also possible that neural concordance promotes more synchronized sleep patterns between parents and their child such that the general status of the brain's intrinsic networks can serve as a biomarker for predicting individual's behavior (e.g., Rosenberg et al., 2015). Because our findings are based on the correlational approach and are not longitudinal, we cannot determine whether the functional connectivity patterns preceded and contributed to sleep concordance or vice versa.

Importantly, we observed that there is variability of sleep concordance across dyads. This variability indicates that not all adolescents are necessarily tuned with their parents. That is, sleep and neural connectivity patterns developed in adolescents are not based solely on the sleep and neural connectivity patterns of their parents. Adolescence is a period of social attention shifting beyond family (Nelson et al., 2005) with decreased closeness to their parents (Tsai et al., 2013). Moreover, parental influence on their children's sleep declines during adolescence, and adolescents tend to take more control over their own sleep schedule. Thus, understanding such variability will be important for future research. Given that concordance in sleep behavior and DMN connectivity is associated with more optimal sleep quality, interventions aimed at promoting better sleep should move beyond variables centered on the individual adolescent (e.g., reducing technology use) to a more family-focused approach. By identifying which adolescents are more in-tuned with their parents will be one direction for understanding which youth may benefit most from such a family-focused approach. Indeed, prior work has found that families reporting more closeness tend to show greater similarity in their sleep patterns (e.g., Kalak et al., 2012). Whatever the possible source may be, the current variability indicates that dyadic concordance is a mutual process between parents and children that may vary depending on the context of the family (e.g., high closeness). Future research should unpack what other factors, such as quality of adolescent-parent interactions, are

involved in the reconfiguration of neural and behavioral dynamics and how dyadic concordance can change across development by adopting a longitudinal design.

In the current study, we focused on how the DMN is wired to other intrinsic networks. This is because previous studies have indicated that the DMN is significantly involved in wakefulness regulation (Basner et al., 2013; Horovitz et al., 2009; McKenna and Eyler, 2012; Picchioni et al., 2013) by changing its connectivity with other networks to initialize and terminate sleep (Larson-Prior et al., 2011; Laufs et al., 2003; Picchioni et al., 2013; Sämann et al., 2010). However, in the current study, we did not collect resting state while our participants were asleep. Therefore wiring patterns of the DMN we observed are more likely to be a reflection of intrinsic regulatory processes playing a role in internally and externally directed cognitive processes (Buckner et al., 2008; Fox et al., 2005) rather than capturing ongoing functional dynamics in active sleep regulation. Future studies should examine similarity in functional dynamic changes in the DMN as well as other intrinsic functional networks in parent-child dyads during their actual sleep. Also, given previous evidence highlighting the major role of DMN in sleep, we focused on DMN's between network connectivity. Therefore, we cannot make claims about the specificity of the DMN's connectivity profile in being related to our construct of interest. Thus, it would be informative to observe neural circuit similarity at the global level of the connectome to see how large-scale brain architecture is harmoniously involved in dyadic concordance and shared interpersonal processing (e.g., Lee et al., 2017). Finally, we focused on global measures of sleep quality using the Pittsburgh Sleep Quality Index (PSQI; Buysse et al., 1989). This is a widely used measure that captures clinical levels of poor sleep (i.e., those scoring above 16). While our focus was on global sleep quality, future research should examine multiple aspects of sleep, including insomnia, as well as use more objective measures of sleep with actigraphy. It is possible that different domains of sleep are associated with different neural network connectivity patterns. For example, the degree of anti-correlation in DMN connectivity with other networks (e.g., right frontoparietal network) is related to insomnia (e.g., De Havas et al., 2012). Therefore, future investigation focusing on subcomponents of sleep quality will increase our understanding of the neurobiological basis of sleep. Finally, given the novelty of our approach, and the small sample size, it is important for future research to replicate these effects in larger samples.

In conclusion, a novel feature of the current study is the use of multi-method techniques, including daily diaries across 14 days as well as dyadic brain scans. Our daily diary method to monitor how sleep-time changes day-by-day captures real life experience as it is lived (Bolger et al., 2003) and does not rely on retrospective accounts. To our knowledge, the current dyadic neural concordance analysis is the first empirical analytic approach to evaluate interpersonal neural connectivity beyond one single individual's brain and link it to sleep patterns. Thus, the current multi-level approach significantly contributes to our understanding of adolescents' sleep behavior with a robust and integrated perspective of brain-behavior associations. In sum, we provide the first empirical evidence to highlight the importance of dyadic concordance at both the behavioral and neural level in conferring benefits to adolescents' sleep.

Contributions

All of the authors contributed to the preparation of the manuscript. EHT designed the study and acquired the data. Data were analyzed by THL with MM and EHT.

Conflict of interest

None

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References

Adam, E.K., Snell, E.K., Pendry, P., 2007. Sleep timing and quantity in ecological and family context: a nationally representative time-diary study. J. Fam. Psychol. 21 (1), 4–19.
Basner, M., Rao, H., Goel, N., Dinges, D.F., 2013. Sleep deprivation and neurobehavioral dynamics. Curr. Opin. Neurobiol. 23 (5), 854–863.
Beckmann, C., Smith, S., 2004. Probabilistic independent component analysis for functional magnetic resonance imaging. IEEE Trans. Med. Imaging 23 (2), 137–152.
Beckmann, C., Mackay, C., Filippini, N., Smith, S., 2009. Group comparison of resting-state fmri data using multi-subject ica and dual regression. Neuroimage 47 (Suppl. 1), S148.
Beebe, D.W., 2011. Cognitive, behavioral, and functional consequences of inadequate sleep in children and adolescents. Pediatr. Clin. North Am. 58 (3), 649–665.
Bolger, N., Davis, A., Rafaeli, E., 2003. Diary methods: capturing life as it is lived. Annu. Rev. Psycholol. 54 (1), 579–616.
Boubela, R.N., Kalcher, K., Huf, W., Kronnerwetter, C., Filzmoser, P., Moser, E., 2013. Beyond noise: using temporal ica to extract meaningful information from high-frequency fmri signal fluctuations during rest. Front. Hum. Neurosci. 7 (168), 1–12.
Buckner, R.L., Andrews-Hanna, J.R., Schacter, D.L., 2008. The brain's default network. Ann. N. Y. Acad. Sci. 1124 (1), 1–38.
Buysse, D.J., Reynolds, C.F., Monk, T.H., Berman, S.R., Kupfer, D.J., 1989. The pittsburgh sleep quality index: a new instrument for psychiatric practice and research. Psychiatry Res. 28 (2), 193–213.
Carskadon, M.A., Dement, W.C., 1987. Sleepiness in the normal adolescent. In: Guilleminault, C. (Ed.), Sleep and Its Disorders in Children. Raven Press, New York, pp. 53–66.
Carskadon, M.A., Vieira, C., Acebo, C., 1993. Association between puberty and delayed phase preference. Sleep 16, 256–258.
Chang, A.-M., Aeschbach, D., Duffy, J.F., Czeisler, C.A., 2015. Evening use of light-emitting ereaders negatively affects sleep, circadian timing, and next-morning alertness. Proc. Natl. Acad. Sci. U. S. A. 112 (4), 1232–1237.
Cohen, J.R., Gallen, C.L., Jacobs, E.G., Lee, T.G., D'Esposito, M., 2014. Quantifying the reconfiguration of intrinsic networks during working memory. PLoS One 9 (9), e106636.
Cole, D., Smith, S., Beckmann, C., 2010. Advances and pitfalls in the analysis and interpretation of resting-state fmri data. Front. Syst. Neurosci. 4 (8), 1–15.
Dahl, R.E., Lewin, D.S., 2002. Pathways to adolescent health sleep regulation and behavior. J. Adolesc. Health 31 (6), 175–184.
Dahl, R.E., Holttum, J., Trubnick, L., 1994. A clinical picture of child and adolescent narcolepsy. J. Am. Acad. Child Adolesc. Psychiatry 33 (6), 834–841.
De Havas, J.A., Parimal, S., Soon, C.S., Chee, M.W., 2012. Sleep deprivation reduces default mode network connectivity and anti-correlation during rest and task performance. Neuroimage 59 (2), 1745–1751.
Diez, R., 2002. A glossary for multilevel analysis. J. Epidemiol. Community Health 56 (8), 588–594.
Drummond, S.P., Bischoff-Grethe, A., Dinges, D.F., Ayalon, L., Mednick, S.C., Meloy, M., 2005. The neural basis of the psychomotor vigilance task. Sleep 28 (9), 1059–1068.
El-Sheikh, M., Buckhalt, J.A., Mize, J., Acebo, C., 2006. Marital conflict and disruption of children's sleep. Child Dev. 77 (1), 31–43.
Feis, R.A., Smith, S.M., Filippini, N., Douaud, G., Dopper, E.G., Heise, V., Trachtenberg, A.J., van Swieten, J.C., van Buchem, M.A., Rombouts, S.A., 2015. Ica-based artifact removal diminishes scan site differences in multi-center resting-state fmri. Front. Neurosci. 9 (395), 1–10.
Feldman, R., Magori-Cohen, R., Galili, G., Singer, M., Louzoun, Y., 2011. Mother and infant coordinate heart rhythms through episodes of interaction synchrony. Infant Behav. Dev. 34 (4), 569–577.
Filippini, N., MacIntosh, B., Hough, M., Goodwin, G., Frisoni, G., Smith, S., Matthews, P., Beckmann, C., Mackay, C., 2009. Distinct patterns of brain activity in young carriers of the apoe-ε4 allele. Proc. Natl. Acad. Sci. U. S. A. 106 (17), 7209–7214.
Fox, M.D., Snyder, A.Z., Vincent, J.L., Corbetta, M., Van Essen, D.C., Raichle, M.E., 2005. The human brain is intrinsically organized into dynamic, anticorrelated functional networks. Proc. Natl. Acad. Sci. U. S. A. 102 (27), 9673–9678.
Fuligni, A.J., Tsai, K.M., Krull, J.L., Gonzales, N.A., 2015. Daily concordance between parent and adolescent sleep habits. J. Adolesc. Health 56 (2), 244–250.
Gabard-Durnam, L., Gee, D., Goff, B., Flannery, J., Telzer, E., Humphreys, K., Lumian, D., Fareri, D., Caldera, C., Tottenham, N., 2016. Stimulus-elicited connectivity influences resting-state connectivity years later in human development: a prospective study. J. Neurosci. 36 (17), 4771–4784.
Gee, D., Gabard-Durnam, L., Flannery, J., Goff, B., Humphreys, K., Telzer, E., Hare, T., Bookheimer, S., Tottenham, N., 2013. Early developmental emergence of human amygdala-prefrontal connectivity after maternal deprivation. Proc. Natl. Acad. Sci. U. S. A. 110 (39), 15638–15643.
Halbower, A.C., Degaonkar, M., Barker, P.B., Earley, C.J., Marcus, C.L., Smith, P.L., Prahme, M.C., Mahone, E.M., 2006. Childhood obstructive sleep apnea associates with neuropsychological deficits and neuronal brain injury. PLoS Med. 3 (8), e301.

Herting, M.M., Maxwell, E.C., Irvine, C., Nagel, B.J., 2012. The impact of sex, puberty, and hormones on white matter microstructure in adolescents. Cereb. Cortex 22 (9), 1979–1992.
Horne, J.A., 1993. Human sleep, sleep loss and behaviour: implications for the prefrontal cortex and psychiatric disorder. Br. J. Psychiatry 162 (3), 413–419.
Horovitz, S.G., Braun, A.R., Carr, W.S., Picchioni, D., Balkin, T.J., Fukunaga, M., Duyn, J.H., 2009. Decoupling of the brain's default mode network during deep sleep. Proc. Natl. Acad. Sci. U. S. A. 106 (27), 11376–11381.
IBM_Corp, 2013. IBM Spss Statistics for Windows, Version 24.0. IBM Corp., Armonk, NY.
Jafri, M.J., Pearlson, G.D., Stevens, M., Calhoun, V.D., 2008. A method for functional network connectivity among spatially independent resting-state components in schizophrenia. Neuroimage 39 (4), 1666–1681.
Jan, J.E., Reiter, R.J., Bax, M.C., Ribary, U., Freeman, R.D., Wasdell, M.B., 2010. Long-term sleep disturbances in children: a cause of neuronal loss. Eur. J. Paediatr. Neurol. 14 (5), 380–390.
Joel, S.E., Caffo, B.S., van Zijl, P., Pekar, J.J., 2011. On the relationship between seed-based and ica-based measures of functional connectivity. Magn. Reson. Med. 66 (3), 644–657.
Kalak, N., Gerber, M., Kirov, R., Mikoteit, T., Pühse, U., Holsboer-Trachsler, E., Brand, S., 2012. The relation of objective sleep patterns, depressive symptoms, and sleep disturbances in adolescent children and their parents: a sleep-eeg study with 47 families. J. Psychiatr. Res. 46 (10), 1374–1382.
Komada, Y., Adachi, N., Matsuura, N., Mizuno, K., Hirose, K., Aritomi, R., Shirakawa, S., 2009. Irregular sleep habits of parents are associated with increased sleep problems and daytime sleepiness of children. Tohoku J. Exp. Med. 219 (2), 85–89.
Kopp, C., Longordo, F., Nicholson, J.R., Lüthi, A., 2006. Insufficient sleep reversibly alters bidirectional synaptic plasticity and nmda receptor function. J. Neurosci. 26 (48), 12456–12465.
Laird, A., Fox, P., Eickhoff, S., Turner, J., Ray, K., McKay, D., Glahn, D., Beckmann, C., Smith, S., Fox, P., 2011. Behavioral interpretations of intrinsic connectivity networks. J. Cogn. Neurosci. 23 (12), 4022–4037.
Larson-Prior, L.J., Power, J.D., Vincent, J.L., Nolan, T.S., Coalson, R.S., Zempel, J., Snyder, A.Z., Schlaggar, B.L., Raichle, M.E., Petersen, S.E., 2011. Modulation of the brain's functional network architecture in the transition from wake to sleep. Prog. Brain Res. 193, 277–294.
Laufs, H., Krakow, K., Sterzer, P., Eger, E., Beyerle, A., Salek-Haddadi, A., Kleinschmidt, A., 2003. Electroencephalographic signatures of attentional and cognitive default modes in spontaneous brain activity fluctuations at rest. Proc. Natl. Acad. Sci. U. S. A. 100 (19), 11053–11058.
Lee, T.-H., Telzer, E.H., 2016. Negative functional coupling between the right fronto-parietal and limbic resting state networks predicts increased self-control and later substance use onset in adolescence. Dev. Cogn. Neurosci. 20, 35–42.
Lee, T.-H., Miernicki, M.E., Telzer, E.H., 2017. Families that fire together smile together: resting state connectome similarity and daily emotional synchrony in parent-child dyads. Neuroimage 152, 31–37.
Lemieux, L., Salek-Haddadi, A., Lund, T.E., Laufs, H., Carmichael, D., 2007. Modelling large motion events in fmri studies of patients with epilepsy. Magn. Reson. Imaging 25 (6), 894–901.
Li, S., Zhu, S., Jin, X., Yan, C., Wu, S., Jiang, F., Shen, X., 2010. Risk factors associated with short sleep duration among chinese school-aged children. Sleep Med. 11 (9), 907–916.
McKenna, B.S., Eyler, L.T., 2012. Overlapping prefrontal systems involved in cognitive and emotional processing in euthymic bipolar disorder and following sleep deprivation: a review of functional neuroimaging studies. Clin. Psychol. Rev. 32 (7), 650–663.
Mirescu, C., Peters, J.D., Noiman, L., Gould, E., 2006. Sleep deprivation inhibits adult neurogenesis in the hippocampus by elevating glucocorticoids. Proc. Natl. Acad. Sci. U. S. A. 103 (50), 19170–19175.
Nelson, E., Leibenluft, E., McClure, E., Pine, D., 2005. The social re-orientation of adolescence: a neuroscience perspective on the process and its relation to psychopathology. Psychol. Med. 35 (2), 163–174.
Paolini, M., Keeser, D., Ingrisch, M., Werner, N., Kindermann, N., Reiser, M., Blautzik, J., 2015. Resting-state networks in healthy adult subjects: a comparison between a 32-element and an 8-element phased array head coil at 3.0 tesla. Acta Radiol. 56 (5), 605–613.
Pernet, C.R., Wilcox, R., Rousselet, G.A., 2012. Robust correlation analyses: false positive and power validation using a new open source matlab toolbox. Front. Psychol. 3 (606), 1–18.
Picchioni, D., Duyn, J.H., Horovitz, S.G., 2013. Sleep and the functional connectome. Neuroimage 80, 387–396.
Poldrack, R.A., Mumford, J.A., Nichols, T.E., 2011. Handbook of Functional Mri Data Analysis. Cambridge University Press, Cambridge,U.K.
Power, J.D., Barnes, K.A., Snyder, A.Z., Schlaggar, B.L., Petersen, S.E., 2012. Spurious but systematic correlations in functional connectivity mri networks arise from subject motion. Neuroimage 59 (3), 2142–2154.
Pruim, R., Mennes, M., Buitelaar, J., Beckmann, C., 2015a. Evaluation of ica-aroma and alternative strategies for motion artifact removal in resting state fmri. Neuroimage 112, 278–287.
Pruim, R., Mennes, M., van Rooij, D., Llera, A., Buitelaar, J., Beckmann, C., 2015b. Ica-aroma: a robust ica-based strategy for removing motion artifacts from fmri data. Neuroimage 112, 267–277.
Raudenbush, S., Bryk, A., Congdon, R., 2013. Hlm 7.01 for windows [hierarchical linear and nonlinear modeling software].
Roane, B.M., Taylor, D.J., 2008. Adolescent insomnia as a risk factor for early adult depression and substance abuse. Sleep 31 (10), 1351–1356.
Rosenberg, M.D., Finn, E.S., Scheinost, D., Papademetris, X., Shen, X., Constable, R.T., Chun, M.M., 2015. A neuromarker of sustained attention from whole-brain functional connectivity. Nat. Neurosci. 19 (1), 165–171.
Sämann, P.G., Tully, C., Spoormaker, V.I., Wetter, T.C., Holsboer, F., Wehrle, R., Czisch, M., 2010. Increased sleep pressure reduces resting state functional connectivity. Magn. Reson. Mater. Phys. Biol. Med. 23 (5–6), 375–389.
Satterthwaite, T.D., Elliott, M.A., Gerraty, R.T., Ruparel, K., Loughead, J., Calkins, M.E., Eickhoff, S.B., Hakonarson, H., Gur, R.C., Gur, R.E., 2013. An improved framework for confound regression and filtering for control of motion artifact in the pre-processing of resting-state functional connectivity data. Neuroimage 64, 240–256.
Shirer, W., Ryali, S., Rykhlevskaia, E., Menon, V., Greicius, M., 2012. Decoding subject-driven cognitive states with whole-brain connectivity patterns. Cereb. Cortex 22 (1), 158–165.
Smith, S., Fox, P., Miller, K., Glahn, D., Fox, P., Mackay, C., Filippini, N., Watkins, K., Toro, R., Laird, A., Beckmann, C., 2009. Correspondence of the brain's functional architecture during activation and rest. Proc. Natl. Acad. Sci. U. S. A. 106 (31), 13040–13045.
Starck, T., Nikkinen, J., Rahko, J., Remes, J., Hurtig, T., Haapsamo, H., Jussila, K., Kuusikko-Gauffin, S., Mattila, M.-L., Jansson-Verkasalo, E., 2013. Resting state fmri reveals a default mode dissociation between retrosplenial and medial prefrontal subnetworks in asd despite motion scrubbing. Front. Hum. Neurosci. 22 (802), 1–10.
Tamnes, C.K., Østby, Y., Fjell, A.M., Westlye, L.T., Due-Tønnessen, P., Walhovd, K.B., 2010. Brain maturation in adolescence and young adulthood: regional age-related changes in cortical thickness and white matter volume and microstructure. Cereb. Cortex 20 (3), 534–548.
Tazawa, Y., Okada, K., 2001. Physical signs associated with excessive television-game playing and sleep deprivation. Pediatr. Int. 43 (6), 647–650.
Telzer, E.H., Goldenberg, D., Fuligni, A.J., Lieberman, M.D., Gálvan, A., 2015. Sleep variability in adolescence is associated with altered brain development. Dev. Cogn. Neurosci. 14, 16–22.
Tohka, J., Foerde, K., Aron, A.R., Tom, S.M., Toga, A.W., Poldrack, R.A., 2008. Automatic independent component labeling for artifact removal in fmri. Neuroimage 39 (3), 1227–1245.
Tsai, K.M., Telzer, E.H., Fuligni, A.J., 2013. Continuity and discontinuity in perceptions of family relationships from adolescence to young adulthood. Child Dev. 84 (2), 471–484.
Van den Bulck, J., 2004. Television viewing, computer game playing, and internet use and self-reported time to bed and time out of bed in secondary-school children. Sleep 27 (1), 101–104.
Wheatley, T., Kang, O., Parkinson, C., Looser, C.E., 2012. From mind perception to mental connection: synchrony as a mechanism for social understanding. Soc. Pers. Psychol. Compass 6 (8), 589–606.
Wolfson, A.R., Carskadon, M.A., 1998. Sleep schedules and daytime functioning in adolescents. Child Dev. 69 (4), 875–887.
Wong, M.M., Brower, K.J., Nigg, J.T., Zucker, R.A., 2010. Childhood sleep problems, response inhibition, and alcohol and drug outcomes in adolescence and young adulthood. Alcohol.: Clin. Exp. Res. 34 (6), 1033–1044.
Zhang, L.-j., Wang, H.-s., Huang, X.-n., 2010. Sleep patterns amongst chinese children. Biol. Rhythm Res. 41 (3), 203–215.