Geographic Variation in the Prevalence of Attention-Deficit/Hyperactivity Disorder: The Sunny Perspective

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Archival Report

Geographic Variation in the Prevalence of Attention-Deficit/Hyperactivity Disorder: The Sunny Perspective

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Background: Attention-deficit/hyperactivity disorder (ADHD) is the most common psychiatric disorder of childhood, with average worldwide prevalence of 5.3%, varying by region.

Methods: We assessed the relationship between the prevalence of ADHD and solar intensity (SI) (kilowatt hours/square meters/day) on the basis of multinational and cross-state studies. Prevalence data for the U.S. were based on self-report of professional diagnoses; prevalence data for the other countries were based on diagnostic assessment. The SI data were obtained from national institutes.

Results: In three datasets (across 49 U.S. states for 2003 and 2007, and across 9 non-U.S. countries) a relationship between SI and the prevalence of ADHD was found, explaining 34%–57% of the variance in ADHD prevalence, with high SI having an apparent preventative effect. Controlling for low birth weight, infant mortality, average income (socioeconomic status), latitude, and other relevant factors did not change these findings. Furthermore, these findings were specific to ADHD, not found for the prevalence of autism spectrum disorders or major depressive disorder.

Conclusions: In this study we found a lower prevalence of ADHD in areas with high SI for both U.S. and non-U.S. data. This association has not been reported before in the literature. The preventative effect of high SI might be related to an improvement of circadian clock disturbances, which have recently been associated with ADHD. These findings likely apply to a substantial subgroup of ADHD patients and have major implications in our understanding of the etiology and possibly prevention of ADHD by medical professionals, schools, parents, and manufacturers of mobile devices.

Key Words: ADHD, chronobiological, circadian, light, prevalence, solar intensity

Attention-deficit/hyperactivity disorder (ADHD) is the most common psychiatric disorder of childhood, with average worldwide prevalence of 5.3% (1). Heritability of ADHD has been reported as 60%–70% (2). Pre- and perinatal factors (preterm birth, low birth weight [LBW]) also play an important role in the etiology of ADHD (3). Additional factors might include global living conditions that could be investigated by inspecting geographic differences in ADHD prevalence.

Previous research reported that 78% of adult unmedicated (4) and one third of medication-naïve pediatric (5) ADHD patients have idiopathic sleep onset insomnia (SOI), accompanied by delayed circadian phase as measured by delayed dim light melatonin onset (6). This finding is supported by melatonin signaling deficiencies (7), clock gene abnormalities (8), and a higher prevalence of "evening types" in adult ADHD (9). Furthermore, a recent meta-analysis incorporating data from 35,936 healthy children reported that sleep duration correlates positively with school performance, executive function, and negatively with internalizing and externalizing behavior problems (10). In addition, a sleep restriction regime for 3–7 days results in cumulative impairment of attention in healthy adults (11,12) and children (13) and in a tendency for increased θ electroencephalogram (EEG) power (effect size d = .53), (14), which is a measure reported in some ADHD studies, also reflective of fatigue or drowsiness (15,16). These impairments of attention did not normalize after a single night of recovery sleep but only after at least the same number of recovery nights as the number of sleep-restricted nights (11,12). Chronobiological interventions in ADHD patients with insomnia, such as early-morning bright light (17) and long-term melatonin treatment (18), have resulted in an improvement of ADHD symptoms. Additionally, improvement of ADHD symptoms was shown after treatment of other sleep disorders, such as dopamine agonist (19) or iron supplementation (20) for restless legs syndrome and adenotonsillectomy in sleep apnea by (21). Together, these results suggest that, at least in a subgroup of ADHD, the symptoms are exacerbated by sleep problems. In the aforementioned SOI studies, patients were unmedicated (4) or medication-naïve (5). Many well-controlled studies in unmedicated ADHD patients have reported these sleep problems [for review see Yoon *et al.* (22)]. Thus the increased prevalence of sleep problems in ADHD cannot be explained by effects of stimulant medication. Nevertheless, stimulants often aggravate sleep problems that already existed before stimulant use.

The association of ADHD with circadian disturbances and the powerful impact of bright light on the circadian rhythm, sleep, and daytime function led us to investigate in more detail the relationship between environmental light exposure and ADHD prevalence. This was further encouraged by a graph from the U.S. Centers for Disease Control and Prevention (CDC) (23) depicting differences in ADHD prevalence for the U.S. (Figure 1, left) and solar intensity (SI) maps for the U.S., available from the National Renewable Energy Laboratory (NREL) (Figure 1, right). We noted that the U.S. states with maximum SI (Arizona, New Mexico,

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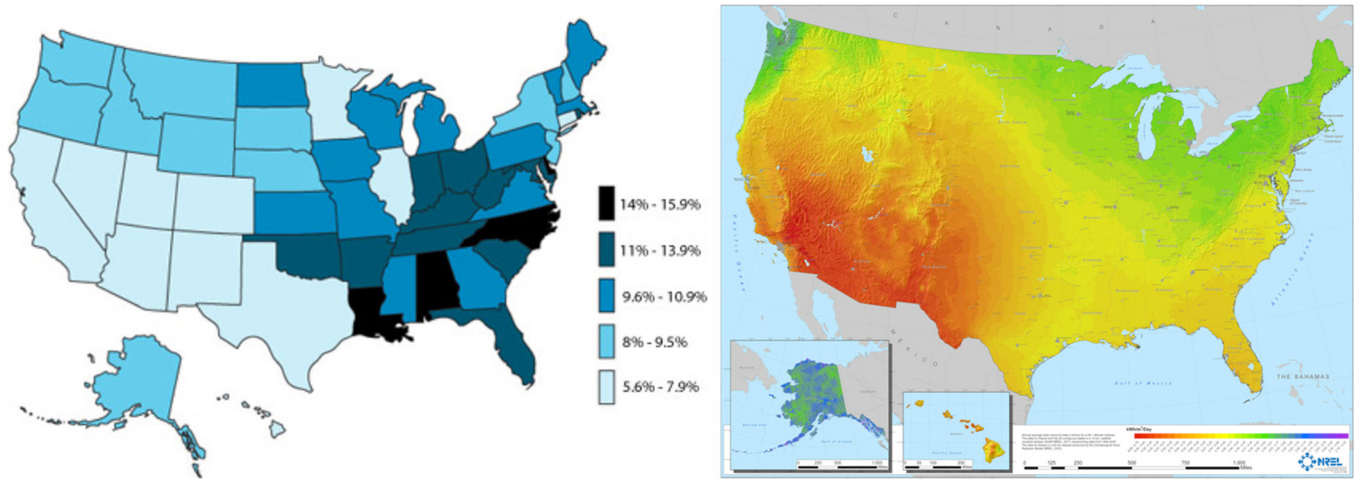

Figure 1. Comparison between the prevalence of attention-deficit/hyperactivity disorder (ADHD) and solar intensity (SI) in the U.S. This figure demonstrates on the left map the prevalence rate of ADHD among different U.S. states (U.S. Centers for Disease Control and Prevention, 2010; http://www.cdc.gov/ncbddd/adhd/prevalence.html). The figure on the right depicts the horizontal irradiation or SI (SI in kilowatt hours/square meters/day) across the U.S. as reported by the National Renewable Energy Laboratory (map created by the National Renewable Energy Laboratory for the Department of Energy). Note the similarity between the areas with highest SI (depicted in red) and those with the lowest prevalence of ADHD (palest blue).

Nevada, California, Utah, and Colorado) also had the lowest prevalence of ADHD. Therefore, in this study we sought to systematically investigate the relationship between the prevalence of ADHD and SI as a measure of visible and nonimage forming light (expressed in kilowatt hours/square meters/day). We hypothesized that high SI is associated with lower rates of ADHD by its overwhelming phase-advancing effect on the circadian clock.

Methods and Materials

ADHD Prevalence Estimates

Searches were performed to uncover statistics about the prevalence (PREV) of ADHD around the world or across states employing standardized procedures, to facilitate valid comparisons of PREV estimates.

Solar Intensity

Horizontal irradiation (radiation reaching the surface of the earth on a horizontal plane, expressed in kilowatt hours/square meters/day; SI data: SI) were obtained from national and international agencies: U.S. National Renewable Energy Laboratory (NREL[A]) for the U.S. (NREL: P. Gray-Hann, personal communication, August 24, 2012), global horizontal irradiation, State University of New York (Mexico), global horizontal irradiation, National Institute for Spatial Research (Colombia), and the European Institute for Energy and Transport (IET[B]) for Europe and other countries.

The radiation model used by NREL was developed by Perez *et al.* (24) and "…uses hourly radiance images from geostationary weather satellites, daily snow cover data, and monthly averages of atmospheric water vapor, trace gases, and the mount of aerosols in the atmosphere to calculate the hourly total insolation…falling on a horizontal surface…" (25). Similar methods were used by IET; for more details see Šúri *et al.* (26).

Statistics

The relationship between PREV and SI was investigated by curve estimation. For nonlinear relationships, an appropriate transformation (log or square root) was applied to obtain the most significant linear trend. A correlation was calculated between PREV and SI as well as with other potential confounding variables, such as latitude, altitude, and (for the U.S.) average income (socioeconomic status), LBW, and infant mortality (IM)/state (CDC data) and other factors reported by the ADHD PREV studies obtained (Pearson correlation, two-tailed). Those factors that were found significantly correlated with PREV were introduced as control variables in partial correlation analyses between PREV and SI (two-tailed). Finally, to investigate specificity for ADHD, we conducted the same analyses for autism spectrum disorders (ASD) and major depressive disorder (MDD) in the U.S. data that were also obtained from the CDC.

Results

Two data sources were identified that employed identical methods to estimate the prevalence of ADHD. These sources were the PREV estimates in children from the CDC/U.S. state (23) and the PREV estimates for adults across several countries (27).

ADHD Prevalence Across U.S. States: CDC

The CDC data were collected in 2003 and 2007 and were part of the National Survey of Children's Health in children under 18 years of age. This survey was a national, cross-sectional, random-digit-dialed landline telephone survey, where one child was selected randomly from each household to be the focus of the parent or guardian interview (response rates: 68.8% in 2003 and 46.7% in 2007) (23). Parents were asked whether or not a doctor or other healthcare provider had ever told them that their child had "attention-deficit disorder or attention-deficit/hyperactivity disorder, that is, ADD or ADHD" (23). Higher rates of ADHD were found among boys, multiracial children, and children covered by Medicaid.

Figure 2 demonstrates the relationship between SI and PREV for 2003 and 2007. As can be seen from Figure 2, this relationship does not seem linear, which was confirmed by curve estimation (all statistics are represented for 2003 | 2007, respectively). The best fit was obtained by a sigmoidal dose-response relation

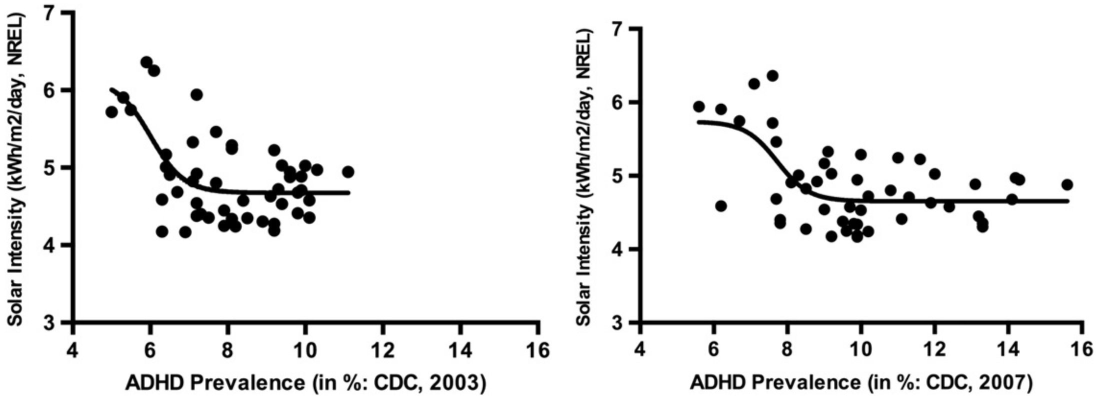

Figure 2. The association between attention-deficit/hyperactivity disorder (ADHD) prevalence rates and solar intensity for different U.S. states for 2003 (left) and 2007 (right). A sigmoidal dose-response relation best describes the association. CDC, U.S. Center for Disease Control and Prevention; NREL, National Renewable Energy Laboratory.

[$F_{1,46} = 13.78$; $p = .0006$; $R^2 = .36$ $\mid$ $F_{1,46} = 16.38$; $p = .0002$; $R^2 = .37$], and log transformation of PREV and SI resulted in the best linear fit with a significant correlation between PREV and SI [$p = .002$; $r = -.429$ $\mid$ $p = .004$; $r = -.409$; $df = 49$]. The PREV (log transformed) did not correlate with socioeconomic status (all p values $< .199$) but did correlate with latitude for 2003 [$p = .050$; $r = -.281$; $df = 49$] but not for 2007 [$p = .094$; $r = -.242$; $df = 49$]. Furthermore, PREV was positively associated with percentage LBW [$p = .001$; $r = .468$; $df = 49$ $\mid$ $p = .003$; $r = .422$; $df = 49$] and IM [$p = .001$; $r = .468$; $df = 49$ $\mid$ $p = .001$; $r = .457$; $df = 49$], in line with the literature (3). Partial correlations including the aforementioned variables as covariates made the relationship between PREV and SI stronger [$p < .000$; $r = -.637$; $df = 44$ $\mid$ $p < .000$; $r = -.580$; $df = 44$], resulting in explained variance of 34%–41% of ADHD prevalence.

To control for the demographic differences in ADHD PREV as reported by CDC (23), we also conducted partial correlations adding Medicaid coverage (CDC data), male/female ratio (census data) and percentage multi-raciality (census data)/state, which did not change the relationship between ADHD PREV and SI for 2003 or 2007 (all p values $\leq .003$). To control further for racial and ethnic differences, we also included the percentage Hispanic or Latino population/state (census data), which slightly lowered the correlation between SI and PREV but still resulted in a significant association [$r = -.443$; $p = .002$; $df = 43$ $\mid$ $r = -.353$; $p = .017$; $df = 43$].

To test whether the aforementioned findings were moderated by seasonal fluctuations in light intensity, the aforementioned analyses were repeated with SI data/month and PREV (2003 or 2007). Partial correlations correcting for latitude, LBW, and IM resulted in significant correlations between all monthly SI values and ADHD PREV for 2003 and 2007 (all $p < .004$; $-.415 < r < -.661$), suggesting that differences in monthly SI did not influence the association SI with PREV.

When we repeated the aforementioned analysis excluding states with an SI > 5.5 (Arizona, New Mexico, Nevada, California, Utah, and Colorado, representing 22% of the total U.S. population on the basis of the 2010 U.S. census data) from the analysis, the partial correlation between SI and PREV weakened but remained significant for 2003 [$p = .041$; $r = -.324$; $df = 38$] and marginally significant for 2007 [$p = .082$; $r = -.278$; $df = 38$], suggesting these effects are mostly driven by the high SI in these six states. These six states are all characterized by a higher elevation, so we checked for effect of high-altitude thin air. There was a significant correlation for the whole sample between PREV and elevation [$p < .000$; $r = -.672$; $df = 49$ $\mid$ $p < .000$; $r = -.571$; $df = 49$] and between SI and elevation [$p < .000$; $r = .644$; $df = 49$]. However, partial correlations between PREV and elevation were no longer significant when adding SI as a covariate ($p < .4$), whereas adding elevation as a covariate still allowed marginally significant and significant associations between PREV and SI for 2003 and 2007, respectively ($p = .076$ $\mid$ $p = .007$).

Prevalence rates were also available for ASD (14 U.S. States, 2008) and MDD (44 U.S. states, 2006–2008) from the CDC. The aforementioned analyses were repeated for ASD and MDD as well and did not result in any significant associations between PREV and SI ($p < .913$).

Non-U.S. Adult ADHD Prevalence Rates, Cross-National Data

In a multinational study by Fayyad *et al.* (27) identical methods were used to estimate the PREV of adult ADHD. A total of 11,422 respondents (18–44 years of age) from multi-stage household probability samples were screened by face-to-face surveys. Subjects were retrospectively assessed for childhood ADHD with the Diagnostic Interview Schedule for DSM-IV (28), and if subjects met childhood ADHD criteria, they were asked about whether they continued to have current problems with attention or hyperactivity-impulsivity. The response rate was 67.9% (27).

These authors reported a significantly lower PREV for Spain, Lebanon, Colombia, and Mexico and a significantly higher PREV for France as compared with Italy, Germany, Belgium, The Netherlands, and the U.S. sociodemographic effects on ADHD PREV were only found for a higher prevalence in men and among people educated less than at University level, albeit with modest magnitude ($1.5 <$ odds ratio < 3.0), and these factors did not differ between countries. The four countries with lower prevalence also have the highest SI compared with the other countries, as can be seen in Figure 3. In this study we excluded the results from the U.S., because those have been analyzed in the preceding text in more detail.

A sigmoid dose-response was observed for the relation between PREV and SI [$F_{1,6} = 15.83$; $p = .0073$; $R^2 = .85$]. A log transformation of PREV and SI resulted in a linear relationship and a significant correlation between PREV and SI [$p = .018$;

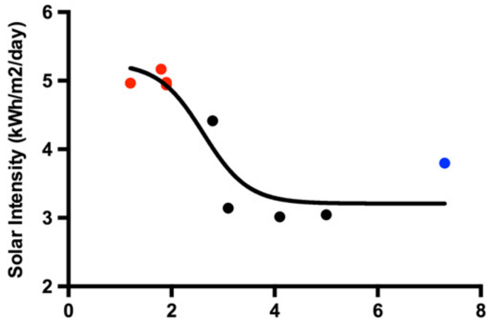

Figure 3. Association between attention-deficit/hyperactivity disorder (ADHD) prevalence rates and solar intensity for non-U.S. countries. The relation between solar intensity and the prevalence of adult ADHD in several non-U.S. countries and the sigmoid dose-response trend, which best explained the data. Red depicts the four countries (two points are overlaid) that had a significantly lower prevalence of adult ADHD, and blue indicates the country with a significant increased prevalence of adult ADHD (France), as was reported by Fayyad *et al.* (27).

$r = -.758$; $df = 9$] and no association with latitude [$p = .111$; $r = .567$; $df = 9$] or altitude [$p = .136$; $r = -.537$; $df = 9$], suggesting that SI explained 57% of the variance in the prevalence of non-U.S. adult ADHD. This is a small sample but essentially replicates the pattern that was also observed for the U.S.

Discussion

The data as presented here demonstrate an interesting relationship between SI and the prevalence of ADHD, where SI explained 34%–41% of the variance in ADHD diagnosis in children from the U.S. and 57% of the variance in adult ADHD prevalence in non-U.S. countries. Although LBW and IM also explained 18%–22% of the variance in ADHD PREV, in agreement with earlier studies (3), correcting for these factors only made the effects for SI stronger, suggesting these are independent risk factors. This relationship between ADHD PREV and SI has to our knowledge not been investigated before in ADHD. More specifically, as can be seen in Figures 1–3, it seems that a high SI is associated with lower prevalence rates and can thus be considered a "protective" factor for ADHD symptoms, also clear from the sigmoid dose-response relations found for all data. These results seem specific to ADHD, because this relation was not found for ASD and MDD and could not be explained by other factors such as elevation, LBW, IM, or local differences in healthcare availability and accessibility. The U.S. results were mainly driven by the 6 U.S. states with $SI > 5.5$; these states represent 22% of the U.S. population, so it would not be reasonable to disregard the findings from these states as "outliers."

The lack of an association between SI and MDD PREV seems surprising. The CDC prevalence rates for MDD are in line with an earlier cross-national study by Kessler *et al.* (29), where the lowest MDD prevalence was found in the Northeast and Midwest of the U.S., further ruling out SI or latitude to have a protective effect on MDD PREV in the U.S. Although it is well known that for seasonal affective disorder (SAD) bright light is an effective treatment (30), the CDC data did not incorporate a separate SAD category but only MDD. Therefore, these data specifically relate to general MDD and do not represent specific SAD prevalence. Furthermore, light treatment as a standalone treatment in non-SAD depression has yielded inconsistent results (30).

Limitations of these results consist of the fact that the CDC data employed screening methods not identical to diagnostic procedures as carried out in standard care, which might result in an overestimation of ADHD PREV (31). Furthermore, in both studies attrition rates were between 32% and 63%, and it is unclear how this would affect the ADHD PREV estimates. Given the large number of respondents in both studies (CDC: 73,123 families; Fayyad: 11,422 respondents) and that the results were similar for both datasets, these results at least suggest a relationship between SI and attention deficit syndromes. Another limitation is that, for the two analyses, SI data were obtained from different agencies (U.S.: NREL; and Europe: IET). The SI estimates from NREL in kilowatt hours/square meters/day seem to be higher as compared with the IET estimates (also compare Figures 2 and 3). Therefore, future studies should aim to "standardize" these values internationally, whereby this relationship can be modeled more accurately. Finally, it cannot be ruled out that another—as yet—unknown factor could explain this relationship (e.g., cultural attitudes). These data only reflect a "correlation" and not "causation."

A variety of reasons for the associations observed in this study might be hypothesized. An obvious one could be vitamin D levels, which depend to a large extent on solar radiation changing 7-dehydroxycholesterol in the skin to vitamin D. A search on the relation between ADHD and vitamin D did not yield relevant results, except for a recent large study that found no association between vitamin D and behavioral problems in children (32); hence it is unlikely that this result can be explained by vitamin D deficiency specific to ADHD. Although vitamin D could contribute partially or indirectly to the observed protective effect, it does not seem to be the prime explanation. Conversely, approximately 80% of adult ADHD patients and one third of children with ADHD suffer from SOI, characterized by a delayed circadian phase and delayed melatonin peak (4,6). Recent studies have suggested that the increased use of modern (social) media (iPads, mobile phones) by children and adolescents, especially shortly before bedtime, results in delayed sleep onset (33,34), shorter sleep duration (34), and melatonin suppression (35,36). Technological advances have increased the screens, pixels, and light intensity of modern media substantially across the last 10–15 years, and the closer proximity of mobile devices to the eyes as compared with television sets results in exposure to increased light intensity, particularly within the 464–484-nm spectrum. This wavelength of blue light has been shown to affect the photosensitive melanopsin receptors responsible for non–image-forming visual processes, which directly project to the suprachiasmatic nuclei, which act as the circadian pacemaker. Furthermore, the increasing popularity of social media has substantially increased the time spent with mobile devices compared with 10 years ago. In addition a recent meta-analysis reported that the EEG θ/β ratio (an EEG marker found in some ADHD studies) linearly increased over the last 10 years for healthy children, reflecting increasing levels of daytime drowsiness (16). Thus we hypothesize that increased evening use of modern media and social media might result in suppressed evening melatonin levels, delayed circadian phase with associated delayed sleep onset, reduced sleep duration, and finally increased θ EEG power as well as an increased prevalence of ADHD in genetically susceptible children. The apparent preventative effect of high SI on ADHD prevalence might thus result from the ability of intense natural light during the morning to counteract the phase delaying effects of modern media in the evening, thus preventing the delayed sleep onset and reduced sleep duration.

This hypothesis is in line with recently reported results of morning bright light as a treatment for adults with ADHD (17) as well as with the sigmoidal dose-response effects for SI and PREV, which is also reported for circadian measures such as multi-unit activity of the suprachiasmatic nuclei in response to light intensity (37) and melatonin suppression as a result of exposure to light (38). Interestingly, in the CDC data the most significant increase in ADHD PREV between 2003 and 2007 was noted for adolescents 15–17 years old ($p < .001$) compared with 4–10-year ($p = .013$) and 11–14-year ($p = .016$) age groups. Furthermore, a recent study on the use of stimulant medication in the U.S. also found that across the last 12 years there was a slow but steady increase in pediatric stimulant use, primarily as a result of increased stimulant use in adolescents (39). We would expect that adolescents more often engage in modern media use in the evening as compared with younger children.

Controlled studies are required to prospectively replicate these findings and investigate whether intense light/solar exposure during the day, particularly in the morning, or reduced light exposure in the evening (especially blue 464–484-nm light) could reduce ADHD symptoms or maybe "treat" ADHD. This could open the way to prevention of a subgroup of ADHD or children with attention-deficit syndrome in a variety of ways: for example, exposing children more to natural light during the day (skylight to bring natural light into classrooms and scheduling more outside play time in the morning rather than in the afternoon); reducing exposure to blue light in the evening by parental control; or encouraging device manufacturers to control the emission of blue light from mobile devices on the basis of time of day. Furthermore, these results suggest that future genetic studies of ADHD might include genes involved in the circadian system and incorporate sleep data (e.g., actigraphy) and circadian parameters (dim light melatonin onset). These might define intermediate endophenotypes in the etiology of ADHD subgroups from gene $\times$ environment interactions.

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MA initiated the article and has been involved with the data collection, data analysis, and manuscript writing and had full access to the data and takes responsibility for the integrity of the data and the accuracy of the data analysis. KVDH and JLK have contributed with suggestions for data analysis, interpretation of results, and manuscript editing at all stages. LEA has critiqued the ideas from the beginning, contributed several alternative hypotheses to be tested (e.g., elevation and vitamin D), and reviewed/edited several drafts of the article.

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