Geographic variation in the prevalence of ADHD: The Sunny perspective.

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Abstract:

Background: Attention-Deficit/Hyperactivity Disorder (ADHD) is the most common psychiatric disorder of childhood with an average worldwide prevalence of 5.3%.

Methods: In this study we assessed the relationship between the prevalence of ADHD and solar intensity (SI: kWh/m2/day) based on multinational and cross-state studies employing identical methods to estimate the prevalence of ADHD. SI data were obtained from national institutes.

Results: In three datasets (across 49 US States for 2003 and 2007 and across 9 non-US countries) a clear 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 (SES), 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 nor major depressive disorder.

Conclusions: In this study we found a lower prevalence of ADHD in areas with high SI for both US and non-US data. This association has not been reported before in the literature. The preventative effect of high SI may be related to an improvement of circadian clock disturbances, which have recently been associated with ADHD. These findings likely apply to a substantial sub-group 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.

Introduction

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 may include global living conditions that could be investigated by inspecting geographic differences in ADHD prevalence.

Previous research reported that 78% of adult (4) and one-third of pediatric (5) unmedicated ADHD patients have idiopathic sleep onset insomnia (SOI), accompanied by delayed circadian phase as measured by delayed Dim Light Melatonin Onset (DLMO) (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 theta EEG power (ES d=0.53), (14), which is a biomarker associated with ADHD that reflects 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

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 US Centre for Disease Control and Prevention (22) (CDC) depicting differences in ADHD prevalence for the US (figure 1, left) and solar intensity maps for the US, available from the National Renewable Energy Laboratory (NREL; figure 1 right). We noted that the US states with maximum solar intensity (Arizona, New Mexico, 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 solar intensity (SI). We hypothesized that high SI is associated with lower rates of ADHD by its overwhelming phase advancing effect on the circadian clock.

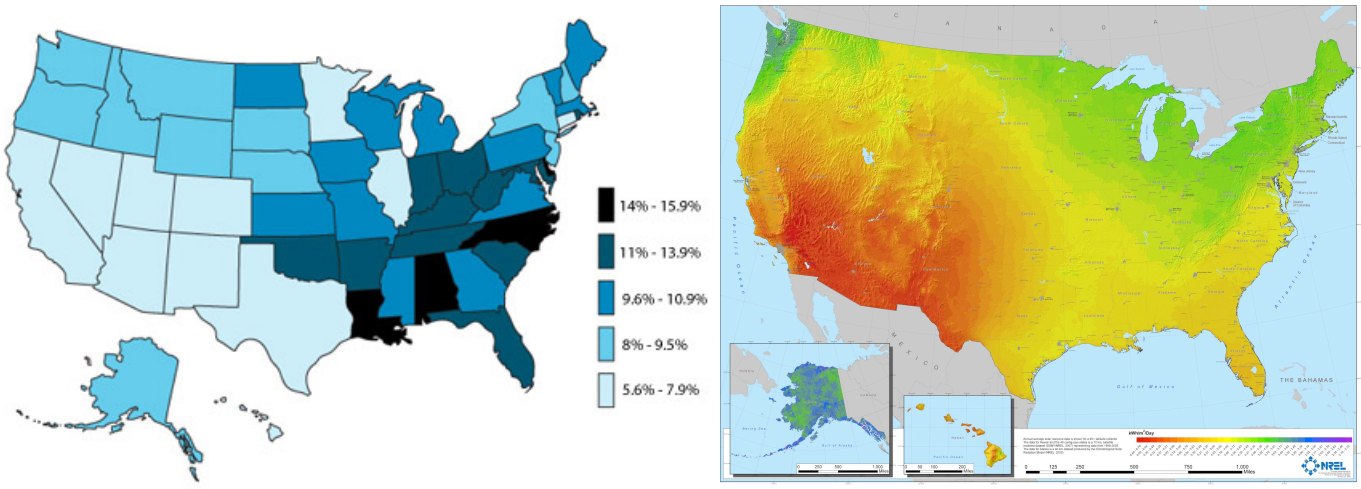

Figure 1. Comparison between the prevalence of ADHD and solar intensity in the US.

This figure demonstrates on the left map the prevalence rate of ADHD among different US states (CDC, 2010). The figure on the right depicts the horizontal irradiation or solar intensity (SI in $kWh/m^2/day$) across the US as reported by NREL (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).

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, in order to facilitate valid comparisons of PREV estimates.

Solar Intensity (SI)

Horizontal irradiation (radiation reaching the earth's surface on a horizontal plane, expressed in $kWh/m^2/day$; solar intensity data: SI) were obtained from (inter-) national agencies: US National

Renewable Energy Laboratory (NREL[A]) for the US (NREL: Gray-Hann personal communication), GHI SUNY (Mexico) and GNI INPE (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. (23) and: *"...uses hourly radiance images from geostationary weather satellites, daily snow cover data, and monthly averages of atmospheric water vapour, trace gases, and the mount of aerosols in the atmosphere to calculate the hourly total insolation ... falling on a horizontal surface..."* (24). Similar methods were used by IET; for more details see (25).

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 US) average income (SES), LBW and infant mortality (IM) per state (CDC data) and other factors reported by the ADHD PREV studies obtained. Those factors that were found significantly correlated with PREV, were introduced as control variables in partial correlation analyses between PREV and SI. Finally, in order to investigate specificity for ADHD, we conducted the same analyses for Autism Spectrum Disorders (ASD) and Major Depressive Disorder (MDD) in the US data that were also obtained from the CDC.

Results

Two data sources were identified which employed identical methods to estimate the prevalence of ADHD. These sources were the PREV estimates in children from the CDC per US state (22) and the PREV estimates for adults across several countries (26).

ADHD Prevalence across US states: CDC

The CDC data were collected in 2003 and 2007 and were part of the National Survey of Children's Health (NSCH) 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) (22). Parents were asked whether or not a doctor or other health-care provider had ever told them that their child had "attention-deficit disorder or attention-deficit/hyperactivity disorder, that is, ADD or ADHD" (22). 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 appear 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 ($p=0.0006$; $F=13.78$; $R^2=0.36$; $DF=1,46$ | $p=0.0002$; $F=16.38$; $R^2=0.37$; $DF=1,46$), and log transformation of PREV and SI resulted in the best linear fit with a significant correlation between PREV and SI ($p=0.002$; $r=-0.429$ | $p=0.004$; $r=-0.409$; $DF=49$). PREV (log transformed) did not correlate with SES (all $p>0.199$) but did correlate with latitude for 2003 ($p=0.050$; $r=-0.281$; $DF=49$) but not for 2007 ($p=0.094$; $r=-0.242$; $DF=49$). Furthermore, PREV was positively associated with percentage LBW ($p=0.001$; $r=0.468$; $DF=49$ | $p=0.003$;

r=0.422; DF=49) and IM (p=0.001; r=0.468; DF=49 | p=0.001; r=0.457; DF=49), in line with the literature (3). Partial correlations including the above variables as covariates made the relationship between PREV and SI stronger (p<0.000; r=-0.637; DF=44 | p<0.000; r=-0.580; DF=44), resulting in explained variance of 34-41% of ADHD prevalence.

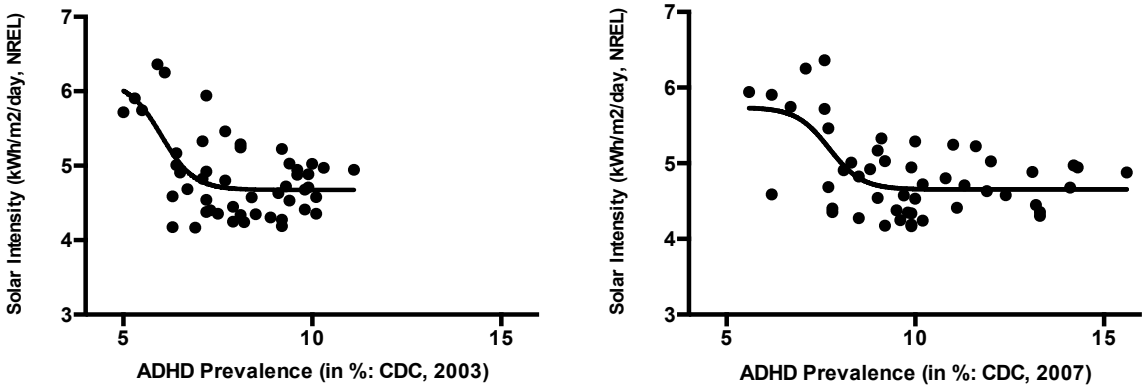

Figure 2. The association between ADHD prevalence rates and solar intensity for different US states for 2003 (left) and 2007 (right). A sigmoidal dose-response relation best describes the association.

In order to control for the demographic differences in ADHD PREV as reported by CDC (22), we also conducted partial correlations adding Medicaid coverage (CDC data), male:female ratio (CENSUS data) and percentage multi-raciality (CENSUS data) per state, which did not change the relationship between ADHD PREV and SI for 2003 nor 2007 (all $p \leq .003$).

To test whether the above findings were moderated by seasonal fluctuations in light intensity, the above analyses were repeated with SI data per 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 $p<0.004$; $-0.415 < r < -0.661$), suggesting that differences in monthly SI did not influence the association SI with PREV.

When we repeated the above analysis excluding states with a SI>5.5 (Arizona, New Mexico, Nevada, California, Utah and Colorado, representing 22% of the total US population based on the 2010 US Census data) from the analysis, the partial correlation between SI and PREV weakened but remained significant for 2003 ($p=0.041$; $r=-0.324$; $DF=38$) and marginally significant for 2007 ($p=0.082$; $r=-0.278$; $DF=38$), 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 ($p<0.000$; $r=-0.672$; $DF=49$ | $p<0.000$; $r=-0.571$; $DF=49$) and between SI and elevation ($p<0.000$; $r=0.644$; $DF=49$). However, partial correlations between PREV and elevation were no longer significant when adding SI as a covariate ($p>0.4$), whereas adding elevation as a covariate still allowed marginally significant and significant associations between PREV and SI for 2003 and 2007 respectively ($p=0.076$ | $p=0.007$).

Prevalence rates were also available for ASD (14 US States, 2008) and MDD (44 US States, 2006-2008) from the CDC. The above analyses were repeated for ASD and MDD as well, and did not result in any significant associations between PREV and SI ($p>0.913$).

Non-US adult ADHD prevalence rates, cross-national data: Fayyad et al. (26)

In a multi-national study by Fayyad et al. (26) 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 using a diagnostic interview schedule based on the DSM-IV 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% (26).

These authors reported a significantly lower PREV for Spain, Lebanon, Colombia and Mexico and a significantly higher PREV for France as compared to Italy, Germany, Belgium, the Netherlands, and the US. Socio-demographic 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 ($1.5<OR<3.0$) and these factors did not differ between countries. The four countries with lower prevalence also have the highest solar intensity compared to the other countries, as can be seen in figure 3 below. In this study we excluded the results from the US since those have been analyzed above in more detail.

A sigmoid dose-response was observed for the relation between PREV and SI ($p=0.0073$; $R^2=0.85$; $F=15.83$; $DF=1,6$). A log transformation of PREV and SI resulted in a linear relationship and a significant correlation between PREV and SI ($p=0.018$; $r=-0.758$; $DF=9$) and no association with latitude ($p=0.111$; $r=0.567$; $DF=9$) and altitude ($p=0.136$; $r=-0.537$; $DF=9$), suggesting that SI explained 57% of the variance in the prevalence of non-US adult ADHD. This is a small sample but essentially replicates the pattern that was also observed for the US.

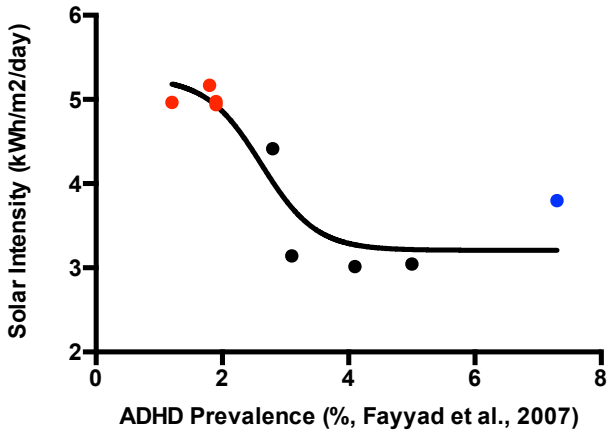

Figure 3. Association between ADHD prevalence rates and solar intensity for non-US countries. *The relation between solar intensity and the prevalence of adult ADHD in several non-US countries and the sigmoid dose-response trend, which best explained the data. Red depicts the four countries (two points 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. (26)*

Discussion

The data as presented here demonstrate a clear relationship between SI and the prevalence of ADHD, where SI explained 34-41% of the variance in ADHD diagnosis in children from the US and 57% of the variance in adult ADHD prevalence in non-US 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 appears 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 since 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 health care availability and accessibility. The US results were mainly driven by the 6 US states with SI> 5.5; these states represent 22% of the US population, so it would not be reasonable to disregard the findings from these states as 'outliers'.

Limitations of these results consist of the fact that both the CDC data and the Fayyad et al. (26) study employed screening methods not identical to diagnostic procedures as carried out in standard care. Furthermore, in both studies attrition rates were between 32-63%, and it is unclear how this would affect the ADHD PREV estimates. However, given the large number of respondents in both studies (CDC: 73,123 families; Fayyad: 11,422 respondents) and the fact that the results were similar in regard to the ADHD and SI relationship, it is reasonable to accept these estimates as accurate.

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 a recent large study that found no association between vitamin D and behavioral problems in children (27); 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 appear to be the prime explanation. On the other hand, around 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, (28,29) shorter sleep duration (29) and melatonin suppression, (30,31). Technological advances have increased the screens, pixels, and light intensity of modern media substantailly across the last 10-15 years and the closer proximity of mobile devices to the eyes as compared to television sets results into exposure to increased light intensity, particularly within the 464-484 nm spectrum. This wave length 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 to 10 years ago. In addition a recent meta-analysis reported that the EEG Theta/Beta ratio (an EEG marker often found in ADHD) linearily 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 may result in supressed evening melatonin levels, delayed circadian phase with associated delayed sleep onset, reduced sleep duration and finally increased Theta EEG power as well as an increased prevalence of ADHD in genetically susceptible children. The apparent preventative effect of high SI on ADHD prevalence may 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). Interestingly, in the CDC data the most significant increase in ADHD PREV between 2003 and 2007 was noted for adolescents 15-17 yrs. old ($P<0.001$) compared to 4-10 yrs ($p=0.013$) and 11-14 yrs. ($p=0.016$) age groups. We would expect that adolescents more often engage in modern media use in the

evening as compared to younger children. These trends could potentially explain the increased prevalence of ADHD over the last 15 years.

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 sub-group of ADHD in a variety of ways: for example, exposing children more to natural light during the day (solar tubes 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 based on 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 (DLMO). These may define intermediate endophenotypes in the etiology of ADHD subgroups from gene X environment interactions.

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Author contributions

MA initiated the manuscript and has been involved with the data collection, data analysis, manuscript writing and had full access to the data and takes responsibility for the integrity of the data and the accurarcy of the dataanalysis. 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 ms.

Disclosures

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MA, KVDH and JLK report no financial conflicts of interest.

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