CORRESPONDENCE

Reply to: Attention-Deficit/Hyperactivity Disorder and Solar Irradiance: A Cloudy Perspective

We thank Hoffmann and colleagues for thought-provoking correspondence and for sharing their data for additional analyses. However, in contrast to their assertion, our analyses were based on solar irradiance (SI) data from only one source (National Renewable Energy Laboratory) for the U.S. attention-deficit/hyperactivity disorder (ADHD) prevalence (PREV) (1); therefore, it is unlikely that our results were affected by SI source; we used only one source within these analyses.

The meta-analytical data from Hoffman reported ADHD PREV between .2% and 26.8%, reflecting a wide range of prevalence rates, possibly due to differing methodology in the numerous sources that distorts true prevalence. We included only data from sources that used standardized assessment of ADHD prevalence, allowing valid and reliable comparisons. Although Hoffmann used meta-regression to account for moderators linked to ADHD, post hoc correction is less accurate than using standardized procedures. In our (and other) analyses, infant mortality and low birth weight explained 18% to 22% of ADHD PREV variance, independent of SI. Adjustments for these associations were not made in their meta-regression. Furthermore, several reports suggest ADHD PREV might be increasing over time (2); thus, comparing ADHD PREV from 2003 in one study to ADHD PREV from 2007 in another study can introduce increased error variance because of this trend. To check this, we randomly shuffled our ADHD PREV rates for 2003 and 2007 for all US states and reanalyzed the correlation between PREV and SI. For the random sequence the effect dropped markedly to $p = .027$; $r = -.315$, and the percentage explained variance was reduced by half, demonstrating that even with a standardized data set from the same agency, mixing data from two time points affected the effect size considerably. Therefore, given the differences in methods between studies and large range in ADHD PREV (.2%–26.8%), we are not surprised Hoffmann could not replicate our findings.

After careful inspection of Figure 1 from Hoffmann, we noted an interesting cluster of data with low SI (<3 kWh/m^2/day) and low PREV. From our graphs (Figure 1, top panel), it becomes clear that we were only able to investigate the association of PREV with SI values of 3 to 7 kWh/m^2/day. When the Hoffmann data are plotted for only the countries with SI ≥3 kW/m^2/day (with the same y axis, Figure 1, bottom panel), the pattern appears similar to ours, although with more variation in the Hoffmann data.

When we statistically reanalyzed the data kindly shared by Hoffmann and colleagues, restricting our statistical analysis to 3 to 7 kWh/m^2/day (Figure 1B, bottom panel) and using log-transformed data, the correlation between SI and PREV became significant in the expected direction ($p = .040$; $r = -.214$; $df = 68$; one-tailed), whereas for the full data set a nonsignificant linear correlation was found ($p = .285$; $r = .064$; $df = 82$), in agreement

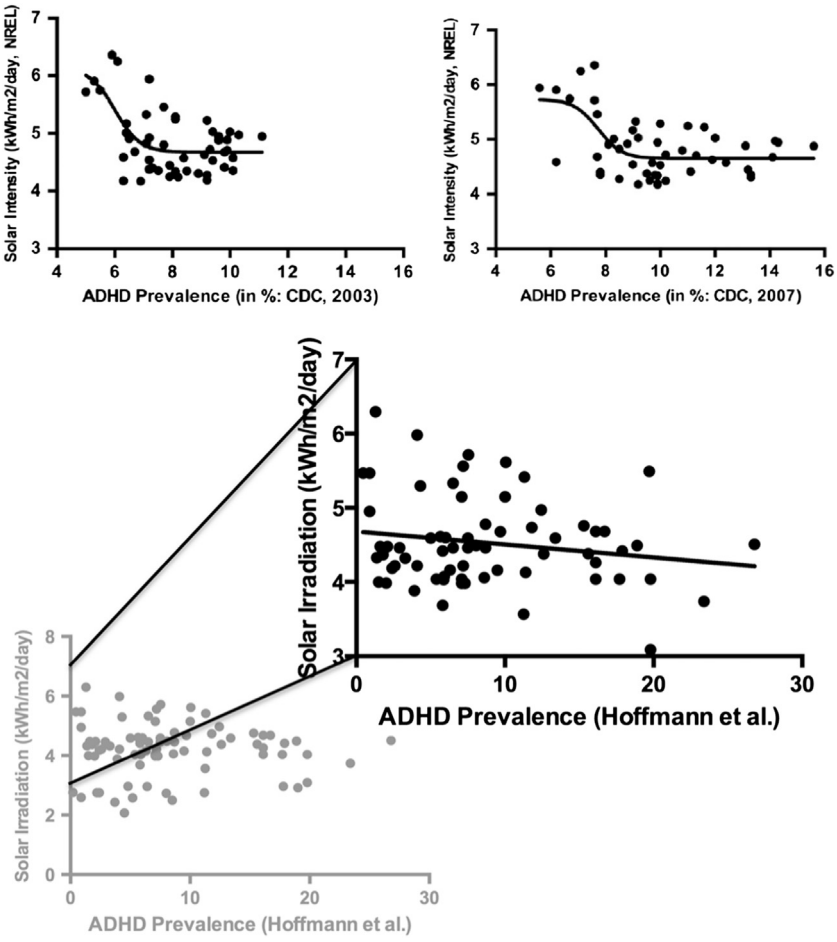

Figure 1. The top graph (A) displays the original figure from Arns *et al.* representing the relationship between attention-deficit/hyperactivity disorder (ADHD) and prevalence (PREV) for 2003 and 2007. Note that here only data with an solar irradiance between 3 and 7 kWh/m^2/day are displayed. The bottom graph displays the data from Hoffmann *et al.* plotted in the same way with the same axis. Note that when looking within the same range, the Hoffmann *et al.* data support the same association as we reported (higher solar irradiance associated with lower ADHD PREV). CDC, Centers for Disease Control; NREL, National Renewable Energy Laboratory.

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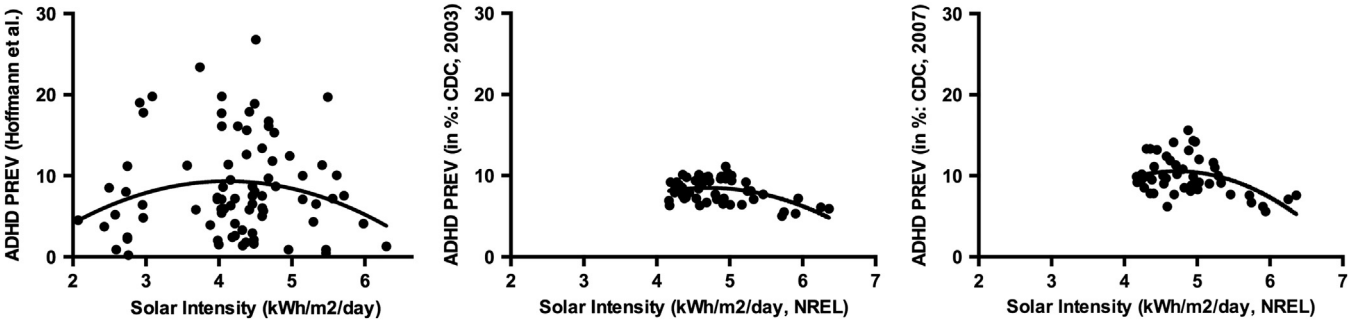

Figure 2. This figure demonstrates the data for Hoffman *et al.* (left) and the Centers for Disease Control (CDC) data from 2003 (middle) and 2007 (right). Note that the quadratic trend better explains the relationship in all three datasets, compared with a linear trend. The parameters from these three models were also not statistically different. ADHD PREV, attention-deficit/hyperactivity disorder prevalence; NREL, National Renewable Energy Laboratory.

with Hoffmann's report. Given the interesting cluster of low-SI (<3 kWh/m^2/day) with low PREV, this association might be better explained by a quadratic relationship, which was confirmed by curve estimation (Prism 6.0); a quadratic trend better explained the Hoffmann data (Akaike's informative criteria [AIC]: 65.23% probability of quadratic) with 4.2% explained variance versus .02% for the linear relationship (Figure 2A). Testing this same comparison on our data from the United States confirmed that the quadratic trend explained the data better, compared with a linear trend (AIC: 2003: 87.37%; 2007: 82.48%), with explained variance 26.8% and 23.1%, respectively (Figure 2B,C) versus 16.8% and 14.0% for the linear trend. These percentages are lower compared with the 36% and 37% reported in our original study using a sigmoidal trend; however, given this new knowledge on low SI areas and the consistency between these data sets, we think this quadratic trend reflects the association between SI and PREV more accurately and possibly generalizes better. Finally, there were no statistically significant differences between the three model parameters of these three models (Hoffmann; Centers for Disease Control 2003 and 2007; AIC: 97.85%), further supporting that the quadratic model best explains the association between SI and ADHD PREV for all three data sets.

This quadratic relationship can be explained in several ways and requires additional study. For example, we hypothesize that the areas with low SI are likely areas such as Scandinavia, Iceland, and Scotland/United Kingdom (confirmed by the authors; personal communication with Giovanni Salum, September 6, 2013), where fish consumption is higher compared with other regions (Earthtrend database, World Resources Institute, Washington, DC; Faostat, Food and Agriculture Organization of the United Nations: http://www.grida.no/graphicslib/detail/fish-protein-world-consumption_c33d). Because free fatty acid supplementation (e.g., omega-3) has clinical benefit in ADHD (3,4), higher fish consumption in these areas could have a preventative effect, partly explaining lower ADHD PREV in these areas. Furthermore, in these areas, SI may be of such a low level that the amount of artificial lighting is much higher than the amount of natural lighting, making SI measures an unreliable proxy measure of blue-light exposure.

Finally, people in these areas may have genetically adapted to these low-SI conditions, making them more resilient to the effects of persistent low levels of SI. This has been demonstrated for seasonal affective disorder, with people from Icelandic descent exhibited lower rates of that disorder (5). Furthermore, the reported trend that children sleep more than 1 hour less compared with 100 years ago was not found or was even reversed for Scandinavia and the United Kindom (6), further reflective of the fact that people in these regions are less susceptible to the disruptive effects on the circadian system of evening exposure to blue light and subsequent reduced sleep duration (7). The dopamine receptor type 4 deserves additional attention. It has genetic variants (9) that have interesting circadian properties (8). Some variants increase risk for ADHD and have a well-known geographic distribution (10,11). These factors may interact and affect the relationship between SI and ADHD PREV.

In summary, on closer inspection, the data from Hoffmann and colleagues do not refute the relationship between SI and ADHD prevalence for areas with SI larger than 3 kWh/m^2/day, which probably applies to most areas in the world, except for Nordic areas (Scandinavia, Iceland) and the United Kingdom. The relationship between SI and ADHD prevalence might be better explained by a quadratic trend, and future research should investigate the explanations behind the low ADHD prevalence rates in low-SI areas, with possible candidates being higher fish consumption and evolutionary/genetic differences, including the DRD4 7R allele.

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