ADRB1: The Gene Responsible for Shorter Sleep Patterns

The Johnson family has several members who sleep significantly less than the recommended eight hours per night. After looking into the genome of such family members, scientists were able to detect a shared mutation of the ADRB1 gene. Upon viewing the behavior of the gene when engineered into mice, they observed that the afflicted mice also required less sleep (about an hour's worth) when compared to their standard mice counterparts.

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Background

In their paper "A Rare Mutation of β1-Adrenergic Receptor Affects Sleep/Wake Behaviors" the researchers attempt to answer the question of whether or not (and how) a specific mutation of the β_1AR (ADRB1) gene could be responsible for the unusually short sleep patterns exhibited by the Johnson family (Shi et all., 2019). The researchers explain that such a finding would be greatly beneficial since little is known about the β-receptors this gene affects and any new information could prove useful to further understanding human sleep duration/regulation.

It would seem the researchers hypothesized that this gene would be a contributing factor to the shorter sleep patterns as they identified it as a common trait amongst afflicted members of the Johnson family. See **Figure 1** for more details. Another key piece of their reasoning was the clinical use of β-blockers. These drugs are commonly associated with difficulty falling asleep and would suggest that if the mutation of the ADRB1 gene was able to do the opposite, then it would result in better quality sleep and possibly shorter sleep duration (Shi et all., 2019).

Logic of Study

In order to better understand this genetic mutation, researchers engineered it into a population of mice they would study for generations. To observe if the mutation made a difference in sleep duration, they compared the average sleep patterns of mice with the mutated gene to that of ordinary mice without the mutation. The total time asleep, in REM sleep, in non-REM sleep, and in the movement were all recorded. These results were compared in three phases: over the whole 24 hour day, in the dark period of the 24 hours, and in the light period of the 24 hours. Each of the four cases was graphed across the three different phases. The scientists also performed other experiments on how the genetic mutation affects brain chemistry but these are beyond the scope of this analysis and can be found in the original paper.

If the research team's hypothesis was correct then they would expect to see mice with the mutated gene sleeping less in all cases and also exhibiting a higher amount of movement throughout the day compared to their unaffiliated counterparts. This would mean a lower bar compared to the normal mice on the graph. In contrast, if the team's hypothesis is incorrect, they would expect to see no noticeable

difference in the two populations of mice. Graphically, this would mean the two bars on each and every graph would be nearly identical.

Results

Figure 2 depicts the results and shows that during each 24 hour and dark phase, the afflicted mice sleep noticeably less (lower bar) and move noticeably more (higher bar) than those unafflicted. However, each graph depicting time asleep in the light phase shows very little difference in the two populations of mice (bars are, or are close to, the same height).

Discussion

The results depicted in **Figure 2** support the researchers' hypothesis and suggest the mutated gene does play a role in the shorter sleep patterns of the afflicted mice. Their average time asleep decreased by 55 min. However, the data also suggest that the mutation only causes the mice to sleep for shorter periods of time during the dark phase of the day. Furthermore, the data does not directly correlate to the two-hour difference experienced in the Johnson family and raises the question of how the severity of this gene varies across species.

Secondary Critique

The New York Times article "Can Genetics Explain Why Some People Thrive on Less Sleep?" by Francie Diep explains the case of the Johnson family and describes the study performed (Diep, 2019). The article does an acceptable job depicting the study at a comprehensive level as it explains that the scientists used mice and studied the ADRB1 gene specifically. Diep also explains how the presence of the mutated gene correlated with roughly an hour less sleep for the afflicted mice. These are indeed the key points of the study, however, the article fails to mention the differences between light and dark periods which is central to the expression of the mutation, and thus if why the author's work is only considered acceptable. Overall, Diep's piece was a great introduction to the topic but curious readers should read the original paper for extra details and a more nuanced understanding.

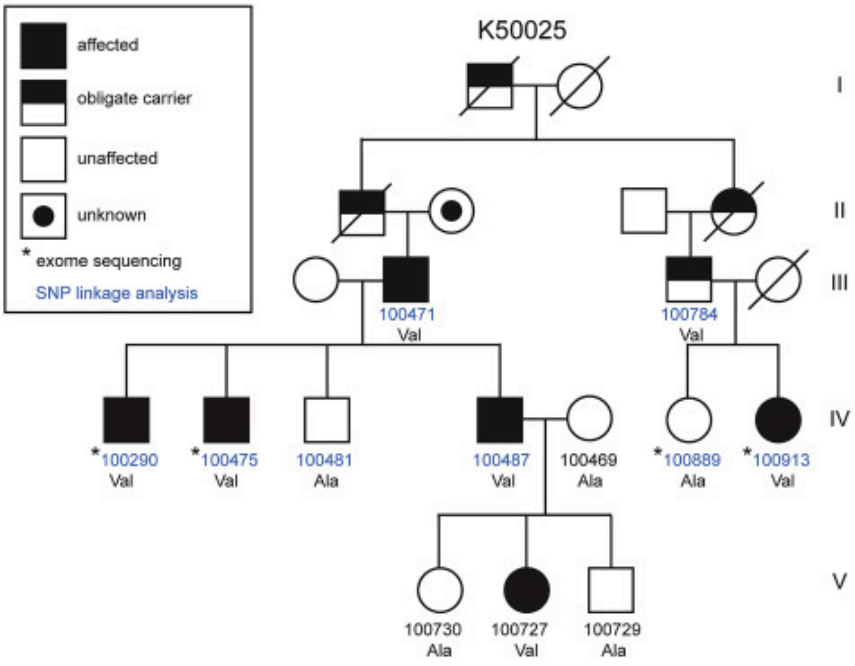

Figure 1: Family Tree with the ADRB1 Mutation

(Shi et al., 2019)

This is a family tree of a human family with the same mutation as the Johnson family. This figure was used in the study to show that the allele was dominant. The “Val” and “Ala” underneath individual members from the 3rd-5th generations correspond to the presence of a specific amino acid in one of the proteins impacted by the gene and are abbreviations for alanine and valine respectively.

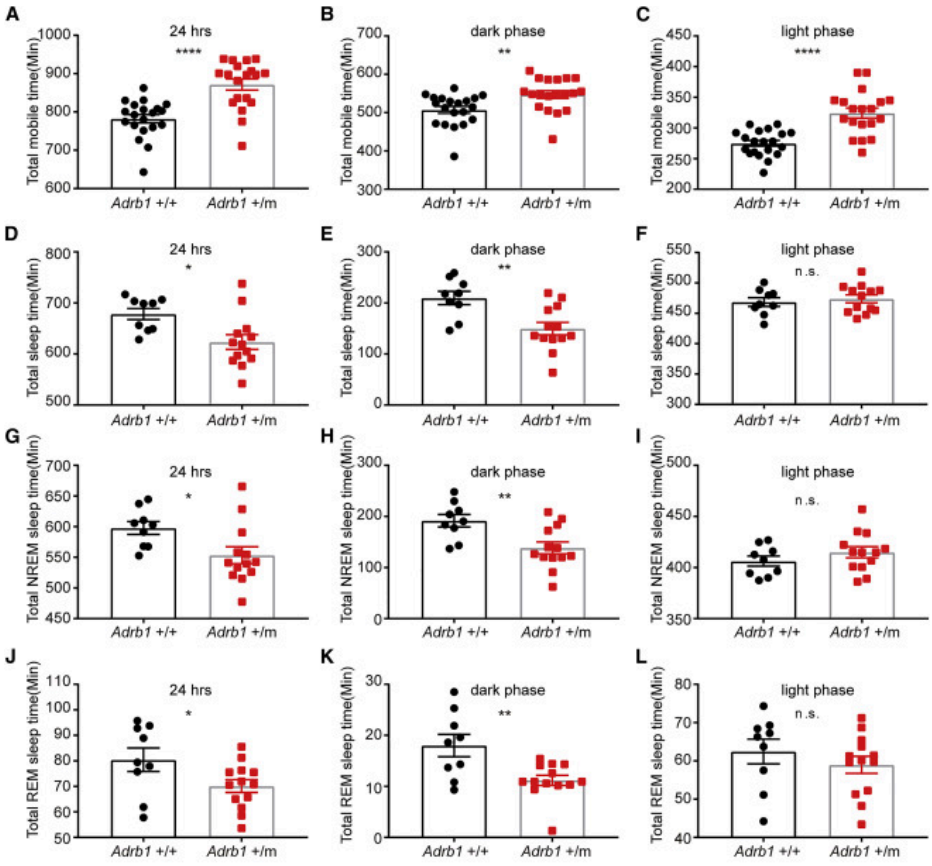

Figure 2: Results comparing sleep and movement durations across both mice populations (Shi et all., 2019)

Rows show the different types of behavior of the mice and the columns show the different phases where the data was recorded. The black Adrb1 +/+ are the control mice without the mutation and the red Adrb1 +1/m are the mice with the genetic mutation.

Literature Cited

Guangsen Shi, Lijuan Xing, et al. 2019. A Rare Mutation of β1-Adrenergic Receptor Affects Sleep/Wake Behaviors. Neuron 103: 1044-1055. DOI: 10.1016/j.neuron.2019.07.026

Francie Diep. 2019. Can Genetics Explain Why Some People Thrive on Less Sleep?. The New York Times. From https://www.nytimes.com/2019/08/30/science/sleep-gene.html