An Overview of Predicting the Prevalence of ADHD

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ABSTRACT

The prevalence of attention-deficit/hyperactivity disorder (ADHD) has been increasing these days, and this phenomenon has raised the interest and attention of both society and the academy. While ADHD is believed to be one of the most common mental disorders in children, it may be essential to track back how to prevent such a disease to avoid further deficits in children's cognitive functions affected by the early prevalence of ADHD. This paper reviews the possible causes of ADHD based on previous research. The results show that the dimension of causes could be split into three types: neutral or biological-related causes, natural and behavioral causes, and cognitive function deficit. With all these mentioned factors contributing to the prevalence of ADHD, and with a deeper knowledge of ADHD, the prediction of ADHD could be more high-efficient.

Keywords: *Attention-deficit/hyperactivity disorder, natural causes, neutral causes, cognitive deficit, statistical trend.*

1. INTRODUCTION

Attention-deficit/hyperactivity disorder (ADHD) has become one of the most common disorders among children across the world, while the diagnosis onset, the accurate causes, and other basic information are still under debate these days. ADHD is categorized by the deficit of attention or manner in the impulsivity of hyper-activity or disorganization, while children who are diagnosed with ADHD may even have risks of other learning disorders [1]. As observed by BlueCross BlueShield [2], a health organization in America, the diagnosis rate of ADHD has increased by more than 30% over the past eight years in the US, and the current prevalence is 10% in the US. Compared to children who were not diagnosed with ADHD, children with ADHD would have lower educational attainment, lower income, lower employment, and lower self-esteem [3]. This indicated an urgent need for deeper study or a significant response to the improvement of the diagnosis of ADHD. The diagnosis of ADHD is difficult to administrate like the other mental disorders, compared to physical disorders, since there may be many other neutral or natural factors impacting the prevalence or diagnosis of ADHD [4].

An overview of predicting ADHD has been urgent since the impact such a disease would have on children would be lifelong. An early prevalence would be more beneficial for later ADHD recovery and would also reduce the further influence on other executive functions. As an adequate database concerning the possible causes of ADHD, the overview could be an appropriate layout of how to predict the ADHD, while the statistical trend concerning how the ADHD would happen based on common symptoms and disease onset would also be a strong predictor rooted in the studied regions.

To conduct such an overview, a clearer predictor or method to cue ADHD could be considered. Rather than learning the causality, the appearance or trends would be more efficient to predict. In this case, the stress from family, society, and the hospital could all be released to some degree, while the children being diagnosed with ADHD could also benefit from such progress. To conclude, a smaller effort could be made to earn a more accurate diagnosis and prevent ADHD.

2. POSSIBLE NATURAL CAUSES FOR ADHD

The possible causes of ADHD have long been studied, and many assumptions have been made both on the natural or genetic side and the neutral or experienced side. A large database and hypothesis have been concluded, presenting a large blue picture of the possible causality. Meanwhile, research into the origin and further research into ADHD could be conducted based on such findings.

2.1. Genetic factors

Tannock [5] conducted a review study demonstrating the genetic factor affected the prevalence of ADHD, and there has been much evidence through family studies, and twin and adoptive studies showing that genetics may be essential in the prevalence of ADHD. However, although some of the studies indeed showed a higher rate of prevalence of ADHD across the first generation compared to the second [6], some other studies came up with findings presenting monozygotic twins with the same heritable genetic information shared a higher risk of diagnosis compared to dizygotic twins [7]. While genetic factors can predict 60 to 90 percent of the prevalence of ADHD through twin and adoption studies, genetic factors cannot be proven as the sole cause of ADHD [8].

2.2. Family study

The methodology of the family study could not rule out the environmental influence on the prevalence of ADHD completely, and although there may be a strong database of how ADHD may pass through a family and be compared through generations, the causality remained uncertain. Before the twin and adoptive study analyses, the twins shared a similar cognitive level and personality, but it is difficult to determine whether this similarity was due to genetic or environmental influences [9].

While other researchers focused on the methodology of segregation analysis, which is a mathematical approach to illness transmission across the family to determine the genetic or heritable information, and molecular genetic study utilized in the family study. Faraone and Biederman mentioned the possibility of multi-gene inheritance, while the single-gene study also came up with some correlation with the familial transmission of ADHD in a study from 2000 [3]. One of the first genes implicated in ADHD published was the dopamine transporter gene (DAT1), and it was soon replicated, receiving results from both positive and negative sides [10]. Because of the complex symptoms of ADHD, the identical specific candidate gene for the overall symptoms or the general prevalence has not yet been found. The single gene may explain only limited symptoms.

Researchers assumed that the reason why it was difficult to investigate the specific gene, may be either several genes were contributing to a similar symptom, or the relating genes may have lower rates of expression, and not all individuals inheriting this gene developed the symptoms [11]. A meta-analysis from Gizer and colleagues in 2009 demonstrated the association between CHRNA4 and SNAP25, while the candidate gene polymorphisms were emphasized [11]. Other genes such as the dopamine D1 receptor gene (DRD1) were also highlighted in the preliminary single-nucleotide polymorphisms research or meta-analysis [12].

The genetic factor that would influence the prevalence of ADHD is still under debate. The question of whether a direct and distinct influence from single- or poly-genetic causes resulted in the prevalence remains open. Further studies are still planned.

2.3. Neurotransmitters

Dopamine is stated as the primary neurotransmitter implicated in ADHD. Excessive dopaminergic activity may result in poor motor impulse control and the worse control of impulsivity has become one of the main symptoms of ADHD [13]. The excess dopaminergic activity was related to impulse control or action dysfunction. This finding has been supported by a PET study by Ernst and colleagues in 1999. Rather than dopamine, another kind of neurotransmitter, norepinephrine, has also been identified as an essential mark for ADHD. Norepinephrine has also been linked to ADHD-related cognitive symptoms such as inhibitory control, and dopamine and norepinephrine-related drugs have been used to treat or suppress ADHD [14].

2.4. Oxidative biomarkers

The meta-analysis confirmed that the level of oxidative biomarkers in blood, urine, and saliva was observed to be higher than normal in children and adult ADHD patients [15]. However, the situation still differs in some cases. For example, MDA, which is also a significant oxidative biomarker, was found to be less than controls in children with ADHD patients [16]. A possible explanation is that what is associated with ADHD prevalence is the balance of oxidative stress and antioxidant activity. For the ADHD patients, their antioxidant response is insufficient to overcome oxidative stress, and this may be one of the reasons why such biomarkers trends presenting in patients' bodies. Rather than identifying the level of oxidative biomarkers as a distinct feature of ADHD, the ratio of antioxidant response to oxidative stress shows a more accurate association for ADHD patients [17]. Yet, the causality was not built.

3. POSSIBLE NEUTRAL CAUSES FOR ADHD

Besides the physical characteristics such as genetic factors, dopamine, and norepinephrine that may affect the prevalence of ADHD, scientists also conducted other possibilities of environmental or behavioral impact.

3.1. Maternal behavior

Maternal behavior, such as prenatal smoking, may be closely related to the prevalence of children's ADHD. Milberger and colleagues concluded a positive-correlated higher risk for children to develop ADHD if exposed to

prenatal smoking as infants [18]. In the context of a study on schizophrenia, the intake of nicotine would affect the number of nicotinic receptors, influencing the dopaminergic system, which was a vital mechanism in the prevalence of ADHD.

3.2. Unhealthy diet

An unhealthy diet may contribute to the prevalence of ADHD. It was thought that eating Western food high in fat and sugar in early childhood was linked to a higher risk of ADHD diagnosis in late childhood or adolescence [19]. It can be assumed that insufficient nutrition uptake would influence brain development and result in a higher risk of a diagnosis of ADHD. For example, Omega-3, which is essential nutrition for the body, was discovered to be lower in ADHD patients [20]. Meanwhile, eating Western food was also related to worse behavior or habits found in children and adolescence, and eating green leafy vegetables more was associated with a higher score on the Child Behaviour Checklist [21]. In this case, it can be assumed that it was both the unhealthy diet and poor daily behavior and habits that contributed to the prevalence of ADHD, or there may be a mediator exit. The conclusion remains unknown.

3.3. Sleeping deficit

While sleep deprivation is a common symptom of ADHD, with issues in the dimensions of sleeping onset, frequently awakened, and sleep-disturbance breathing, sleep disturbance may be one of the reasons why ADHD was raised. Although the detailed causality relation was not determined yet, there were four scenarios for the relationship between ADHD and sleep disturbance discussed by Hvolby in his review paper [22]. The first was that it was the hyperactive behavior indicating the sleep disturbance, or sleep disturbance was the consequence of ADHD. Another was that sleep disturbance contributes to ADHD daytime symptoms. Some researchers believed that if that were the case, in the treatment of ADHD, doctors could choose to regulate patients' sleeping breath to eliminate sleeping-related symptoms. The third scenario was that there was a bidirectional relationship between sleep disturbance and ADHD. While lack of sleep can have an impact on emotional regulation and attentional functioning, ADHD hyperactivity may result in worse sleeping quality. Finally, there may be an overlap between ADHD and sleep disturbance in neurophysiological mechanisms. The four scenarios raised here could be applied and considered to assist treatment of ADHD, while further studies are looking forward to seeing the actual causality.

4. OTHER COGNITIVE FUNCTIONS

Some researchers believe that the main deficits of ADHD mainly fall into two dimensions: cognition and behavior, while cognition includes functions such as memory, language, and planning [23]. Children diagnosed with ADHD may have impaired executive function and will under-develop their overall functions.

For the study carried out by Tsal and colleagues, after gathering the questionnaire concerning ADHD from the children patients' families and eliminating those who had a severe intelligence deficit, the ADHD patients and the corresponding control group were chosen to take the four cognitive tasks [24] Tsal. Before the tasks, the practice tasks were initially assigned to both groups to ensure the instruction was well understood by both groups of child participants. The target was marked white against a black background to highlight the main visual focuses and draw out other potentially distracting elements while measuring executive cognitive functions such as selective attention, executive attention sustained attention, and orienting of attention. For the task of selective attention, participants would be shown a pair of targets combined with different colors and shapes. The participants were required to focus on a specific target through instruction, and what they should do was to drive their attention to the required suggested target. For the executive attention test, the participants would be asked to attend a Stroop-like test. To keep the complexity at a similar level, participants would be shown two arrows orienting up or down and presented on the upper or bottom of the screen. In some trials, participants were required to answer the direction of the arrows directly, while in other trials, participants would answer the position of the target arrow in the corresponding area on the screen. For sustained attention, participants would be presented with an image formed with the color blocks, coming up with 16 possible animals such as dogs, horses, and elephants. The participants were required to focus on the center of the screen where the image would appear, and they should report what animal the color block formed as fast as possible. For the orienting of attention task, after fixating on the center of the screen, there would be a cue presented and the participants should answer with the accurate orientation where the target would occur. The cue would indicate whether it is valid (75%) or invalid (25%). For the general result, the ADHD patients performed a significant deficit on the selective attention tasks, the executive attention tasks, the sustained attention tasks, and the orienting of attention tasks, characterized by both a lower accuracy rate and a longer reaction time. The results showed a strong correlation between the prevalence of ADHD and four cognitive functions. With the test result or the general regularity found in the study, the cognitive test result may be a prediction of the prevalence of ADHD.

Another study conducted by Weigard and colleagues demonstrated the contribution of memory tests to the prevalence of ADHD [25]. In this study, after assigning the ADHD children's group and the control group, the demographic data was collected. Children would be

tested by assuming whether the number of asterisks presented on the screen was greater than 50 or lower and responding using mouse clicks. For some of the trials, the answer may be obvious, such as the fact that there were approximately 30 or 70 asterisks in total. Feedback would be delivered right after the answer was reported by the participants. The participants were announced to respond as accurately and quickly as possible. The experimental data were collected, analyzed, and fitted into the linear ballistic accumulator model (LBA), which is a cognitive model trying to form a linear and smooth relationship between two factors. The results indicated a significant finding that the application of formal experience or testing results concerning ADHD-related cognitive tests could improve the operationalizing and testing of the predictions from the etiological theories of ADHD. The evidence also supported the neuroscientific test account for the cognitive deficit in ADHD.

5. DISCUSSION

The identical causes of ADHD remained uncertain. It is possible that with a more confident conclusion of causes, individuals and families would benefit from using strategies to prevent ADHD from reflecting on the causes. It may be too difficult to figure out the significant causes of ADHD since the cause may be a mixture of plenty of possible reasons. It would be much more efficient to conclude the trend of symptoms with results coming from the huge database. The studies shown above both suggested the potential contribution of cognitive tests and former experience or data that could support the prediction of the prevalence of ADHD, and even provided evidence for neuroscientific tests for characteristic features of ADHD patients.

Some other factors may influence the future improvement of children's ADHD rather than simply studying the possible causes and trying to avoid the nonadaptive behavior. For example, the earlier the age at onset is, the better and more efficient medical intervention could be in improving the children's ADHD [26]. Often, the first symptoms of ADHD are reported by the parents or primary caregivers of potential ADHD patients. If they could recognize the symptoms earlier, it can be assumed that doctors and psychiatrists could provide appropriate therapy or medical assistance and improve the later healing from ADHD. Also, children's ADHD may accompany other neurodevelopmental disorders, with an earlier onset and intervention. Other risks may be prevented at an early stage and relieve some stress for both the children and their families.

However, the possible causes of ADHD have remained uncertain for several years. A more efficient approach to preventing or predicting ADHD would be to look at the statistical trend of each region. This paper would present a glimpse of possible causes of ADHD and recruit the statistical trend concerning ADHD that could predict the prevalence of ADHD. Rather than tracking back the exact causes of ADHD, the statistical trend of the database could contribute more to predicting ADHD.

6. CONCLUSION

Drawing from what has been reviewed, genetics, biological transmitters, sleeping deficit, unhealthy diet, and maternal behavior could all be the potential causes of the prevalence of ADHD, though the perfect or exact causality remains uncertain and all the relations mentioned here were correlations rather than causes. Although some factors passed the manipulation experimental design, the later follow-up experiment would still defeat such a conclusion. To come up with a more accurate and efficient approach to predicting the prevalence of ADHD, it would be a better choice to focus on the statistics analyzed from the interested regions.

This paper is not free of limitations. The literature reviewed here was limited, and more data was looking forward to being added. Also, the exact approach for the family to observe and assist them in predicting the potential ADHD children was not concluded here. This paper aimed to improve the prediction of ADHD and try to release the stress for both society and the family. In this case, a more public standard concerning the prevalence of ADHD is still in a significant debate space. Further study could concentrate on how to form a more public way to predict the prevalence of ADHD and then improve the situation and release the stress caused by ADHD.

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