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Recent Increase in Autism and ADHD: True or Inflated?

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The prevalence of autism spectrum disorder (ASD) has increased markedly over the past 40 yr (1). It was reported to be 4-10/10,000 by the early 1990s, but increased to 20-116/10,000 after the year 2000, and was even up to 1/88 in recent studies (1, 2). The prevalence of ASD in Korea was reported to be as strikingly high as 2.64% (3). The change in the prevalence of attention deficit hyperactivity disorder (ADHD) is not as dramatic as that of ASD, but the overall trend has been similar over the past 10 yr. A survey from the United States reported that ADHD increased from 7.8% to 9.5% between 2003 and 2007 (4). There are several different interpretations as to how this change occurred within only decades.

The first possible influential factors are *diagnostic substitution* and *changes in the definitions of the diseases*. In particular, some studies have suggested that diagnostic substitution (diagnosing as one condition at one time and subsequently as another condition at another time) has played an important role in the increasing prevalence of autism over time. Diagnostic substitution is closely related to *changes in the diagnostic system*, particularly of the broadened concept of ASD. Autism was first narrowly defined by Leo Kanner in 1943, but after the 1980s, the diagnostic system started to acknowledge milder phenotypes. The most dramatic change was listing Asperger's disorder (social impairment without language delay) and pervasive developmental disorder not otherwise specified (PDD NOS; individual who fails to meet age criteria or does not display all the key elements of autism or other subtypes of PDDs) in the DSM-IV (1994).

Regarding ADHD, diagnostic substitution has not played as large a role as in ASD because the diagnostic criteria have largely remained unchanged since 1980. However, possible confounders may contribute to the variable prevalence of ADHD. The most problematic notions of the DSM-IV criteria for ADHD are *functional impairment and dysfunction evident in at least 2 different settings*. As the degree and range are poorly defined and are dependent on the raters, the criteria may allow room for a subjective judgment of diagnosis.

The second potentially influential factors are *the methods of case identification and source of data*; these factors raise one of the most essential questions about whether the increased prevalence reflects a true upsurge in incidence or an increased discovery rate. In particular, when the cases are identified from a pre-registered database, such as national registers, the prevalence can be significantly biased by accessibility to healthcare and education services, the attitude of parents toward mental health problems, and the clinician's experience and level of expertise.

As both ASD and ADHD are diagnosed based on behavioral profiles reported from multiple data sources and direct observation, factors affecting prevalence may be present even in population-based epidemiological studies. Typically, first-line screening depends on the checklists completed by parents, teachers, or both. Therefore, the positive screening rate might be influenced by the sensitivity of the screening instruments, the level of the raters' understanding of the questionnaire, and their pre-existing concerns regarding the disease screened. Also, the participation rate for the screening and confirmation process can be a major issue (nonresponse bias). Diagnoses are also influenced by the instruments used for the diagnostic confirmation. Thus, epidemiological studies should be cautiously interpreted as to whether these potential confounders have been carefully controlled, particularly when the reported prevalence is extremely high.

The third and most controversial element in the increased prevalence is the *environmental contribution* to the incidence of ASD and ADHD. Environmental influences can be accounted for by gene-environmental interactions or epigenetic modification of risk gene expression. Suggested factors for ASD are mainly biological, such as intrauterine infection, advancing maternal age, air pollution, organophosphates, and heavy metals (5, 6). The relationship between the rapid increase in autism and MMR vaccinations was once debated very intensely; however, large-scale epidemiological studies have clearly disproven the possible relationship between those factors (7). The environmental risk factors for ADHD include prenatal substance exposure, heavy metals, maternal folate levels, obesity, food additives, early traumatic events, and maternal stress (8). Compared to ASD, there might be a higher possibility that recent environmental changes have contributed to the increase in ADHD. However, it has yet to be confirmed whether there is a direct, quantita-

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tive correlation between these factors and increased prevalence.

Although the increase in ASD and ADHD is verging on a social issue as well as a public health concern, the underlying etiology is likely a multi-factorial one, much more complicated than the phenomenon itself. Prospective studies with identical protocols are needed for measuring genuine incidence. In the long run, the establishment of biological markers of ASD and ADHD could provide clearer answers by clarifying debates over the stability of the diagnostic system and its reliability.

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