

# WHERE DOES ADHD COME FROM?

JOEL NIGG, PhD

**B**EHAVIORAL GENETIC (such as twin studies) and molecular genetic studies have made it clear that genetic influences play an outsized role in explaining why some people have more ADHD-related traits than other people do. Yet, large epidemiological studies have made it equally clear that several environmental exposures are also associated with ADHD. For example, ADHD is robustly associated with certain maternal health problems during pregnancy, as well as with low birth weight. It also has associations with exposure in early life to various pollutants such as lead (with still-debated associations with several other pollutants). Though less robust, evidence is accumulating that in some cases, dietary factors may also play a role. How do we understand the involvement of genes and the involvement of environmental factors together?

One straightforward way is to think of the genetic profile as a liability—it conveys a tendency or propensity for ADHD to emerge. However, the experiences of an individual when they are developing will ultimately determine if a risk for ADHD ultimately materializes into the full disorder. Thus, exposure to environmental risk factors affects some children more than others. This is a common-sense notion when we apply it to everyday risks like the common cold (or more immediately, infectious disease like COVID-19). Not everyone exposed to risk factors gets ill, and even when they do, not all get ill to the same degree. The reasons include other health factors and other exposures, but also the makeup of a person's genes. It's a real possibility that something analogous happens with ADHD.

We can think of the genetic and environmental inputs as cumulative—in the presence of a very strong genetic tendency, minimal environmental exposures may be sufficient for ADHD to emerge. Conversely, in the presence of massive environmental disadvantage for the developing nervous system, ADHD may emerge even when genetic tendency is only slight. Multiple environmental dis-

advantages, including small ones that add up over time, may gradually move a child from “typical distraction” or “typical impulsivity” to more extreme challenges with self-control and self-regulation.

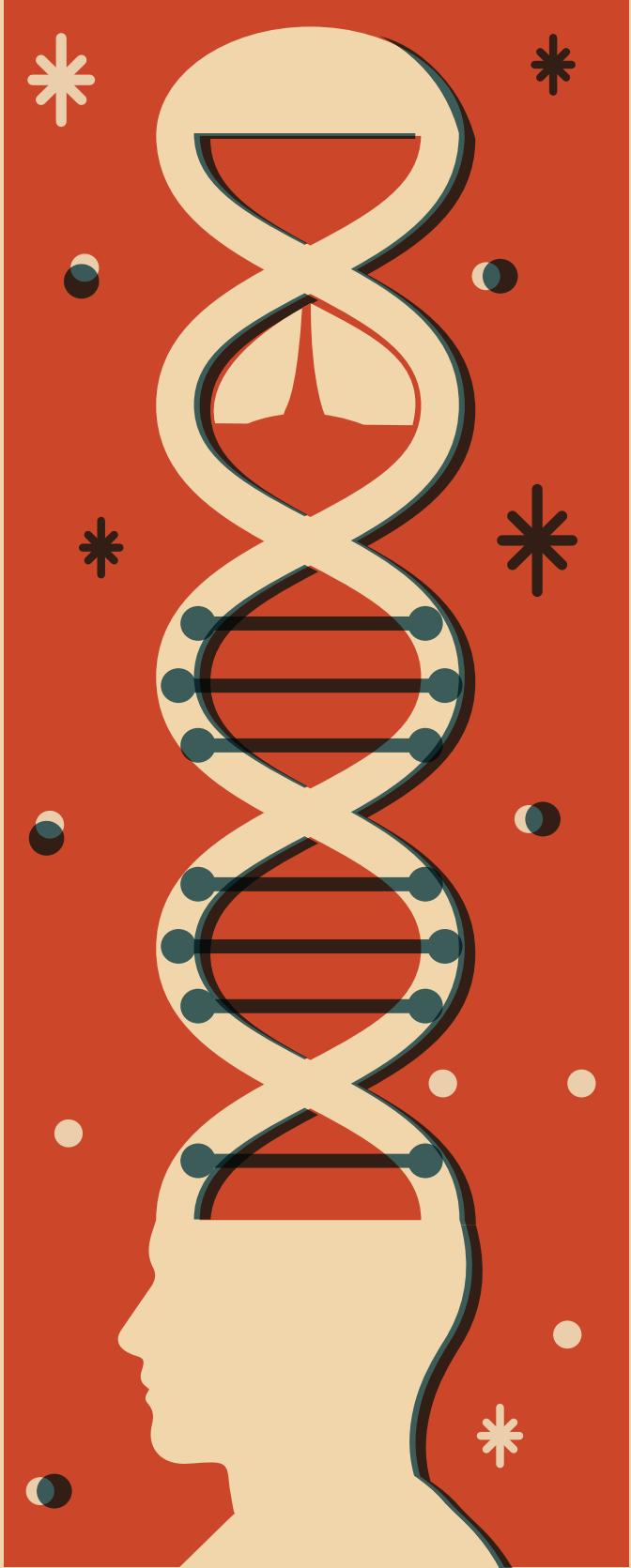
Think of attentional focus or impulse control as bidirectional—too little, or too much, is not adaptive for a given situation. Over-focused or over-controlled individuals may face their own challenges, quite different from those of people prone to be under-focused and under-controlled. People vary along these traits, but at either extreme, a person will experience challenges in their daily life. An accumulation of both environmental disadvantages and genetic risk factors will move individuals along that spectrum—sometimes in a subtle way, other times more markedly.

## Where is ADHD going?

Perhaps a more relevant question, however, is *what influences the path of development for a child with ADHD?* The level of their genetic risk clearly still plays a part—those with higher genetic risk are more likely to have persistent, high levels of symptoms. Sometimes genetic risks are so high that ADHD is inevitable.

Many environmental factors also appear to play a role. These include the degree of emotional conflict in a child's relationships with adults and peers (behavior deteriorates more under high levels of emotionally intense or intensely negative social interactions), subjective feelings of stress (these cause all of us to lose our best self, but the effect is more dramatic for people with ADHD), physical health including diet and exercise and fitness, and the development of compensating strengths and skills.

In this regard, our challenge as a field is to isolate “what works for whom” to identify the precise factors that influence each individual person's expression of ADHD. This is the work of the current and upcoming generation of scientists looking at the developmental path of individuals with ADHD, which can vary widely.



## Scientific challenges

Certain challenges to this view guide scientific inquiry today. One challenge is that genes and environments are not independent of each other—they tend to co-occur. People with higher genetic risk for ADHD also tend to be exposed to more environmental risks for ADHD. Separating the influences is thus a scientific challenge.

Likewise, a correlated risk factor may not be causal. For example, women with ADHD may be more likely to smoke during pregnancy and have offspring with ADHD due to the genes they share with their child. This can create a false impression that smoking caused the ADHD. On the opposite side, genes may influence a parent to behave more inconsistently with a child—disrupting the child from learning how to properly self-regulate, and emerging as a primarily environmental influence on the child. Determining where the causal arrows lie is the key challenge for the current generation of scientific studies.

## What does it mean for me?

What does this mean for you? The answer is complex because many exposures are unavoidable. Although at a personal level it may be troubling to think that something in your child's history contributed to their challenges, for future children it is a sign of hope. Further, as we gain insight into those early causal influences, it becomes increasingly possible to imagine identifying safe ways to protect full health for children with high genetic risks or very high exposure risks. For example, in one study from our lab, we found evidence that a healthier maternal diet protected offspring from the risk of maternal ill health in pregnancy otherwise.

Further, looking forward at the individual level, it is empowering to realize that the path of a child with ADHD is not written in stone. Thus, rather than looking back and wondering what could have been different, we can look ahead and see many opportunities to help a child (or an adult) move in a more fulfilling direction. 



**Joel Nigg, PhD**, is a licensed clinical psychologist and vice chair for psychology, professor of psychiatry, and director of the Center for ADHD Research at Oregon Health & Science University in Portland, Oregon. He obtained a bachelor's degree from Harvard College, a master's in social work from the University of Michigan, and a PhD in clinical psychology from the University of California, Berkeley, with clinical residency at Seattle Children's Hospital. He served on the psychology faculty at Michigan State University for thirteen years before moving to OHSU to launch the ADHD research program in 2008–2009. He is known for his scientific contributions in ADHD, totaling over 200 papers with over 20,000 citations.