Main ADHD research findings

Our research team studies the benefits and risks of ADHD medication using large-scale population-data. We have for the first time shown that ADHD medication reduce the risk for serious and public-health relevant outcomes, including criminality [1] and traffic accidents [2]. Our studies found no evidence for harmful effects of ADHD medication on substance abuse [3]; rather the association between ADHD and substance abuse was explained by shared familial risk factors [4]. Our studies also suggest that the co-occurrence of ADHD and suicidal behavior is due to shared familial factors [5], rather than to harmful effects of ADHD medications [6].

Our research team attempts to map the stability and change in ADHD symptoms from childhood into early adulthood. This far, we have been able to demonstrate that symptoms of inattention tend to persist from childhood into adolescence to a greater extent than symptoms of hyperactivity-impulsivity [7]. We have also shown that the level of the inattention symptoms may increase in some individuals from childhood to adolescence [8], which may lend developmental insight into how the presentation of ADHD symptoms changes from childhood to adulthood. We have used twin data to demonstrate that both stable and dynamic genetic risk factors influence ADHD over the course of the development from childhood into adulthood [7, 9]. We have recently shown that physical activity in adolescence might decrease ADHD symptoms in early adulthood [10]. Our research highlight that ADHD is a developmentally complex phenotype characterized by both continuity and change across the life span.

Our research team is interested in the question of whether ADHD is best viewed as a categorical disorder or as an extreme of a continuous trait. We have used twin data to demonstrate a strong genetic link between the extreme and the sub-threshold variation of ADHD symptoms [11]. We have also shown that the association between reduced birth weight and ADHD symptoms extend beyond the extreme end of the ADHD distribution, including the sub-diagnostic threshold range [12]. Together, this suggest that ADHD is best viewed as the quantitative extreme of genetic and environmental factors operating dimensionally throughout the distribution of ADHD symptoms, indicating that the same etiologic factors are involved in the full range of symptoms of inattention, hyperactivity and impulsivity.

Our research group use twin data to study how genetic and environmental risk factors contribute to ADHD across the life span. Among many things, we have been able to demonstrate that ADHD in adults is strongly influenced by genetic risk factors and that the previous reports of low heritability for ADHD in adults are best explained by rater bias [13-16]. We have recently published a review of the available twin research on ADHD in adults [17].

It is well-established that ADHD frequently co-occurs with other psychiatric conditions. We use twin and family data to obtain a better understanding of why individuals with ADHD also presents with other psychiatric disorders. We have clarified the role of genetic and environmental factors for several comorbid disorders and traits, including asthma [18], externalizing behavior [12], substance use problems [19, 20], autistic traits [21, 22], temperamental traits [23], and emotional lability [24]. We have also conducted a large family study to show that ADHD share genetic risk factors with bipolar disorder and schizophrenia [25].

Our research team uses genetically informative designs to better understand associations between early risk factors and ADHD. We have shown that even though
maternal smoking during pregnancy [26] and maternal obesity [27] in early pregnancy are associated with ADHD in offspring, they are not causally related; rather these associations were explained by unmeasured familial confounding. Using the same methods we have shown that cousins within the extended family and siblings within the same nuclear family who were differentially exposed to family income during early childhood actually differed in ADHD risk, which support a causal interpretation [28]. Sibling and cousin analyses have also revealed that preterm birth [29] and advancing paternal age [30] is strongly associated with offspring risk for ADHD even after controlling for genetic confounding, again consistent with a causal interpretation. We have also explored how ADHD associates with maternal stress during pregnancy [31] and maternal age at childbirth [32].


