

# Autism spectrum disorders in high functioning adolescents

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## Societal relevance

### Relevance

In the Netherlands a growing number of high average intelligent adolescents with ASD is in the need for special secondary education (waiting lists are growing). These adolescents often experience many problems in their everyday life, and executive functioning problems are commonly recognized by parents, caregivers and teachers. Diagnostic interviews, observations, and neuropsychological tests, however, are challenging to apply in these high functioning adolescents with ASD. Because of their ability to masquerade their symptoms, especially in clinical or laboratory settings, it is very difficult to diagnose and inventory their problems (Rump, Giovannelli, Minshew, & Strauss, 2009; Salter, Seigal, Claxton, Lawrence, & Skuse, 2008). Making a good diagnosis is extra important at this age while, although the social difficulties are independent of age and developmental level, the related psychosocial stress may disproportionally increase during adolescence (Nicpon, Doobay, & Assouline, 2010; E. Pellicano, 2010b). This is especially noticeable in high-functioning adolescents with ASD, as they more than their low-functioning counterparts, seek and initiate social interaction with peers (Bauminger, Shulman, & Agam, 2003; Hauck, Fein, Waterhouse, & Feinstein, 1995). Despite the need for and known difficulties in diagnosing these high-functioning adolescents, in clinical practice and scientific research little recommendations are offered. This is why in the first part of this thesis, we take a specific interest in investigating the use of diagnostic instruments and the neurocognitive profile of high-average intelligent adolescents with ASD.

In the second part of this thesis, we were interested in gaining more insight in the mechanisms behind this disorder in high-functioning adolescents, as to our opinion this is an essential issue for understanding autism and for designing therapies and treatments. Nowadays it is generally recognized that besides the well-known triad of problems, many people with ASD experience problems in executive functioning (Hill, 2004). Given the central role of executive functions in both higher and lower cognitive processes, problems in these functions could provide a good explanation for the symptoms seen in ASD. As working memory is generally seen as a central process in many, if not all executive functions, we propose that it is highly plausible that working memory plays a more leading role in both the social and non-social symptoms seen in ASD. To date, however, studies have failed to give conclusive evidence about the relationship between working memory functioning and the symptoms seen in high-functioning adolescents with ASD. Therefore, in this thesis, we started to explore the role of working memory functioning in high-functioning adolescents' ASD symptomatology.

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## Main findings

In the first part of this thesis, clinical studies are presented that investigated the use of diagnostic instruments and the neurocognitive profile of high-average intelligent adolescents with ASD. We found that high-functioning adolescents with ASD *do* experience many social cognition and executive functioning difficulties in daily life situations, and that these difficulties cannot always be objectified in a (neuro) psychological assessment. In more structured settings, these intelligent teens are able to apply compensation strategies, although these strategies are often not functional on the level of multisensory information processing that is needed in real life social interaction. In general, this thesis shows that structured (neuro)psychological assessment in high-functioning adolescents with ASD should always be complemented with real life observations and by-proxy reports of significant others, to get a full 'Aha-erlebnis' and as a psychologist be able to give good advice.

The second part of this thesis described research into the mechanisms underlying ASD in high-functioning adolescents. We examined the working memory brain network integrity in high-functioning adolescents with ASD, investigated the neurodynamics of the brain resting state networks, and MR spectroscopy was performed and directed to two major neurotransmitters: glutamate, the excitatory neurotransmitter and gamma aminobutyric acid (GABA), the main inhibitory neurotransmitter. These studies showed evidence for a loss of global efficiency, changes in resting-state fMRI temporal neurodynamics, and imbalanced neurotransmitter levels of excitation and inhibition associated with ASD in high functioning adolescents, which may be related to the constant compensatory tendency in these youngsters.

## Target group

First of all, the findings of this study are relevant for high-functioning adolescents with ASD, as these findings might, in the future, improve diagnostic processes and therapeutic treatments in ASD. This thesis is also of interests for their psychologists; who might benefit from the recommendations and may adjust their standard ASD assessment and therapeutic treatment in these high-functioning teens. Furthermore, our MRI studies provide results that may enhance our understanding of neuronal mechanisms that underlie important characteristics of ASD, and might give new directions for future research on the functional integrity of the brain and the mechanisms underlying ASD. Individuals with ASD may benefit from this as well, because (in the future) it may give insights for other (more suitable) therapeutic interventions or treatments in this high-functioning group.

### **Innovation and future directions**

Since the publication of Kanners first paper on Autistic children in 1943, much research has focussed on phenotyping, genotyping, describing and investigating diagnostic measures and treatment of individuals with a disorder on the autism. However, most of this research is focussed on children/childhood and far less is known of how ASD manifests itself in high-functioning adolescents. The studies presented in this thesis should be considered as explorations on the use of diagnostic instruments and the neurocognitive profile of high-average intelligent adolescents with ASD, and the mechanisms behind this disorder in these teens. We recommend that future studies concentrate more on combining research methods. This may give researchers and clinicians more insight into the relationships between behaviour, cognition (inside and outside the laboratory), and the functional integrity of the brain. Moreover, studying behaviour, cognition, and the brain in the same cohort will make it easier to interpret fMRI results and the effect of (dys)functional neural networks on everyday living and potential compensation strategies. These insights may help researchers and clinicians to develop and use better intervention techniques for this group of important pervasive developmental disorders.