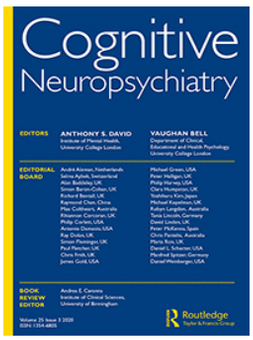

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# Similar impairments shown on a neuropsychological test battery in adolescents with high-functioning autism and early onset schizophrenia: a two-year follow-up study

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**ABSTRACT**

**Introduction:** Cognitive impairments are common in both Autism Spectrum Disorders (ASD) and schizophrenia, but it is unclear whether the pattern of difficulties is similar or different in the two disorders. This cross-sectional and longitudinal study compared the neuropsychological functioning in adolescents with ASD with adolescents with Early Onset Schizophrenia (EOS).

**Methods:** At baseline and at two-year follow-up, participants were assessed with a brief neuropsychological test battery measuring executive functions, visual and verbal learning, delayed recall and recognition and psychomotor speed.

**Results:** We found similar levels of neuropsychological impairment across groups and over time in the adolescents with ASD or EOS. Adolescents in both groups did not improve significantly on verbal learning, verbal delayed recall, visual learning, visual delayed recall or visual delayed recognition, and both groups performed poorer on verbal recognition. Both groups improved on measures of psychomotor processing and executive functions.

**Conclusion:** The findings suggest that it may be difficult to differentiate adolescents with EOS and ASD based on neuropsychological task performance. An implication of the results is that adolescents with either disorder may benefit from a similar approach to the treatment of cognitive impairment in the disorders.

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Early onset schizophrenia; high functioning autism; autism spectrum disorder; neuropsychological function; longitudinal

## Introduction

Autism Spectrum Disorders (ASD) and schizophrenia are disorders with different ages of onset, separate presenting characteristics and divergent developmental courses (Konstantareas & Hewitt, 2001). Nevertheless, both disorders have been conceptualised as neurodevelopmental disorders with a shared genetic architecture (Fiksinski et al., 2017; Ionita-Laza et al., 2014;

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Lee et al., 2013; Lionel et al., 2013). Increasing evidence suggests that ASD and schizophrenia co-occur at elevated rates. However, the prevalence of ASD in individuals with schizophrenia is more common than the prevalence of schizophrenia in ASD (Canitano & Pallagrosi, 2017; Chisholm, Lin, Abu-Akel, & Wood, 2015; Zheng, Zheng, & Zou, 2018). There is evidence of similar grey matter volume reduction in the limbic–striato–thalamic circuitry in both ASD and schizophrenia (Cheung et al., 2010). In addition, there are indications of an overlap of clinical symptoms in the two disorders (Spek & Wouters, 2010; Tordjman, 2008). Impairment in social functioning is one of the primary features of the clinical presentations in both ASD and schizophrenia (Couture et al., 2010). Problems with social functioning may be reflected in social withdrawal, little or no social communication ability or reduced facial or emotional expression and problems with close personal relationships (Wakabayashi, Baron-Cohen, & Ashwin, 2012). Impairments in social cognition and theory of mind are also common in both disorders (Lugnegård, Hallerbäck, Hjärthag, & Gillberg, 2013; Tin et al., 2018).

Although there has been an increasing interest in possible connections between ASD and schizophrenia in the past decade, there have been few studies examining similarities or differences in neuropsychological functioning between the disorders. Research on adults with ASD and schizophrenia have found similar deficits in their neuropsychological functioning, with the largest shared impairment involving speed of processing (Eack et al., 2013). Further, a recent study found an association between left prefrontal cortex dysfunction and neuropsychological deficits in both disorders (Pu et al., 2018). Marinopoulou, Lugnegård, Hallerbäck, Gillberg, and Billstedt (2016) found that adults with schizophrenia and adults with ASD shared similar neuropsychological profiles. Although their study revealed that both groups had relatively low working memory and processing speed scores compared with typically developing controls, full-scale IQ was significantly higher in the ASD group. In another study involving adults, processing speed was found to be significantly lower in the patients with schizophrenia compared with the patients with ASD (de Boer, Spek, & Lobbestael, 2014).

Results from neuropsychological studies on adult patients with ASD or schizophrenia may not directly apply to children and adolescents with the same disorders, due to the considerable cognitive development takings place between childhood and adulthood. Research results on adult patient samples may also be confounded by varying durations of illness or treatment within the groups that can impact neuropsychological performance. In addition, social isolation over time can be a confounding factor. Therefore, it is important to investigate whether neuropsychological deficits found in adult samples of ASD and schizophrenia are similar to those present in children and adolescents with the same disorders. Early Onset Schizophrenia (EOS) is often defined as onset before age 18. The lifetime prevalence of schizophrenia is 1% (Perälä et al., 2007). The prevalence of EOS is about 0.2%–0.5% (Excellence, 2013). EOS has been associated with a more severe clinical course than adult onset schizophrenia (Oie, Sundet, & Ueland, 2011). In 2012, the global prevalence of ASD was estimated by the World Health Organization to be 1% (Elsabbagh et al., 2012). A better understanding of the differences or similarities in neuropsychological functioning in children and adolescents with ASD or EOS would allow for cognitive interventions to be applied earlier and on a more evidence-based foundation.

Several cross-sectional studies have focussed on neuropsychological functioning in children and adolescents with ASD or EOS compared to typically developing (TD) controls.

In children with ASD, significant impairments have been documented in executive functioning (Demetriou et al., 2017; Hill, 2004; Lai et al., 2017) and memory performance (Williams, Goldstein, & Minshew, 2006). Some studies have also indicated a working memory developmental delay in longitudinal studies of children with ASD (Andersen, Hovik, Skogli, Egeland, & Oie, 2013; Andersen, Skogli, Hovik, Geurts, et al., 2015; Luna, Doll, Hegedus, Minshew, & Sweeney, 2007; O'Hearn, Asato, Ordaz, & Luna, 2008). Other longitudinal studies have found persistent mental flexibility and response inhibition impairments in children with ASD compared with TD controls (Geurts, van den Bergh, & Ruzzano, 2014; Pellicano, 2010).

In a review of cross-sectional studies of neuropsychological functioning in individuals with EOS, Frangou (2010) found impairments of medium to large effect sizes in IQ, attention, memory and executive functions compared with TD controls. One-year and two-year longitudinal studies of neuropsychological impairments in EOS found no change in the magnitude of deficits in patients with EOS (Cervellione, Burdick, Cottone, Rhinewine, & Kumra, 2007; Juuhl-Langseth, Holmen, Thormodsen, Oie, & Rund, 2014; Teigset et al., 2018). In two studies featuring four-year and 13-year follow-up intervals, there was a decline in verbal memory, attention and processing speed in individuals with EOS (Frangou, Hadjulis, & Vourdas, 2008; Oie et al., 2011; Wozniak, Block, White, Jensen, & Schulz, 2008). To sum up, both children and adolescents with ASD and EOS have been found to have executive and memory impairments compared to TD controls. One-year and two-year longitudinal studies indicate working memory delay in children and adolescents with ASD compared to no delay in children and adolescents with EOS. However, studies with longer follow-up intervals have found neuropsychological delay in young persons with EOS as well.

Only a few studies have compared children or adolescents with EOS directly with ASD on neuropsychological performance (Asarnow, Tanguay, Bott, & Freeman, 1987; Schneider & Asarnow, 1987; Waris et al., 2016). In an early work of Schneider and Asarnow (1987), they found similar levels of impairment in processing speed and executive functioning in children with EOS and ASD. However, children with ASD had significantly more impairments in verbal functioning as indexed by scores on the verbal comprehension factor as assessed using WISC-R compared with children with EOS (Asarnow et al., 1987). Children and adolescents with EOS have been found to have more impaired visual reasoning skills, working memory and processing speed than children and adolescents with ASD (Asarnow et al., 1987; Waris et al., 2016). Waris et al. (2016) also found evidence for impaired visual processing in children and adolescents with EOS compared to young patients with ASD. Thus, there are few comparison studies and some inconsistent findings regarding neuropsychological functioning in children and adolescents with ASD compared with EOS. Earlier research is also hampered by small sample sizes, a focus on single domains of neuropsychological functioning (i.e. IQ level), and an absence of a typically developing control group. Further, to the best of our knowledge, no longitudinal studies have directly compared the development of neuropsychological functioning in adolescents with ASD to that of adolescents with EOS.

As earlier described, although the disorders are distinct, they share central clinical features. It can therefore be difficult to differentiate diagnoses and thereby provide optimal treatment. This is especially important in children and adolescents, as they are in a period of considerable development of neuropsychological and social functions and

identity (Blakemore & Choudhury, 2006). Follow-up studies mapping the neuropsychological functioning of individuals with ASD or EOS may inform the field regarding salient cognitive similarities and differences between the disorders. In order to provide the best possible treatment for individuals with ASD and EOS, there is a need to understand the unique and shared developmental trajectories in the two developmental disorders. The current two-year follow-up study assesses a wider range of neuropsychological domains compared to other studies in a relatively larger sample of adolescents with ASD or EOS.

### The present study

The main aim of the current study is to compare neuropsychological functioning at baseline (T1) and after two years (T2) in adolescents with ASD (IQ > 70) compared with adolescents with EOS. Our research questions are: Are there any differences in neuropsychological functioning between adolescents with ASD and adolescents with EOS at baseline? Is there a difference between the neuropsychological development of the groups over two years? As there are few comparison studies on neuropsychological functioning in EOS and ASD, and conflicting results, we do not develop specific hypotheses.

## Materials and methods

### Participants and procedures

See Table 1 for demographic and clinical characteristics of the participants. In the current study, data from two different research projects were merged. One of the earlier studies had focussed on adolescents with ASD compared to TD controls; another study focused on adolescents with EOS compared to TD controls. In the current study, results from the ASD study were directly compared with results from the EOS study.

**Table 1.** Demographic and clinical characteristics: means and standard deviations by group and assessment time.

| | Baseline (T1) | | Follow-up (T2) | |
|---|---|---|---|---|
| Variable | ASD ($n = 21$) | EOS ($n = 22$) | ASD ($n = 20$) | EOS ($n = 17$) |
| Sex (male/female) | 20/1 | 11/11 | 19/1 | 7/10 |
| Age | 14.4 (1.5) | 15.2 (1.5) | 16.5 (1.5) | 17.6 (1.7) |
| Mother's education (yrs) | 12.9 (2.6) | 13.0 (3.0) | – | – |
| FSIQ[a,d] | 94.7 (17.1) | 99.8 (14.8) | 94.8 (16.4) | – |
| PIQ[b,d] | 95.0 (22.8) | 101.8 (14.8) | 99.4 (15.9) | – |
| VIQ[c,d] | 91.0 (19.0) | 97.1 (15.7) | 94.7 (17.6) | – |
| CGAS/GAF[e] | 49.1 (11.4) | 48.5 (15.7) | 48.4 (9.9) | 60.1 (10.6) |
| PANSS total[f] | – | 55.1 (11.4) | – | 50.4 (13.0) |
| ASSQ[g] | 25.8 (8.9) | – | 21.4 (9.6) | – |

Note: ASD: Autism Spectrum Disorder; EOS: early-onset schizophrenia.
[a]FSIQ; full scale IQ. IQ estimated measures from the Wechsler Abbreviated Scale of Intelligence (WASI).
[b]PIQ; estimated performance intelligence.
[c]VIQ; estimated verbal intelligence.
[d]IQ scores from T2 were not available for the EOS group.
[e]CGAS/GAF; Children's Global Assessment Scale/Global Assessment of Functioning.
[f]PANSS; Positive and Negative Syndrome Scale ($n = 24$).
[g]ASSQ; Autism Spectrum Screening Questionnaire.

The ASD group consisted of 38 participants and the EOS group of 27 participants. In order to obtain similar age distributions in the groups (12–18 years), the ASD group was restricted to 21 adolescents with ASD (20 males/1 female) and the EOS group to 22 (11 males/11 females) at baseline (T1). The ASD group was recruited in 2009–2010 from all Child and Adolescent Mental Health Centers in Innlandet Hospital Trust (CAMHC) in two Norwegian counties (Hedmark and Oppland) with a county-wide population of 375,000 people. In Norway, children with autism without intellectual disability are most often referred to CAMHC, whereas children with ASD and intellectual disability are referred to child habilitation centres. Thus, only children with ASD without intellectual disability were included in the study. See Andersen et al. (Andersen, Skogli, Hovik, Egeland, & Oie, 2015; Andersen, Skogli, Hovik, Geurts, et al., 2015) for a more detailed description of the cohort. Two individuals in the ASD group had comorbid depression and one had comorbid Attention Deficit Hyperactivity Disorder (ADHD). Participants with ASD were assessed with separate interviews of children and their parents using the Schedule for Affective Disorders and Schizophrenia for School Age Children/Present and Lifetime version-2009 (K-SADS-PL) (Kaufman et al., 1997). The K-SADS-PL is a semi-structured diagnostic interview designed to correspond to the Diagnostic and Statistical Manual for Mental Disorders fourth edition (DSM-IV; American Psychiatric Association, 2000). The K-SADS-PL was supplemented with information from the Autism Spectrum Screening Questionnaire (ASSQ; Ehlers & Gillberg, 1993). Clinical significance was assessed by applying normative data from the ASSQ (Ehlers, Gillberg, & Wing, 1999). The interviewers were experienced clinicians and were trained to high levels of interrater reliability for diagnostic assessment. In addition, the diagnostic evaluation was supplemented with information from the Child Behavior Checklist/6–18 (CBCL 6–18; Achenbach & Rescorla, 2001), which was completed by both parents. Diagnosis was confirmed if DSM-IV criteria were met through an exhaustive evaluation of the K-SADS-PL screening interview, K-SADS-PL diagnostic interview for autism, and parent reports together with information from teachers concerning academic and social functioning. Disagreements regarding diagnosis were discussed in meetings with all the clinicians present to arrive at a "best estimate" DSM-IV (American Psychiatric Association, 2000) consensus diagnosis. At baseline, nineteen in the ASD group were diagnosed with Asperger's syndrome and two with pervasive developmental disorder—not otherwise specified (PDD-NOS). One child was using psychostimulants (methylphenidate), but the medication was discontinued 24 h prior to assessment.

Follow-up (T2) assessment of the ASD group was conducted two years following baseline (T1) assessment. The reassessment procedures at T2 were similar to T1. All ASD diagnoses were confirmed. One participant in the ASD group refused to participate at T2. At T2, two participants in the ASD group were using antipsychotic medication and four were using psychostimulants (methylphenidate). The psychostimulants were discontinued 24 h prior to assessment.

The results for the EOS group were collected from an earlier broader research project at the University of Oslo, Norway, on adolescents aged 12–18 years with early onset psychotic disorders and TD controls (Holmen, Juuhl-Langseth, Thormodsen, Melle, & Rund, 2010; Juuhl-Langseth et al., 2012; Thormodsen, Juuhl-Langseth, Holmen, & Rund, 2012). This study had tested patients with EOS and TD controls on a neuropsychological test battery at baseline (between 2005 and 2008), after one year and after two years. In the

current study, we used the results from the baseline study (T1) and the two-year follow-up (T2). At baseline (T1), a total of 22 patients were included in this study (11 males/11 females). The patients were recruited from multiple hospitals and outpatient units in Oslo and Eastern Norway. Patients were included if they were between 12 and 18 years of age and met the DSM-IV (American Psychiatric Association, 2000) criteria for a broad schizophrenia-spectrum disorder (schizophrenia, schizophreniform disorder, schizoaffective disorder, brief psychotic disorder and psychosis not otherwise specified [NOS]). Patients were diagnosed using the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-I), modules A–D. Comorbid diagnoses were not registered in the EOS group. The mean inter-rater reliability (kappa) for the SCID was 0.77. Thirteen patients were on antipsychotic medication and one was on an antidepressant. Seventeen participants consented to further participation after two years. Procedures for re-assessment at follow-up were the same as for baseline. Seven patients in the EOS group were medicated with antipsychotics at the two-year follow-up.

All participants in both projects were offered standard ("treatment as usual") psychological and/or medical treatment in the period between inclusion and reassessment. The exclusion criteria for the ASD and the EOS groups were any neurological disease and an IQ estimate below 70.

There were significantly fewer girls at T1 ($p = .001$) and T2 ($p < .001$) in the ASD group compared to the EOS group. The differences between groups on the Children's Global Assessment Scale (CGAS)/Global Assessment of Functioning-Split version (Split-GAF) and on other demographic characteristics were not significant at T1 (Table 2).

### Measure of psychotic symptoms

Psychotic symptoms were assessed in the EOS group using the Positive and Negative Syndrome Scale (PANSS; Kay, Fiszbein, & Opler, 1987). The PANSS evaluates a multidimensional array of symptoms (e.g., positive, negative, depressive), and involves the use of data from patient reports, caregiver reports, and clinical observations (Kay et al., 1987; Opler, Yavorsky, & Daniel, 2017). Higher scores indicate more problems.

**Table 2.** Demographic characteristics: chi-square and univariate comparisons (Bonferroni corrected for multiple comparisons .05/6 = .008).

| | Baseline (T1) | | Follow-up (T2) | |
|---|---|---|---|---|
| | Group comparisons | | Group comparisons | |
| Variable | Chi-sq./ *F* | *p* | Chi-sq./ *F* | *p* |
| Sex (male/female) | 10.9 | .001 | 12.7 | <.001 |
| Age | (1,41) 3.26 | NS. | (1,35) 4.72 | NS. |
| Mother's education (yrs) | (1,40) .027 | NS. | – | – |
| FSIQ[a,b] | (1,41) 1.10 | NS. | – | – |
| PIQ[c] | (1,41) 1.39 | NS. | – | – |
| VIQ[d] | (1,41) 1.32 | NS. | – | – |
| CGAS/GAF[e] | (1,41) .027 | NS. | (1,33) 11.4 | .002 |

Note: ASD: Autism Spectrum Disorder; EOS: early-onset schizophrenia.
[a]FSIQ: full scale IQ. IQ estimated measures from the Wechsler Abbreviated Scale of Intelligence (WASI).
[b]IQ scores from T2 were not available for the EOS group.
[c]PIQ: estimated performance intelligence.
[d]VIQ: estimated verbal intelligence.
[e]CGAS/GAF: Children's Global Assessment Scale/Global Assessment of Functioning.

### Measure of ASD symptoms

The Autism Spectrum Screening Questionnaire (ASSQ; Ehlers & Gillberg, 1993) was administered to measure ASD symptoms in the ASD group. The ASSQ is a 27-item checklist for assessing symptoms characteristic of Asperger's Syndrome or high-functioning autism spectrum disorders. In the current study, a parent rating was used. The items encompass social interaction problems, communication problems and problems with restricted and repetitive behaviours (Ehlers et al., 1999). Higher scores indicate more problems.

### Global level-of-functioning measure

Global level of functioning in the ASD group was assessed using the CGAS (Shaffer et al., 1983) and functioning in the EOS group was assessed using the Split-GAF (Pedersen, Hagtvet, & Karterud, 2007). The CGAS is an adaptation of the GAF-F for adults. Higher scores on the CGAS or the GAF-F indicate a better level of functioning.

### Measures of neuropsychological functioning

#### Verbal learning and memory

The Hopkins Verbal Learning Test—Revised (HVLT-R) total learning, delayed recall, and recognition score were used (Brandt & Benedict, 2001). Higher raw scores indicate better performance.

#### Visual learning and memory

The Brief Visuospatial Memory Test—Revised (BVMT-R) total learning, delayed recall, and recognition were used (Benedict, 1997). Higher raw scores indicate better performance.

#### Verbal processing speed

The Color-Word Interference Test from the D-KEFS battery (Delis, Kaplan, & Kramer, 2001) runs the following two baseline conditions: colour naming (CW1) of colour patches, and word reading (CW2) of colour-words printed in black. We measured overall time used to complete the task. Higher raw scores indicate difficulties with the task.

#### Inhibition

The Color-Word Interference Test from the D-KEFS battery, Condition 3 (CW3; Delis et al., 2001). The participant must inhibit an overlearned verbal response when naming the dissonant ink colours in which the words are printed. We measured overall time used to complete the task. Higher raw scores indicate difficulties with the task.

#### Mental flexibility

The Color-Word Interference test from the D-KEFS battery, Condition 4 (CW4; Delis et al., 2001). In CW4, the participant must switch between reading printed words written in different colours and name the dissonant ink colour. We measured overall time used to complete the task. Higher raw scores indicate poorer performance.

### Estimated verbal and non-verbal intelligence

Because the participants in the projects underwent a large test battery, it was important to limit the number of tests in order to make it more attractive for them to participate in the follow-up study. To estimate full-scale IQ, the Wechsler Abbreviated Scale of Intelligence was administered (WASI; Wechsler, 2007). WASI is the short version of the Wechsler's intelligence tests. A Norwegian study found almost identical Total IQ on WASI and WAIS / WISC-R in a mixed clinical group sample (Bosnes, 2009). WASI provides a reliable, brief measure of intelligence in clinical, educational and research settings. WASI is a battery of four subtests: Vocabulary, Block Design, Similarities, and Matrix Reasoning. The four subtests comprise an estimate of Full-Scale IQ (FSIQ). The Vocabulary and Similarities subtests are combined to form an estimate of Verbal IQ (VIQ), and the Block Design and Matrix Reasoning subtests yield an estimate of Performance IQ (PIQ) score. Since we only had WASI scores from T1 for the EOS group on this measure, we could only compare the groups on this measure at baseline.

### Statistical analyses

Data analyses were performed with IBM SPSS Statistics, version 25 (IBM, SPSS, Inc., Chicago, IL). Significant results are reported at $p < .05$ level. Demographic characteristics were investigated using the Chi-squared test for independence (gender) and independent measures *T*-test (age, mother's education, IQ and CGAS/GAF). Analyses of variance (ANOVA) was used to investigate differences between the two groups at baseline (T1). Mixed between-within subjects' ANOVAs (mixed ANOVA) were conducted to assess the interaction between diagnosis and time for all neuropsychological measures. We ran separate mixed ANOVAs controlling for the effect of antipsychotic medication at baseline. Due to multiple comparisons, Bonferroni corrections were used to control for chance findings. Since we did not have standard scores for all measures, we used the raw scores when comparing performance between the groups.

### Ethical approval

Both research projects received advance approval by the Regional Committee for Medical Research Ethics in Eastern Norway (REK-Øst), and by the Privacy protection ombudsman for research at Innlandet Hospital Trust or Norwegian Data Inspectorate. We also received ethical approval to merge the two datasets form the different ASD and EOS studies. Consent was given in accordance with the ethical principles specified in the Declaration of Helsinki.

## Results

There was no significant effect of group on the neuropsychological measures. There was a significant effect of time for both groups with improved results on verbal processing speed (CW1) ($p < .001$), inhibition (CW3) ($p < .001$), mental flexibility (CW4) ($p < .001$) (see Table 3). There was a significant effect of time for both groups with decreased results on verbal recognition (HVLT)($p = .004$). However, both groups achieved near maximum scores at both time points. They did not improve significantly over the two

**Table 3.** Results on CW 1–4 (completion time in sec.), HVLT (raw scores) and BVMT (raw scores) at T1 and T2, WASI comprehension and similarities at T1 (*T*-scores): means and standard deviations within the ASD and the EOS groups, and results (Bonferroni corrected for multiple comparisons .05/12 = .004) from Mixed Model ANOVA and ANOVA.

| | ASD ($n = 20$) | | EOS ($n = 17$) | | Group | | Time | | Time × Group | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Variable | T1 | T2 | T1 | T2 | *F* | *p* | *F* | *p* | *F* | *p* | $\eta_p^2$ |
| CW1[a] | 45.2 (12.6) | 38.5 (9.3) | 36.9 (7.8) | 32.2 (7.2) | (1,34) 6.00 | NS. | 22.1 | <.001 | .675 | NS. | .019 |
| CW2 | 30.1 (6.7) | 28.0 (6.4) | 27.1 (7.7) | 24.3 (6.5) | (1,34) 2.54 | NS. | 7.09 | NS. | .166 | NS. | .005 |
| CW3 | 85.6 (25.5) | 69.2 (21.8) | 64.6 (18.6) | 56.1 (18.7) | (1,34) 7.11 | NS: | 14.4 | .001 | 1.44 | NS. | .041 |
| CW4 | 91.1 (25.7) | 71.0 (13.3) | 72.2 (22.4) | 62.6 (22.8) | (1,34) 4.60 | NS. | 19.3 | <.001 | 2.45 | NS. | .067 |
| HVLT learning[b] | 22.7 (6.3) | 20.6 (4.3) | 24.8 (5.4) | 23.9 (5.9) | (1,35) 2.78 | NS. | 3.16 | NS. | .501 | NS. | .014 |
| HVLT delayed recall | 7.6 (2.7) | 7.2 (2.6) | 8.4 (2.9) | 7.4 (2.5) | (1,35) .482 | NS. | 2.40 | NS. | .461 | NS. | .016 |
| HVLT recognition | 11.5 (.6) | 11.1 (1.1) | 11.5 (1.1) | 10.6 (1.3) | (1,35) .581 | NS. | 9.69 | .004 | 1.37 | NS. | .038 |
| BVMT learning[c] | 22.8 (8.4) | 25.3 (9.7) | 25.4 (8.1) | 26.2 (6.2) | (1,33) .451 | NS. | 1.94 | NS. | .492 | NS. | .015 |
| BVMT delayed recall | 9.5 (3.3) | 10.1 (2.9) | 10.1 (2.2) | 9.9 (2.3) | (1,33) .065 | NS. | .445 | NS. | 1.05 | NS. | .031 |
| BVMT recognition | 5.9 (.32) | 6.7 (2.5) | 5.8 (.39) | 6.0 (.0) | (1,33) 1.53 | NS. | 2.86 | NS. | 1.21 | NS. | .035 |
| WASI comprehension[d] | 43.9 (8.9) | | 47.2 (10.9) | | (1,41) 1.23 | NS. | | | | | |
| WASI similarities | 47.7 (11.8) | | 48.5 (11.7) | | (1,41) .054 | NS. | | | | | |

Note: [a]CW1-4: Delis-Kaplan Colour-word interference test conditions 1, 2, 3 and 4 time in seconds.
[b]HVLT: Hopkins Verbal Learning Test—Revised.
[c]BVMT: Brief Visuospatial Memory Test—Revised.
[d]WASI: Wechsler Abbreviated Scale of Intelligence.

years on verbal learning, verbal delayed recall, visual learning, visual delayed recall or recognition. There was no significant interaction effect between time and group on the neuropsychological measures. Analyses conducted controlling for the use of antipsychotics revealed no significant group differences on the neuropsychological measures. Thus, no group differences were found when running analyses controlling for or not controlling for potential medication effects.

Earlier results from the previous ASD study have shown that the individuals displayed stable deficits compared to TD controls on most measures at T1 and T2. The only exception was performance after two years on verbal working memory, where the ASD group showed a decline in results compared to the TD adolescents (Andersen et al., 2013; Andersen, Skogli, Hovik, Geurts, et al., 2015). Earlier results on the EOS group's neuropsychological performance indicate a stable deficit at T1 and T2 on most measures compared to TD individuals (Juuhl-Langseth et al., 2012).

## Discussion

The main finding of the current study is that there were similar levels of neuropsychological impairment in the adolescents with ASD or EOS across groups and over time. Our cross-sectional results are consistent with the results of Schneider and Asarnow (1987). Our results support the hypothesis of shared neuropsychological pathways influencing cognitive development in these two neurodevelopmental disorders and support the notion of an autism-schizophrenia continuum (King & Lord, 2011). However, different neural substrates may be responsible for similar cognitive impairments in EOS and ASD. One study found two distinct areas involved in the brain pathophysiology relevant to cognitive processing in patients with ASD and schizophrenia (Hirata et al., 2018). If different neural substrates cause similar cognitive endophenotypes, then this may indicate an important direction for future research, especially when considering cognitive remediation and medical treatment in these two disorders.

Asarnow et al. (1987) and Waris et al. (2016) found that their EOS groups showed significantly lower performance in processing speed compared with their ASD groups. We found no significant differences between the ASD and the EOS groups in processing speed. An explanation for the difference in results in our study compared with earlier studies may be that the neuropsychological tests used in earlier studies were not the same as in our study. The earlier studies used tests that required motor processing speed, whereas the test used in our study taxed verbal processing speed. Another explanation for our results might be that the processing speed tasks applied in our study were too simple, and thus were not able to differentiate between the two groups. The manipulative load is relatively low in the Color-Word Interference Test conditions 1 and 2. In their study examining working memory function, Conklin, Luciana, Hooper, and Yarger (2007) found that low central executive processing matures prior to more complex ones requiring cognitive manipulation. This might suggest that tasks applying more basic processing requirements were too simple to detect subtle neuropsychological differences between the groups compared to using tasks requiring more cognitive effort.

Asarnow et al. (1987) found that children with ASD had significantly more impairments in verbal functioning compared with children with EOS. Many of the children with ASD in their study, however, had deficits in language development and/or peculiar

speech patterns, which is in contrast to our study in which none in our ASD group had any delayed language development issues. Further, the participants in the Asarnow et al. (1987) study were younger than our participants. These factors may explain the differences in results between our studies.

Waris et al. (2016) also found evidence of impaired visual processing measured with all of the visual cognition subtests from WISC-III in EOS compared with children and adolescents with ASD. In our study, two performance IQ tests were used from the WASI, and we used the BVMT to investigate visual cognition. The use of different outcome tests for visual cognition in the two studies means that caution must be exercised when comparing the results in this domain.

We found no differences between the groups in the two-year longitudinal course of the neuropsychological functions. As far as we know, no other studies have investigated neuropsychological similarities or differences over such a long period of time during a critical developmental period in adolescents with ASD and EOS. More longitudinal studies comparing neuropsychological abilities across the two disorders are needed.

### *Strengths and limitations of the study*

A strength of our study is that the sample size was larger than other comparison studies regarding neuropsychological functioning in adolescents with EOS or ASD. Still, small sample size may represent a weakness in this study. A small sample, however, is a common problem in longitudinal studies of EOS, due to the rarity of the illness and the challenges of long-term follow-up of this population (Cervellione et al., 2007). Other strengths of this study are the use of a neuropsychological test battery and the longitudinal design. However, the neuropsychological test battery was relatively brief and a broader neuropsychological testing could have been even more helpful to understand the cognitive functioning of the two groups. Further, the lack of WASI data for the EOS group at T2 is a limitation. Due to the lack of detailed information regarding interventions given during the follow-up period, we were not able to control for interventions. Further, some EOS patients were assessed without antipsychotic medication at T2 and with medication at T1. In addition, two participants in the ASD group were using antipsychotic medication at T2 but not at T1. This may have confounded the course of cognition with potential medication effects. Another limitation may be the lack of having used clinical diagnostic observation tools such as the Autism Diagnostic Interview—Revised (ADI-R) or Autism Diagnostic Observation Scale (ADOS) for the ASD group. Although neuropsychological functioning matures considerably in the adolescent years, it is possible that two years is too short a period to detect any differing trajectories in neuropsychological development between the groups in the study. Future research should therefore include a longer follow-up period. Further, a one-year follow-up in both groups would have allowed us to model the data with more complex statistical models that could have provided a clearer picture of cognitive change over time.

### *Clinical implications*

Our findings provide support for the position that there are close similarities in neuropsychological deficit patterns between ASD and EOS. A possible clinical implication is that

measures of neuropsychological functioning cannot differentiate EOS from ASD in clinical practice. Because of similarities in symptomatology and cognitive deficits in ASD and EOS, clinicians may be less likely to detect the co-occurrence or continuity of autism and schizophrenia conditions. Thus, if a child has a diagnosis of ASD and develops schizophrenia later on, clinicians may be less likely to diagnose it (Pina-Camacho, Parellada, & Kyriakopoulos, 2016). A recent study found transdiagnostic validity of the Measurement and Treatment Research to Improve Cognition in Schizophrenia (MATRICS) Consensus Cognitive Battery (MCCB) across the autism-schizophrenia spectrum in adults (Kuo, Wojtalik, Mesholam-Gately, Keshavan, & Eack, 2019). The study highlights MCCB's applicability to ASD and supports its utility for standardising treatment evaluations of cognitive outcomes across the autism-schizophrenia spectrum. The data from our research project on adolescents with ASD and EOS provide support for the position that there are close similarities in neuropsychological deficit patterns between ASD and EOS and that standardised assessment tools can be valid and reliable for use in both populations. EOS and ASD are associated with educational failure, occupational impairment and reduced quality of life (Baxter et al., 2015; Woolfenden, Sarkozy, Ridley, Coory, & Williams, 2012). Our findings identifying a shared pattern of neuropsychological impairment in individuals with ASD and EOS offers possible common ground for early cognitive remediation efforts for these two devastating developmental disorders.

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No potential conflict of interest was reported by the authors.

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