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Childhood Executive Function Predicts Internalizing and Externalizing Symptoms in Emerging Adults With and Without Autism: A 10-Year Longitudinal Study

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ABSTRACT

Individuals with autism spectrum disorder (ASD) and typically developing individuals were assessed on three neuropsychological tests of executive function (EF) and on scales of autism symptoms and co-occurring internalizing and externalizing symptoms at baseline (T1; $N = 88$, $M_{age} = 11.8$ years, 73% males), 2-year (T2; 99% retention, $M_{age} = 13.9$ years), and 10-year follow-ups (T3; 75% retention, $M_{age} = 21.4$ years). An EF composite score from T1 significantly predicted internalizing symptoms at T2 (β = .228) and internalizing and externalizing symptoms at T3 (β = .431 and .478, respectively), when controlling for age and autism symptoms. OThe findings suggest that EF difficulties are a long-term risk factor for more co-occurring symptoms. .

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Co-occurring symptoms are more prevalent among individuals with Autism Spectrum Disorder (ASD) compared to the general population (Lai et al., 2019). More than 70% of children and adolescents with ASD display co-occurring symptoms (Lai et al., 2014). A recent large-scale meta-analysis on co-occurring mental health diagnoses in the ASD population found pooled prevalence estimates of 20% for anxiety disorders, 11% for depressive disorders, 12% for disruptive, impulse-control, and conduct disorders, and 28% for attention-deficit/hyperactivity disorder (Lai et al., 2019). In addition, the levels of co-occurring symptoms, although not meeting the diagnostic cutoffs, are high (Andersen et al., 2015; Guerrera et al., 2019). Findings indicate that co-occurring symptoms are prevalent among individuals with ASD throughout the life span (Lever & Geurts, 2016; Orm et al., 2021; Uljarevic et al., 2020).

Co-occurring symptoms are considered a central source of disability in ASD, and may cause as many difficulties as the ASD features do (Gillberg & Coleman, 2000; Lai et al., 2014). There is evidence that comorbid psychiatric disorders in children and adolescents with ASD are related to a lower level of functioning (Mattila et al., 2010). Greater severity of anxiety and depressive symptoms was related to reduced quality of life, after they accounted for autistic traits, in a study of individuals with and without ASD (Oakley et al., 2021). Correspondingly, depressive symptoms had a large negative influence on various quality of life domains in a study of individuals with ASD aged 15–80 years (L. P.Lawson et al., 2020). There is evidence that co-occurring symptoms predict lower quality of life in individuals with ASD (L. P.Lawson et al., 2020; Mason et al., 2018; Oakley et al., 2021). The importance

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of co-occurring symptoms is highlighted by their link to quality of life and reduced functioning, and leads to the question of which factors contribute to the high levels of co-occurring symptoms.

Difficulties with executive function (EF) have been suggested as a transdiagnostic indicator of atypical development (Abramovitch et al., 2021; Zelazo, 2020), and are considered a hallmark of ASD (Craig et al., 2016). EF difficulties are one potential mechanism underlying the development and maintenance of co-occurring symptoms among individuals with ASD (Andersen et al., 2017; R. A. Lawson et al., 2015). EF refers to a set of cognitive processes that are necessary for goal-directed behaviors, by guiding, monitoring, and regulating actions and behaviors important for learning and everyday performance tasks (Bagetta & Alexander, 2016). Working memory, inhibition, and flexibility are three widely recognized core components of EF (Miyake et al., 2000).

A large body of research has demonstrated that EF difficulties are common among individuals with ASD (Demetriou et al., 2017). One meta-analysis building on 235 studies found support for broad, as opposed to component-specific, EF difficulties in ASD (Demetriou et al., 2017). EF difficulties in individuals with ASD seem to persist across age, indicating a long-lasting vulnerability (Demetriou et al., 2017; Fossum et al., 2021). Relatedly, EF difficulties have been suggested as an underlying risk factor for developing and maintaining emotional and behavioral problems (Zelazo, 2020). EF difficulties during childhood could influence later mental health via poorer attentional, emotional, and behavioral control (Nelson et al., 2019). Internalizing symptoms typically involve difficulties controlling attention or emotion, while externalizing symptoms typically involve difficulties controlling emotion or inappropriate behaviors (Achenbach et al., 2016; American Psychiatric Association, 2013; Yang et al., 2022). Further, due to the relative stability of EF difficulties, such difficulties could over time influence several developmental areas and outcomes that indirectly affect mental health, such as education and occupation, social relationships with friends, family, and partner, independent living, and economic management.

A comprehensive meta-analysis on prospective longitudinal studies in clinical and non-clinical samples of children/adolescents reported that better child EF was associated with less internalizing and externalizing symptoms in the future (Yang et al., 2022). Only a minority of the included studies, however, followed the participants into adulthood. The authors emphasized that for internalizing symptoms especially, there was a need for further examination of these longitudinal associations in clinical samples (Yang et al., 2022). There is some evidence for a link between EF difficulties and co-occurring symptoms in individuals with ASD. In a cross-sectional study on children and adolescents with ASD, more EF difficulties were associated with more symptoms of anxiety, but not depression (Hollocks et al., 2014). In another study from the same sample, cognitive inflexibility was cross-sectionally associated with emotional problems, but not behavioral problems at age 16, while EF difficulties prospectively predicted more emotional and behavioral problems at age 23 (Hollocks et al., 2021). Relatedly, the levels of autism symptoms and inattention symptoms positively predicted emotional and behavioral symptoms two years later in a longitudinal study on children and adolescents with and without ASD (Andersen et al., 2017). A recent systematic review and meta-analysis targeting one domain of EF, reported that more difficulties with cognitive flexibility were cross-sectionally associated with more internalizing and externalizing symptoms, in children and adolescents with ASD (Lei et al., 2022). Most studies included in this review, however, used rating-measures to assess EF. Overall, longitudinal research on the association between EF and later co-occurring symptoms in the ASD population is scarce. Shedding light on mechanisms that increase the risk for co-occurring symptoms in individuals with ASD may help to determine where to target interventions to improve the mental health and wellbeing of individuals with ASD (Lai et al., 2019; Lei et al., 2022). We argue that investigating broader EF measures is valuable, based on the finding of broad, as opposed to domain-specific EF difficulties in ASD (Demetriou et al., 2017).

The current study

We aimed to expand the understanding of why ASD populations experience higher levels of co-occurring symptoms. EF assessed with neuropsychological tests in childhood/adolescence was investigated as a concurrent and longitudinal predictor of internalizing and externalizing symptoms in indidividuals with ASD and typically developing (TD) individuals. We wanted to identify a potential unique contribution of EF above that of ASD symptoms.

We expected to find significant associations between EF at baseline and internalizing and externalizing symptoms cross-sectionally. We hypothesized that more EF difficulties at baseline would predict higher levels of internalizing and externalizing symptoms 2 years later and 10 years later. Although we decided to investigate the impact of EF on later co-occurring symptoms, the relationship between EF and co-occurring symptoms may be bi-directional (Romer & Pizzagalli, 2021). Separate analyses were run to explore whether early co-occurring symptoms predicted later EF using an exploratory approach. Lastly, we *post-hoc* explored whether EF at 2-year follow-up mediated the relationship between baseline ASD symptoms and internalizing and externalizing symptoms in emerging adulthood.

Methods

Procedure

We used data from the LIllehammer Neurodevelopmental Follow-Up Study (LINEUP), where diagnostic, functional, and neuropsychological assessments were conducted in three waves: baseline in 2009–2010, 2-year follow-up in 2011–2012, and 10-year follow-up in 2018–2020 (Fossum et al., 2021) .

Baseline

At baseline (T1), children and adolescents between the ages of 8 and 17 years were recruited from the Child and Adolescent Mental Health Centers in Innlandet Hospital Trust in Norway, upon consecutive referrals with suspicion of an autism spectrum disorder (ASD). Thirty-eight individuals met criteria for ASD and were included. In addition, our sample included a comparison group with 50 TD children/adolescents who were recruited from local schools.

All participants underwent diagnostic assessments based on separate interviews with children and parents, using the Schedule for Affective Disorders and Schizophrenia for School Aged Children/Present and Lifetime version-2009 (K-SADS-PL; Kaufman et al., 1997). The interviews were performed by experienced psychologists and educational therapists. Information from the Autism Spectrum Screening Questionnaire (ASSQ; Ehlers et al., 1999) was used as a supplement to the interviews. After a comprehensive evaluation of information from K-SADS-PL, self-reports, parent reports, and teacher reports on academic and social functioning, an ASD diagnosis was assigned if criteria from DSM-IV were met. See Andersen et al. (2013) for more details on the diagnostic assessments. The exclusion criteria at T1 for all participants were prematurity (<36 weeks), IQ estimate below 70, and neurological disease. Additional T1 criteria for the comparison group were no history of psychiatric disorder, dyslexia, or head injury with loss of consciousness. All participants completed a neuropsychological test battery.

2-year follow-up

All participants were followed up two years later (T2; mean interval T1 – T2 = 25.4 months, SD = 2.6), except one who declined to participate (1 × ASD). The diagnostic, functional, and neuropsychological assessments were repeated.

10-year follow-up

The diagnostic and functional assessments from T1 and T2 were repeated after 10 years (T3; mean interval T1 – T3 = 115.0 months, SD = 5.5), using age-appropriate measures. The neuropsychological assessment was repeated using the same tests from T1. One educational therapist, one specialist in clinical neuropsychology, and one psychologist performed the interviews, additionally four undergraduate psychology students carried out the testing, under the supervision of the specialist in clinical neuropsychology.

Eight participants in the ASD group met criteria for co-occurring attention-deficit/hyperactivity disorder (ADHD) at baseline, whereof three participants used stimulant medication at T1, four at T2, and two at T3. Participants using stimulant medication discontinued use 24 h prior to testing at each assessment. One participant in the ASD group forgot to discontinue stimulant medication prior to testing at T3.

Participants and sample retention

The baseline sample consisted of 38 individuals with an ASD diagnosis (31 × Asperger syndrome, 7 × unspecified pervasive developmental disorder, mean age 12.0 years) and 50 TD individuals (mean age 11.6 years). See Table 1 for demographic and clinical characteristics for the sample, across assessment waves. Data on mothers' educational level were included as an indicator of socioeconomic status. Data on race/ethnicity were not recorded. In the ASD group, eight participants had co-occurring ADHD, one had an affective disorder, five had anxiety disorders, one had a conduct disorder, and one had an oppositional defiant disorder.

There were 87 participants at T2 (retention rate = 98.9%) and 66 participants at T3 (retention rate = 75.0%). Among the 22 individuals from T1 who did not participate at T3, five were untraceable or deceased (3 × ASD, 2 × TD), while 17 declined further participation (9 × ASD, 8 × TD).

Baseline differences in demographic characteristics and study variables between those who participated at T3 to those who dropped out, were investigated using independent samples T-test. We found no statistically significant differences between participants and drop-outs for age, IQ, mother's educational level, internalizing symptoms, externalizing symptoms, or autism symptoms, but the T3 participants had significantly better baseline EF than those who opted out ($p = .024$).

Ethics

The study was conducted in accordance with the Helsinki Declaration of the World Medical Association Assembly. At T1 and T2, children aged 12 years and older and their parents gave informed written consent prior to inclusion, while children below 12 years of age gave verbal consent prior to inclusion. All participants gave their informed written consent at T3. The Regional Committee for Medical Research Ethics in Eastern Norway (T1: REK Øst-Norge 6-2009-24, T3: 2018/1611/REK Sør-Øst) and the privacy ombudsman for research at the Innlandet Hospital Trust (nr. 95495) approved the study.

Measures

Estimated general cognitive function

The Wechsler Abbreviated Scale of Intelligence was administered at each assessment time point in order to estimate participants' intellectual abilities (Wechsler, 1999). We used the Full-Scale Intelligence Quotient estimates (FSIQ).

Executive function

We computed a global composite measure of EF from the participants' results on three neuropsychological tests assumed to assess three core EF components (Bagetta & Alexander, 2016; Miyake et al.,

Table 1. Demographic and clinical characteristics.

	M	*SD*	*Range*	*M*	*SD*	*Range*	*χ2/F*	*P*	Hedges' g
T1 – baseline		ASD (*n* = 38)			TD (*n* = 50)			Group comparison	
Age	12.03	2.34	9–17	11.56	1.99	8–17	1.016	.316	0.22
% boys/girls	84/16			64/36			4.446	.035	
Full-scale IQ	98.26	17.82	70–137	103.78	12.95	77–133	2.830	.096	0.36
Mothers' education	12.79	2.67	9–17	14.58	2.37	9–18	11.014	.001	0.72
ASSQ	21.47	9.27	4–49	1.62	1.85	0–8	218.657	<.001	3.18
Executive function[a]	2.93	3.18	−3.76–10.91	0.00	2.57	−4.50–5.66	21.738	<.001	1.03
Internalizing symptoms[b]	65.53	10.63	34–82	42.43	8.65	33–66	124.919	<.001	2.42
Externalizing symptoms[b]	59.08	10.59	40–79	40.78	7.52	33–58	88.852	<.001	2.04
T2–2-year follow-up		ASD (*n* = 37)			TD (*n* = 50)				
Sample retention %	97%			100%					
Age	14.21	2.35	11–19	13.62	1.95	10–19	1.673	.199	0.27
% boys/girls	84/16			64/36			4.166	.041	
Executive function[c]	1.39	3.06	−4.58–7.93	−1.81	2.35	−5.90–5.70	29.977	<.001	1.21
Internalizing symptoms[d]	59.82	10.86	34–76	41.10	8.26	33–62	80.451	<.001	1.99
Externalizing symptoms[d]	50.18	8.85	33–69	39.74	5.59	34–51	43.909	<.001	1.47
T3–10-year follow-up		ASD (*n* = 26)			TD (*n* = 40)				
Sample retention (%)	68%			80%					
Age	22.15	2.62	18–27	20.88	1.88	17–26	5.325	.024	0.58
% boys/girls	81/19			65/35			1.911	.167	
Executive function[e]	−0.73	3.04	−4.92–5.47	−4.02	1.97	−8.44–0.22	26.274	<.001	1.37
Internalizing symptoms[f]	56.71	11.72	35–78	45.85	10.38	25–68	14.898	<.001	1.00
Externalizing symptoms[f]	50.04	11.89	29–74	43.33	8.81	29–68	6.681	.012	0.67

Note. ASD = autism spectrum disorder, TD = typically developing. Full-scale IQ estimated from Wechsler Abbreviated Scale of Intelligence; Mother's education in years. ASSQ = Autism Spectrum Screening Questionnaire; Executive function (higher scores indicate more problems) = composite score calculated from results on the Letter-Number Sequencing Test from Wechsler Intelligence Scale for Children-IV, the Color-Word Interference Test, Condition 3, and the Trail Making Test, Condition 4, both from Delis-Kaplan Executive Function System. Internalizing and externalizing symptoms from Achenbach System of Empirically Based Assessment; Children Behavior Checklist at T1 and T2, Adult Self-Report at T3. [a]*n* = 34 ASD, [b]*n* = 49 TD, [c]*n* = 33 ASD, [d]*n* = 34 ASD, [e]*n* = 22 ASD, *n* = 39 TD, [f]*n* = 24 ASD.

2000). The Letter/Number Sequencing Test from the Wechsler Intelligence Scales for Children-IV (Wechsler, 2004) was used to estimate working memory. The Color-Word Interference Test, Condition 3, from the Delis Kaplan Executive Function System (D-KEFS; Delis et al., 2001) was used to estimate inhibition. The Trail Making Test, Condition 4, from D-KEFS (Delis et al., 2001) was used to estimate flexibility. Test scores were converted to Z scores based on the baseline mean and standard deviation in the TD group, where the new variables correlated perfectly with the original variables and retained all inter-individual variance. Higher scores indicate more EF difficulties. See Supplemental Table 1 for correlation coefficients between the three neuropsychological tests that were combined in the EF composite score.

Autism symptoms

Parents completed the Autism Spectrum Screening Questionnaire (ASSQ) at T1. The 27 items concern social interaction problems, communication problems, and problems with restricted and repetitive behavior (Ehlers et al., 1999). Each item is rated on a 3-point scale (0 = no problems, 1 = some problems, 2 = severe problems). A total score is computed by summarizing all responses (max = 54). Higher scores indicate more problems. The ASSQ has excellent test-retest reliability, interrater reliability, sensitivity, and specificity (0.62–0.91) (Ehlers et al., 1999; Posserud et al., 2009). We used the autism symptoms variable instead of a dichotomous group variable because a dimensional variable preserves more variation between individuals and increases statistical power (Agresti & Finlay, 2009).

Co-occurring internalizing and externalizing symptoms

We used two instruments from the Achenbach System of Empirically Based Assessment (ASEBA) for assessing emotional and behavioral symptoms. Parents filled out the Child Behavior Checklist 6–18 (CBCL; Achenbach & Rescorla, 2001) at T1 and T2. At T3, the participants filled out the corresponding version Adult Self Report Scale (ASR; Achenbach & Rescorla, 2003). The CBCL and ASR consist of 113 and 126 items, respectively, which are scored on a 3-point scale (0 = absent, 1 = occurs sometimes, 2 = occurs often). For both instruments, two broadband scores can be computed: *internalizing symptoms* from the syndrome scales anxious/depressed, withdrawn/depressed, and somatic complaints, and *externalizing symptoms* from the scales aggressive behavior, rule-breaking behavior, and intrusive. We used the internalizing and externalizing symptoms T-score ($M = 50$, $SD = 10$), based on American norms, where higher scores indicate more symptoms. Norwegian norms are lacking, and children in Norway typically obtain lower scores than American children do (Kornør & Jozefiak, 2012). The CBCL and ASR have good levels of reliability ($\alpha \geq .80$), sensitivity (40–83%), specificity (70–94%), and factor structure (e.g. de Vries et al., 2020; Kornør & Jozefiak, 2012).[1]

Statistical analyses

We conducted the statistical analyses in SPSS (version 26). Significant results are reported at $p \leq .05$, $p \leq .01$, and $p \leq .001$ level. We used pairwise deletion to address missing data. First, we ran a Pearson bivariate correlation analysis to determine the associations between EF (T1), autism symptoms (T1), internalizing symptoms (T1, T2, and T3) and externalizing symptoms (T1, T2, and T3).

Next, we used hierarchical multiple-regression analyses to predict the proportion of variance in the dependent variables (internalizing and externalizing symptoms) at T1, T2 and T3, that could be attributed to the independent variables from T1 (EF and autism symptoms). We ran the analyses for the entire sample (ASD and TD) because this yielded a higher N and larger variance in the outcome variables.

Six separate hierarchical multiple-regression analyses were conducted where internalizing and externalizing symptoms at T1, T2, and T3 comprised the dependent variables. The predictors from T1 were entered in three steps. In model 1, we entered age for it to serve as a control variable in subsequent steps. In model 2, we entered autism symptoms. In model 3, we entered the EF composite measure to assess the predictive value of EF above the effect of age and autism symptoms. For each model, we assessed the increase in the explained variance (ΔR^2). The number of data for each analysis is reported in Tables 3 and 4. We ran separate regression analyses where we added mother's education (as a proxy for socio-economic status) as a covariate in model 1.

We decided to do a post-hoc simple mediation model, because the findings in the hierarchical regression analysis indicated a potential mediation effect. The mediation model investigated if there might be an indirect effect of autism symptoms at T1 via EF at T2 to co-occurring symptoms at T3 (internalizing symptoms, externalizing symptoms). We ran a regression-based mediation analysis with the PROCESS Macro version 3.4.1 for SPSS (Hayes, 2018). Estimates of indirect effects were based on 5000 bootstrapped samples. Estimates were considered statistically significant if the 95% confidence intervals did not include zero.

Finally, we switched places for the main predictor and the dependent variable in the additional exploratory regression analyses, to explore whether baseline internalizing or externalizing symptoms predicted EF composite scores at T2 or T3. The aim of the exploratory analysis was to inform the interpretation of our main findings.

Results

Table 2 displays results from the correlation analysis with independent and dependent variables.

Table 2. Bivariate correlations between study variables.

				Baseline (T1)			2-year follow-up (T2)			10-year follow-up (T3)		
		Age	ASSQ	EF	INT	EXT	EF	INT	EXT	EF	INT	EXT
T1	Age	-										
	ASSQ	.24*	-					.				
	EF	−.36**	.36***	-								
	INT	.06	.77***	.39***	-							
	EXT	.10	.73***	.35**	.77***	-						
T2	EF	−.18	.42***	.85***	.38***	.50***	-					
	INT	.09	.69***	.39***	.81***	.58***	.29**	-				
	EXT	.12	.59***	.28**	.63***	.75***	.35**	.70***	-			
T3	EF	.11	.56***	.66***	.49***	.52***	.76***	.39**	.41**	-		
	INT	.16	.39***	.39**	.45***	.23	.42**	.52***	.24	.38**	-	
	EXT	−.03	.28*	.47***	.19	.25*	.54***	.25*	.22	.36**	.54***	-

Note. T1 = baseline, T2 = 2-year follow-up, T3 = 10-year follow-up. ASSQ = Autism Spectrum Screening Questionnaire. EF = Executive function, composite score calculated from results on the Letter-Number Sequencing Test from Wechsler Intelligence Scale for Children-IV, the Color-Word Interference Test, Condition 3, and the Trail Making Test, Condition 4, both from Delis-Kaplan Executive Function System. INT = Internalizing and EXT = externalizing symptoms, from Achenbach System of Empirically Based Assessment; Children Behavior Checklist at T1 and T2, Adult Self-Report at T3. Statistically significant correlations are identified by *$p < .05$, ** $p < .01$, *** $p < .001$.

Table 3. Baseline predictors of internalizing symptoms at baseline, 2-year follow-up, and 10-year follow-up.

	Baseline (T1) $N = 83$				2-year follow-up (T2) $N = 81$				10-year follow-up (T3) $N = 62$			
Predictors from T1	*B*	95% CI	*SE*	ΔR^2	*B*	95% CI	*SE*	ΔR^2	*B*	95% CI	*SE*	ΔR^2
Step 1				.007				.012				.024
Age	.573	[−.986, 2.133]	.784		.705	[−.737, 2.148]	.725		.914	[−.603, 2.431]	.758	
Step 2				.587				.452				.144
Age	−.742	[−1.776, .292]	.520		−.129	[−1.218, .960]	.547		.383	[−1.068, 1.835]	.725	
Autism symptoms	.991***	[.808, 1.175]	.092		.763***	[.576, .951]	.094		.424**	[.159, .689]	.133	
Step 3				.004				.033				.125
Age	−.481	[−1.657, .695]	.591		.624	[−.634, 1.881]	.631		1.634*	[.074, 3.194]	.779	
Autism symptoms	.942***	[.729, 1.154]	.107		.650***	[.441, .859]	.105		.223	[−.054, .499]	.138	
Executive function	.385	[−.439, 1.209]	.414		.946*	[.101, 1.791]	.424		1.847**	[.693, 3.002]	.577	

Note. Dependent variable = internalizing symptoms, Achenbach System of Empirically Based Assessment; Children Behavior Checklist at T1 and T2, Adult Self-Report at T3. Autism symptoms = Autism Spectrum Screening Questionnaire. Executive function = composite score calculated from results on the Letter-Number Sequencing Test from Wechsler Intelligence Scale for Children-IV, the Color-Word Interference Test, Condition 3, and the Trail Making Test, Condition 4, both from Delis-Kaplan Executive Function System. T1 $N = 83$, T2 $N = 81$, T3 $N = 62$. *$p < .05$, **$p < .01$, ***$p < .001$.

Predictors of internalizing symptoms

Table 3 displays results from the three hierarchical regression analyses with internalizing symptoms as outcome variables. At T1, autism symptoms explained 58.7% of the variance in internalizing symptoms ($\Delta R^2 = .587$, $\Delta F = 115.31$, $p < .001$), while EF non-significantly explained an additional 0.4% of the variance ($\Delta R^2 = .004$, $\Delta F = 0.87$, $p = .355$). At T2, baseline autism symptoms explained 45.2% of the variance in internalizing symptoms ($\Delta R^2 = .452$, $\Delta F = 65.74$, $p < .001$), while EF explained an additional 3.3% of the variance ($\Delta R^2 = .033$, $\Delta F = 4.97$, $p = .029$). At T3, baseline autism symptoms explained 14.4% of the variance in internalizing symptoms ($\Delta R^2 = .144$, $\Delta F = 10.22$, $p = .002$). Baseline autism symptoms were no longer a significant predictor when adding baseline EF to the model ($p = .113$), whereas EF significantly predicted and accounted for an additional 12.5% of the variance in internalizing symptoms ($\Delta R^2 = .125$, $\Delta F = 10.26$, $p = .002$). When controlling for mother's education in Model 1 in the regression analysis at T1, T2, and T3, all results remained the same (data not shown).

Table 4. Baseline predictors of externalizing symptoms at baseline, 2-year follow-up, and 10-year follow-up.

	Baseline (T1) $N = 83$				2-year follow-up (T2) $N = 81$				10-year follow-up (T3) $N = 62$			
Predictors from T1	*B*	95% CI	*SE*	ΔR^2	*B*	95% CI	*SE*	ΔR^2	*B*	95% CI	*SE*	ΔR^2
Step 1				.010				.011				.001
Age	.601	[−.697, 1.899]	.652		.434	[−.502, 1.369]	.470		−.139	[−1.468, 1.189]	.664	
Step 2				.535				.324				.108
Age	−.447	[−1.358, .464]	.458		−.024	[−.810, .761]	.395		−.537	[−1.837, .763]	.650	
Autism symptoms	.789***	[.628, .951]	.081		.419***	[.284, .554]	.068		.317**	[.080, .555]	.119	
Step 3				.002				.008				.154
Age	−.290	[−1.329, .749]	.522		.217	[−.713, 1.147]	.467		.663	[−.716, 2.042]	.689	
Autism symptoms	.760***	[.572, .947]	.094		.383***	[.228, .537]	.078		.124	[−.120, .369]	.122	
Executive function	.231	[−.497, .960]	.366		.304	[−.322, .929]	.314		1.772***	[.752, 2.793]	.510	

Note. Dependent variable = externalizing symptoms, Achenbach System of Empirically Based Assessment, Children Behavior Checklist at T1 and T2, Adult Self-Report at T3; Autism symptoms = Autism Spectrum Screening Questionnaire. Executive function composite score calculated from results on the Letter-Number Sequencing Test from Wechsler Intelligence Scale for Children-IV, the Color-Word Interference Test, Condition 3, and the Trail Making Test, Condition 4, both from Delis-Kaplan Executive Function System. T1 $N = 83$, T2 $N = 81$, T3 $N = 62$. *$p < .05$, **$p < .01$, ***$p < .001$.

Predictors of externalizing symptoms

Table 4 displays results from the three hierarchical regression analysis with externalizing symptoms as outcome variables. At T1, autism symptoms explained 53.5% of the variance in externalizing symptoms ($\Delta R^2 = .535$, $\Delta F = 94.17$, $p < .001$), while EF non-significantly explained an additional 0.2% of the variance ($\Delta R^2 = .002$, $\Delta F = .40$, $p = .529$). At T2, baseline autism symptoms were a significant predictor and explained 32.4% of the variance in externalizing symptoms ($\Delta R^2 = .324$, $\Delta F = 38.06$, $p < .001$), while the EF non-significantly accounted for an additional 0.8% of the variance ($\Delta R^2 = .008$, $\Delta F = 0.94$, $p = .337$. At T3, baseline autism symptoms predicted and explained 10.8% of the variance in externalizing symptoms ($\Delta R^2 = .108$, $\Delta F = 7.14$, $p = .010$), but was no longer a significant predictor when adding baseline EF to the model ($p = .314$), the latter explained an additional 15.4% of the variance ($\Delta R^2 = .154$, $\Delta F = 12.08$, $p < .001$). When controlling for mother's education in Model 1 in the regression analysis at T1, T2, and T3, all results remained the same (data not shown).

A pathway from autism symptoms to later co-occurring symptoms via executive function

The results of the post-hoc simple mediation analyses are presented in Figures 1 and 2. We found a statistically significant indirect effect from autism symptoms at T1 to internalizing and externalizing symptoms at T3, via EF at T2. The total models accounted for 27% of the variance in internalizing symptoms ($F(3, 57) = 7.01$, $p < .001$) and 31% of the variance in externalizing symptoms ($F(3, 57) = 8.62$, $p < .001$). When adding EF at T2 as a mediator, we did not find a direct effect of autism symptoms on internalizing or externalizing symptoms 10 years later, suggesting full mediation.

Internalizing and externalizing symptoms as predictors of later executive function. Results from the additional exploratory regression analyses with EF composite at T2 and T3 as outcome variables are displayed in Supplemental Tables 2 and 3. When controlling for age and autism symptoms, internalizing symptoms at T1 were not a significant predictor of later EF at T2 nor at T3. Externalizing symptoms at T1 were a statistically significant predictor of EF at T2 when controlling for age and autism symptoms ($p = .018$), but not a significant predictor of EF at T3.

Discussion

We investigated EF as a concurrent and longitudinal predictor of internalizing and externalizing symptoms in individuals with ASD and TD individuals. EF at age 12 years was a significant predictor for internalizing symptoms both 2 and 10 years later, explaining 3.3% and 12.5% of the variance, respectively. Correspondingly, baseline EF predicted externalizing symptoms after 10 years, explaining

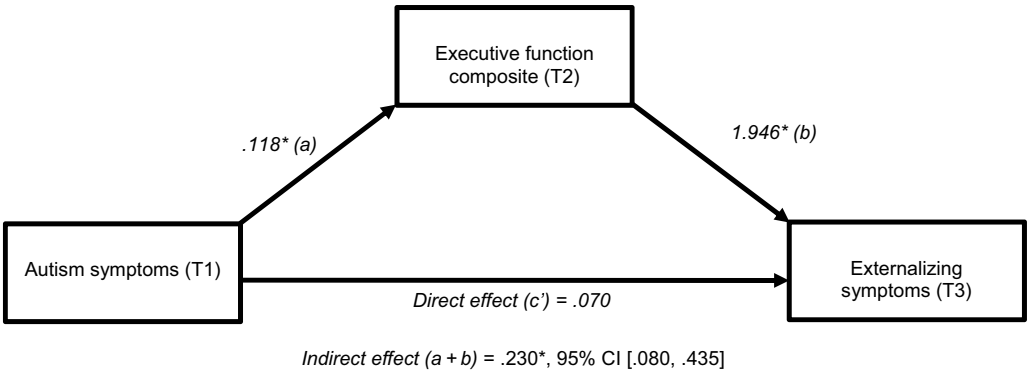

Figure 1.

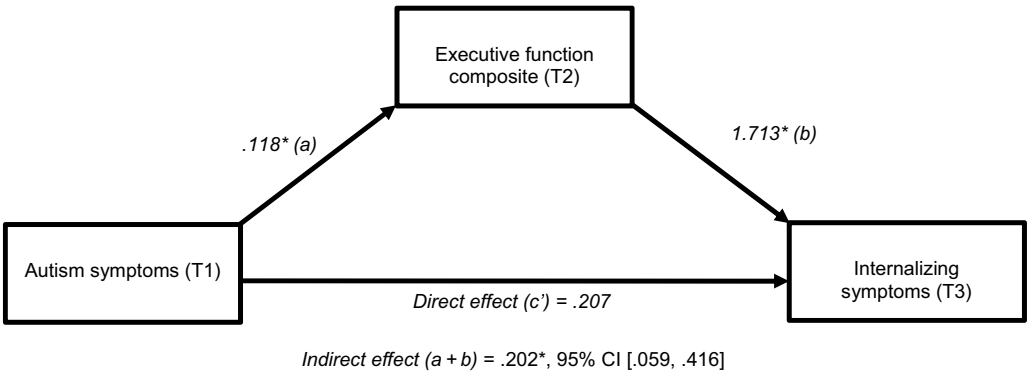

Figure 2. Internalizing and externalizing symptoms as predictors of later executive function.

15.4% of the variance. Our exploratory analyses also indicated that externalizing symptoms at age 12 years predicted poorer EF 2 years later.

In contrast to the findings where difficulties with cognitive flexibility predicted more emotional symptoms in autistic children and adolescents concurrently (Hollocks et al., 2021), we did not find that EF predicted more co-occurring symptoms cross-sectionally. Some differences between Hollocks' and our study may be relevant to understand this divergence. For instance, they used a wider IQ range, and their participants were on average older. We find it likely that our high correlation (>.70) between baseline autism symptoms (predictor) and internalizing symptoms (outcome) may camouflage a cross-sectional impact of EF on internalizing symptoms, as autism symptoms were entered before EF in our analysis. The correlation between autism symptoms and emotional symptoms in Hollocks' sample was lower at .38 (Hollocks et al., 2021). Further, it should be noted that Hollocks et al. (2021) only controlled for one aspect of autism symptoms (restricted and repetitive behaviors), whereas we used a broader measure of autism symptoms, including social communication difficulties. Social communication difficulties may be more closely tied to co-occurring symptoms than restricted and repetitive behaviors.

Longitudinally, more EF difficulties predicted higher levels of self-reported internalizing and externalizing symptoms in the emerging adults with and without ASD. Our findings resemble those where cognitive inflexibility at age 16 predicted emotional and behavioral symptoms at age 23 in individuals with ASD (Hollocks et al., 2021), and support the idea of EF as a mechanism behind a general difficulty in emotional and behavioral regulation (Conner et al., 2020). Our findings are also

in line with findings from clinical and non-clinical samples of children/adolescents where better EF predicted less internalizing and externalizing symptoms longitudinally (Yang et al., 2022).

Further, our findings indicate that the EF difficulties may pose a risk for developing co-occurring internalizing and externalizing symptoms in the long run, above the impact of early autism symptoms. The substantial burden of co-occurring symptoms, and their link to reduced quality of life in adults with ASD (Andersen et al., 2023; Ayres et al., 2018; Mason et al., 2018) underline the importance of our findings. Our data imply that the predictive influence of EF applies to both internalizing and externalizing symptoms 10 years later. Difficulties with EF have been proposed to be an indicator of atypical development more generally, applying across neurodevelopmental and other disorders (Zelazo, 2020). The longitudinal impact of childhood EF on later co-occurring symptoms has previously been demonstrated in individuals with and without ADHD (Orm et al., 2022).

The results from our regression analysis suggested that there might be a mediating effect of EF on the relationship between early autism symptoms and later co-occurring symptoms, because autism symptoms no longer predicted later internalizing and externalizing symptoms when we added EF to the model. The post-hoc simple mediation model supported this notion, giving evidence that EF at T2 acted as a mediator. These additional findings lend support to our main findings that EF may act as a vulnerability factor for developing co-occurring internalizing and externalizing symptoms.

In turn, our findings support the notion of EF difficulties as a risk factor for co-occurring symptoms, potentially via poorer attentional, emotional, and behavioral control (Nelson et al., 2019; Zelazo, 2020). EF difficulties could influence several developmental areas that are important for mental health, and could thereby influence both the development and maintenance of co-occurring symptoms. Such areas include education and occupation, social relationships, independent living and economic management, among others. One potential pathway from early EF difficulties to later co-occurring symptoms is via experiences of struggling or failing in academic or occupational arenas accumulated over the years, as suggested in ADHD studies (Owens & Hinshaw, 2016). In general, EF difficulties are associated with lower academic achievement and difficulties in finding and keeping a job (Diamond, 2013). When children/adolescents grow older, expectations of independence and functional demands increase, while external support typically decreases (Turgay et al., 2012). The gap between the environmental expectations and the actual daily functioning in individuals with ASD may increase over time, possibly influenced by long-lasting EF difficulties (Pugliese et al., 2016).

Another potential pathway from EF to co-occurring symptoms is via social function or social vulnerability. Everyday EF difficulties are associated with lower social function in children/adolescents with ASD (Leung et al., 2016; Torske et al., 2018), and everyday EF difficulties have been identified as a predictor of being bullied in adolescents with and without ASD (Kloosterman et al., 2014). Relatedly, findings indicate that children/adolescents with EF difficulties may be more vulnerable and at risk for victimization (Op den Kelder et al., 2022).

Furthermore, EF may be linked to later co-occurring symptoms via difficulties with emotion regulation (Nelson et al., 2019). EF is considered important for regulating one's thoughts, emotions, and actions (Diamond, 2013). Regulating one's emotions in response to dealing with daily life or larger life events (i.e., coping/mastery) is important for whether or not someone develops psychopathology (Compas et al., 2017). Emotion regulation has been identified as a risk factor for anxiety in populations with ASD (White et al., 2014). Relatedly, EF is vital for the occurrence and management of life stress (Williams et al., 2009). For instance, people with EF difficulties can have difficulties planning and organizing tasks, leading to a feeling of being overwhelmed (Williams et al., 2009). Increased stress can in turn can have a negative impact on mental health over time (Williams et al., 2009). EF can thereby be related to co-occurring symptoms via its impact on emotion regulation and stress.

Regarding the levels of internalizing and externalizing symptoms in our sample, we note that nearly all the mean scores for the two groups across time are within what is typically denoted as the normal range (T score<65). Importantly, Norway is a low-scoring society on CBCL, with mean scores more than 1 SD below the overall mean for Total Problems (Jozefiak et al., 2012; Rescorla et al., 2007). We

interpret the scores in the ASD group as elevated relative to the scores in the TD group, which were well below 50 with mean scores ranging from 39 to 45.

We investigated the impact of early EF on later co-occurring symptoms in this study, but the relationship could also go the other way around or work bi-directionally (Romer & Pizzagalli, 2021). In our additional exploratory analyses, we observed that externalizing symptoms at baseline were a significant predictor for EF 2 years later, but not 10 years later, suggesting a lack of stability of these findings. Internalizing symptoms at baseline did not predict EF 2 or 10 years later. We believe these findings strengthen our interpretation where EF difficulties in childhood pose a risk for more co-occurring symptoms in emerging adulthood, but less so the other way around.

With the early EF explaining between 12 and 15% of the variance in internalizing and externalizing symptoms after 10 years, there is still much variance left unexplained. Nevertheless, we consider this as a substantial contribution, and it is striking when comparing to autism symptoms as a predictor, which after adding EF no longer explained a statistically significant amount of variance in co-occurring symptoms. The amount of variance explained by EF at T3 corresponds to a Cohen's f^2 of .14 for internalizing symptoms and .17 for externalizing symptoms, which indicated small/medium and medium effects, respectively (Cohen, 1992).

Strengths and limitations

The three assessment waves over the relatively long follow-up interval are important strengths of this study. Another asset is the use of standardized neuropsychological tests for assessing EF, which eliminates the influence of a possible negativity bias in parents (Hollocks et al., 2021). We also consider it a strength that we used a composite score of EF, and the high correlations indicate that the composite scores were a good fit for the three EF scores (Supplemental Table 1).

Some limitations should also be noted. Our participants with ASD were recruited from a clinically referred population, and the sample was restricted to individuals with IQ above 70, which reduces the generalizability of the findings. However, shedding light on the later outcomes for individuals from this population may be important in itself. Another issue is that the T3 participants had better EF than the participants from baseline who opted out of the study, and thus may not be representative of individuals with and without ASD. Ideally, the number of participants with ASD, and the retention rates in the ASD group, would have been higher. The use of parent-rated symptom measures at T1 and T2 may be a limitation, especially relevant for internalizing symptoms, as parents rarely have complete access to the inner lives of their children. We know from this sample that the parent-reports of depressive symptoms at T1 and T2 were not significantly correlated with corresponding child reports (Andersen et al., 2017). A related issue is that the same informants (parents) at T1 and T2 filled out the scales of autism and co-occurring symptoms. High associations between autism and rating scales of co-occurring symptoms could have camouflaged a predictive effect of EF on concurrent externalizing symptoms two years later. This within-rater issue (Hollocks et al., 2021) was not present in the 10-year analysis, where symptoms were self-reported. Although it is important to keep in mind the shift in informant when interpreting our findings from T2 versus T3, we consider it a strength that symptom reports came from the participants themselves at T3. Some might argue that we should have controlled for IQ given that IQ and EF are strongly related; however, we decided not to, because doing so could lead to overcorrecting (Dennis et al., 2009).

Clinical and scientific implications

Our findings suggest that over time it is not the level of autism symptoms, but rather the EF difficulties that increase the risk of co-occurring symptoms. This finding highlights the importance of assessing cognitive functioning as part of the diagnostic evaluation. By doing so, we can identify individuals with EF difficulties, for whom support to improve adaptive functioning despite the cognitive vulnerability

could be crucial. Providing such support could potentially have a protective effect on later mental health, and should supplement the support offered for potential challenges related to core autism characteristics. We suggest that future studies should delve into the possible pathways from EF difficulties to later co-occurring symptoms.

Note

1. Internal consistency estimates for the ASSQ, CBCL, and ASR measures could not be calculated in this study, as due to the clinical nature of the study, we only have access to the sum scores.

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No potential conflict of interest was reported by the authors.

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Ethical approval

This study was prospectively reviewed and approved by the Regional Committee for Medical Research Ethics in Eastern Norway (T1-T2: REK 6-2009-24; T3: 2018/1611) and the Privacy Ombudsman for Research at Inlandet Hospital Trust. The study was conducted in accordance with the Helsinki Declaration of the World Medical Association Assembly.

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