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Transdiagnostic neurocognitive compensatory strategies

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ABSTRACT

Background: Some neurodivergent children, such as those with autism spectrum disorder (ASD) or attention-deficit hyperactivity disorder (ADHD) diagnoses, may "compensate" by using executive function skills to support social behaviors despite underlying difficulties in social cognition. However, it is unknown whether compensation (e.g., demonstrating real-world adaptive social behavior despite social cognition difficulties) is transdiagnostic across neurodivergent and neurotypical (NT) development, and whether compensation is evident with meaningful discrepancies between neurobiological, cognitive, and behavioral factors.
Methods: A total of 74 children between 7–12 years-old with diagnostic classification of ASD ($n = 33$), ADHD ($n = 10$), ASD+ADHD ($n = 20$), and NT ($n = 11$) completed a range of neural, cognitive, and behavioral tasks. Children were assigned compensation group status; High Compensators demonstrated more real-world social skills despite spontaneous social cognition difficulties, while Low Compensators demonstrated similar social cognition difficulties accompanied by fewer real-world social skills.
Results: Compared to Low Compensators, High Compensators demonstrated better neurocognitive and electrophysiological inhibition including conflict monitoring, response inhibition, shorter stop-signal reaction times in a Stop-Change Task, and smaller N2 and larger P3 amplitudes in the No-go condition of a cued event-related potential Go/Nogo task. High Compensators did not differ from Low Compensators in intelligence, internalizing and externalizing symptoms, caregiver-reported behavioral regulation, or proportion of diagnostic group classification.
Conclusions: Adaptive social skills behavior despite underlying challenges with spontaneous social cognition may be supported by stronger neurocognitive and electrophysiological executive function skills (i.e., social compensation). Transdiagnostic neurocognitive compensatory strategies are consistent with strength-based and developmental models of neurodiversity and advance the field of neurodiversity.

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1. Transdiagnostic neurocognitive compensatory strategies

Why do some neurodivergent individuals diagnosed with autism spectrum disorder (ASD) and attention-deficit hyperactivity disorder (ADHD) adapt socially despite social cognition difficulties? Are there individual differences in how neurodivergent children approach social situations to utilize neurocognitive compensatory strategies? To advance the field of neurodiversity, we aim to combine neurodiversity-affirmative perspectives that focus on identifying positive factors of neurodivergent individuals (Najeeb & Quadt, 2024) with a developmental framework. This approach is used to identify candidate mechanisms that may make a difference in promoting resilience across neurobiological, cognitive, and behavioral levels of analysis (Altschuler et al., 2026; Altschuler et al., in press; Cicchetti, 1984; Masten, 2024). We leverage developmentally sensitive cognitive neuroscience methods (Abdul Rahman et al., 2017; Hoyniak, 2017) and participatory research methods (Fletcher-Watson et al., 2019) in neurodiversity studies to address: Are some neurodivergent children adapting to "neuronormative" society (Wise, 2023), exhibiting real-world socially adaptive behavior to act in neurotypical ways while, beneath the behavioral surface, there is evidence of transdiagnostic neurocognitive compensatory strategies at neurobiological and neurocognitive levels of analysis?

From a neurodiversity perspective, compensation for cognitive difficulties through cognitive strategies could be viewed as a potential advantage of having neurological differences (Rządeczka et al., 2023). Compensation, referred to within the field of neurodevelopmental conditions as related to camouflaging or masking (Lai et al., 2016; Livingston et al., 2019; Petrolini et al., 2023), is hypothesized to explain a mismatch between adaptive social skills behavior and the pertinent underlying social cognitive skill. For example, social behavioral presentation may be more socially adaptive than would have otherwise been expected based on underlying social cognitive abilities, such as being more flexible in real-world social situations than cognitive skills would predict through use of alternative skills. Children with neurodevelopmental conditions who are social compensators have been found to have enhanced executive function abilities (Hull et al., 2021; Livingston et al., 2018) at the potential "cost" of worse mental health symptoms (Miller et al., 2021), suggesting that enhanced inhibition may be an individual difference factor that facilitates social compensation for some children with adaptive social behavior despite underlying social cognition difficulties. Inhibition may support social adaptation by allowing children to suppress prepotent but socially incongruent responses, attention to irrelevant social cues, or their own perspective in order to take the perspective of a social partner. Thus, children with stronger inhibition may sometimes show more neurotypical-appearing social behavior to adapt to neuronormative expectations (Wise, 2023) even when spontaneous social cognition is more difficult for them.

Given considerable symptom overlap between neurodevelopmental conditions (Martinez et al., 2024; Shaw, 2024) and dimensional neurodiversity approaches (Altschuler & Krueger, 2021; Altschuler & Lyons, 2024; Kotov et al., 2018; Krueger et al., 2018; Michelini et al., 2021), a transdiagnostic study of compensation across neurodivergent conditions is needed to clarify compensatory processes. We used measures of social cognition and real-world adaptive social behavior to define social compensators (i.e., children with more adaptive real-world social behavior despite more difficulties with social cognition). We aim to replicate the existing autism social compensation behavior-cognition discrepancy literature and extend this work using transdiagnostic, neurocognitive methods to answer the question: Do neurodivergent children with adaptive real-world social behavior despite social cognition difficulties show enhanced neurocognitive inhibition across brain, cognition, and behavior?

2. Methods

2.1. Language and Positionality

We acknowledge variation in preferences for language and selected to use terms consistent with the preferences identified by many neurodivergent individuals (Keating et al., 2023). Higher social cognition scores are discussed as having fewer social cognition difficulties, while higher caregiver-reported social autistic traits scores are discussed as having less neurotypical social behavior and thus less real-world adaptive social behavior and fewer social skills, exhibiting more social challenges in a neuronormative society with structures often designed to normalize neurotypical ways of acting (Finke & Dunn, 2025; Wise, 2023). Participatory research methods were employed (Fletcher-Watson et al., 2019); the first author has been diagnosed with and treated for the neurodivergent conditions examined in the present study throughout her childhood and identifies as a social compensator.

2.2. Participants and procedure

As part of a larger clinical trial for children with an autism spectrum disorder (ASD) diagnosis (Faja et al., 2021), and a study of executive function for children with an attention deficit/hyperactivity disorder (ADHD) diagnosis or neurotypical (NT) development (Cremone-Caira et al., 2021), participants were recruited from a participant registry in a hospital setting and from community sources. Participant eligibility and diagnostic classification were based on criteria outlined by Cremone-Caira et al. (2021). ASD diagnoses were confirmed by a licensed clinical psychologist using the Autism Diagnostic Observation Schedule, Second edition (ADOS-2; Lord et al., 2012), Autism Diagnostic Interview-Revised (ADI-R; Rutter et al., 2003) according to Collaborative Programs of Excellence in Autism criteria (Sung et al., 2005), and the Diagnostic and Statistical Manual of Mental Disorders, Fifth edition (DSM-5; American Psychiatric Association, 2013) criteria. ADHD diagnoses were determined using a process aligned with literature (Andersen et al., 2013; Cremone-Caira et al., 2024, 2019, 2021). For children with an existing community ADHD diagnosis, diagnostic status was confirmed using caregiver ratings on the Conners-3 Comprehensive Behavior Ratings Scales (Conners, 2008), applying DSM-based count crtieria of >= 6 symptoms on the Inattention and/or Hyperactivity-Impulsivity scales. In addition, continuous ADHD symptoms were also assessed across all groups using the ADHD subscale of the Child Behavior Checklist (CBCL; Achenbach & Rescorla, 2001). Following Cremone-Caira et al. (2021) and Andersen et al. (2013), children with confirmed ASD diagnoses and CBCL t-scores >= 65 on the ADHD subscale were classified as having clinically significant ADHD symptoms and included in the ASD+ADHD group.

Participants of the present study included 74 children (6 girls), 7–12 years-old with IQs of 80 or above, verified with the Wechsler Abbreviated Scale of Intelligence, Second Edition (WASI-2; Wechsler, 2011), and categorical diagnostic classification of ASD ($n = 33$), ADHD ($n = 10$), ASD+ADHD ($n = 20$), and NT ($n = 11$) (see Table 1 for sample characteristics).

Table 1
Participant demographic characteristics and comparisons for high versus low compensation groups.

	High Compensation (N = 14)	**Low Compensation (N = 29)**	**Overall (N = 43)**	t
Age				
Mean (SD)	96.9 (12.5)	110 (16.7)	106 (16.6)	$F(1, 42)= 7.3$
Median [Min, Max]	90.0 [86.0, 123]	110 [85.0, 144]	104 [85.0, 144]	($p = 0.010$)
Sex				
Male	14 (100 %)	25 (86.2 %)	39 (90.7 %)	$\chi^2(1)= 0.8$
Female	0 (0 %)	4 (13.8 %)	4 (9.3 %)	($p = 0.369$)
Diagnostic Group				
ADHD	2 (14.3 %)	2 (6.9 %)	4 (9.3 %)	
ASD	7 (50.0 %)	14 (48.3 %)	21 (48.8 %)	$\chi^2(3)= 4.0$
ASD + ADHD	2 (14.3 %)	11 (37.9 %)	13 (30.2 %)	($p = 0.259$)
NT	3 (21.4 %)	2 (6.9 %)	5 (11.6 %)	
Race				
American Indian/Alaskan Native	0 (0.0 %)	0 (0.0 %)	0 (0.0 %)	
Asian	0 (0.0 %)	0 (0.0 %)	0 (0.0 %)	
Hawaiian/Pacific Islander	0 (0.0 %)	0 (0.0 %)	0 (0.0 %)	
Black/African American	1 (7.1 %)	2 (6.9 %)	3 (7.0 %)	
White/Caucasian	10 (71.4 %)	21 (72.4 %)	31 (72.1 %)	$\chi^2(4)= 0.8$
More than one race	2 (14.3 %)	4 (13.8 %)	6 (14.0 %)	($p = 0.943$)
Unknown/Not reported	1 (7.1 %)	1 (3.4 %)	2 (4.7 %)	
Other	0 (0 %)	1 (3.4 %)	1 (2.3 %)	
Ethnicity				
Non-Latino/Hispanic	13 (92.9 %)	24 (82.8 %)	37 (86.0 %)	$\chi^2(1)= 0.2$
Latino/Hispanic	1 (7.1 %)	5 (17.2 %)	6 (14.0 %)	($p = 0.670$)
Annual Household Income				
Less than $20,000	0 (0 %)	3 (10.3 %)	3 (7.0 %)	
$21,000 - $35,000	2 (14.3 %)	3 (10.3 %)	5 (11.6 %)	
$36,000 - $50,000	1 (7.1 %)	2 (6.9 %)	3 (7.0 %)	
$51,000 - $65,000	2 (14.3 %)	0 (0 %)	2 (4.7 %)	
$66,000 - $80,000	1 (7.1 %)	0 (0 %)	1 (2.3 %)	$\chi^2(9)= 13.0$
$81,000 - $100,000	0 (0 %)	7 (24.1 %)	7 (16.3 %)	($p = 0.164$)
$101,000 - $130,000	1 (7.1 %)	4 (13.8 %)	5 (11.6 %)	
$131,000 - $160,000	2 (14.3 %)	3 (10.3 %)	5 (11.6 %)	
Over $160,000	5 (35.7 %)	6 (20.7 %)	11 (25.6 %)	
Unknown/Not reported	0 (0 %)	1 (3.4 %)	1 (2.3 %)	
Primary Caregiver Education				
Completed less than 9th grade	0 (0.0 %)	0 (0.0 %)	0 (0.0 %)	
Completed 9th grade	0 (0.0 %)	0 (0.0 %)	0 (0.0 %)	
Some high school (no diploma)	0 (0.0 %)	0 (0.0 %)	0 (0.0 %)	
GED diploma	0 (0 %)	1 (3.4 %)	1 (2.3 %)	
High school graduate	1 (7.1 %)	1 (3.4 %)	2 (4.7 %)	$\chi^2(4)= 3.0$
Associate degree (2 year degree)	2 (14.3 %)	10 (34.5 %)	12 (27.9 %)	($p = 0.553$)
Some college	5 (35.7 %)	6 (20.7 %)	11 (25.6 %)	
BA/BS degree (4 year degree)	6 (42.9 %)	11 (37.9 %)	17 (39.5 %)	
Graduate/professional degree	0 (0.0 %)	0 (0.0 %)	0 (0.0 %)	
CBCL ADHD T Score				
Mean (SD)	57.1 (8.03)	65.0 (8.44)	62.4 (9.03)	$F(1, 42)= 8.6$
Median [Min, Max]	54.5 [50.0, 72.0]	62.0 [50.0, 80.0]	60.0 [50.0, 80.0]	($p = 0.006$)
SRS SCI T Score				
Mean (SD)	54.6 (5.42)	70.7 (7.89)	65.5 (10.5)	$F(1, 42)= 47.5$
Median [Min, Max]	56.0 [39.0, 59.0]	70.0 [60.0, 83.0]	64.0 [39.0, 83.0]	($p < 0.001$)
SAT Score				
Mean (SD)	0.214 (0.0770)	0.200 (0.104)	0.205 (0.0950)	$F(1, 42)= 0.2$
Median [Min, Max]	0.200 [0.100, 0.300]	0.200 [0, 0.300]	0.200 [0, 0.300]	($p = 0.650$)
ADOS Total Severity Score				
Mean (SD)	8.67 (1.87)	9.16 (1.28)	9.03 (1.45)	$F(1, 37)= 0.8$
Median [Min, Max]	10.0 [5.00, 10.0]	10.0 [5.00, 10.0]	10.0 [5.00, 10.0]	($p = 0.388$)
Missing	5 (35.7 %)	4 (13.8 %)	9 (20.9 %)	

ASD = autism spectrum disorder, ADHD = attention-deficit hyperactivity disorder, NT = neurotypical development, CBCL = Child Behavior Checklist, SRS SCI = Social Responsiveness Scale Social Communication/Interaction index, SAT = Social Attribution Task, ADOS = Autism Diagnostic Observation Schedule

2.3. Measures

2.3.1. Intellectual ability

The Wechsler Abbreviated Scale of Intelligence, Second Edition (WASI-2; Wechsler, 2011) estimates intelligence quotient (IQ) for 6–90 year-olds and consists of four subtests. The Vocabulary and Similarities subtests comprise a Verbal Comprehension Index, assessing verbal, crystallized ability, which is used to index verbal IQ (VIQ). The Block Design and Matrix Reasoning subtests comprise a Perceptual Reasoning Index, assessing nonverbal, fluid ability, which is used to index nonverbal IQ (NVIQ). Larger FSIQ, VIQ, and NVIQ scores indicated higher overall, verbal, and non-verbal intelligence, respectively.

2.3.2. Internalizing and externalizing symptoms

The Achenbach System of Empirically Based Assessment (ASEBA; Achenbach & Rescorla, 2001) school-aged Child Behavior Checklist (CBCL 6–18; caregiver report) was used to measure internalizing and externalizing symptoms. Higher CBCL internalizing and externalizing symptoms t-scores indicated more difficulties with real-world internalizing and externalizing symptoms.

2.3.3. Real-world adaptive social behavior

The Social Responsiveness Scale-Parent Report (Constantino et al., 2003) (SRS) is a 65-item caregiver-report questionnaire that measures the degree of observable autistic traits as they occur in real-world social settings. The SRS SCI t-score is calculated by combining raw scores from the Awareness, Cognition, Communication, and Motivation subscales and converting the total into the normed SRS SCI t-score. The SRS SCI t-score reflects the degree to which social communication and interaction difficulties are observed in everyday contexts. Although the SRS is often conceptualized as an index of autistic traits, in the present study we interpret lower SRS SCI t-scores (fewer observable autistic traits) as indicating greater alignment of children's social behavior with neurotypical exectations in real-world contexts, and higher SRS SCI t-scores (more observable autistic traits) as indicating less alignment with these neurotypical expectations. We use the term "adaptation" to refer specifically to adaptation to neuronormative (Wise, 2023) social demands, rather than to imply optimal functioning.

2.3.4. Spontaneous social cognition

In the Social Attribution Task (SAT; Klin, 2000), participants viewed socially animated geometrical figures (Heider & Simmel, 1944) and were asked to describe the meaning of the animation. The SAT video clips, instructions, and coding scheme were identical to Altschuler et al. (2018), with scores showing strong test-retest reliability (Altschuler & Faja, 2022). Higher SAT scores indicated increased spontaneous social cognition.

2.3.5. Real-world behavioral inhibition

Caregiver-report of real-world executive function was obtained with the Behavior Rating Inventory of Executive Function questionnaire (BRIEF; Gioia et al., 2000). The BRIEF assesses inhibition, self-monitoring, shifting, emotional control, initiation, working memory, planning/organizing, task monitoring, and organization of materials in a day-to-day context. Higher BRIEF Behavioral Regulation index t-scores indicated more real-world self-regulation difficulties (i.e., reduced real-world behavioral inhibition).

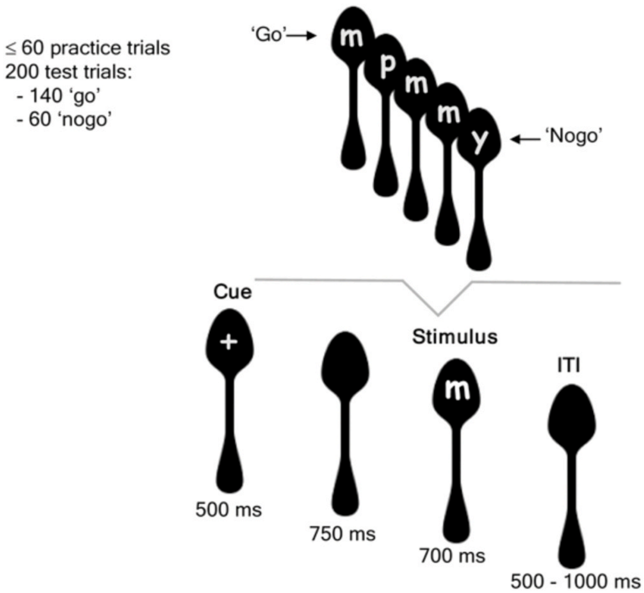

Fig. 1. Schematic of the Go/Nogo task from Cremone-Caira et al. (2021).

2.3.6. Electrophysiological neural inhibition

A cued Go/Nogo task was used to elicit the N2 and P3 event-related potential (ERP) components as electrophysiological indices of inhibition (Fig. 1). During the 'go' trials, children were told to press a single button on the keypad each time a letter was presented on screen. During the 'nogo' trials, children were told to withhold their response when a specific letter appeared on the screen. Only ERPs for correct trials that followed a correct 'go' response were analyzed to ensure consistent motor response on the previous trial. EEG data recording, editing, and abstraction followed that of our past work (Cremone-Caira et al., 2020; Faja et al., 2021, 2016) and are detailed in Supplemental Materials. The P3 and N2 ERP components from the incongruent conditions were analyzed as indices of neural inhibition. Larger P3 mean amplitude scores indicate increased neural response inhibition, while smaller N2 mean amplitude scores indicated less neural effort while conflict monitoring.

2.3.7. Neurocognitive response inhibition

The Stop-Change Task (De Jong et al., 1995) was used to measure neurocognitive inhibition. Following practice, four test blocks included Go trials (75 %) and Change trials (25 %), which were comprised of a visual signal to stop the dominant task (i.e., left/right button press) and change to the spacebar. The dependent variable of the Stop-Change Task was stop signal reaction time (SSRT), which estimates the latency required to inhibit a dominant response when a stop signal was presented (Band et al., 2003; Crone & van der Molen, 2004). Higher SSRT scores indicated slower inhibition and shifting to the change response (i.e., more difficulty with neurocognitive response inhibition).

2.4. Group creation

Based on best practice in cognition-behavior discrepancy compensation literature (Livingston et al., 2018), a median split approach was used to create primary *a priori* groups of interest for comparison (Fig. 2). Participants were split on the SAT median, creating groups with higher (>.3) and lower (<=.3) SAT scores, and the clinical cutoff for SRS SCI, forming groups with higher (>59) and lower (<=59) SRS SCI t-scores. The Unknown and Deep Compensation groups were excluded, and the *a priori* High Compensation ($N = 14$; low SRS SCI t-scores, low SAT scores) and Low Compensation ($N = 29$; high SRS SCI t-scores, low SAT scores) comprised the final study sample (Fig. 2).

2.5. Analytic approach

Analyses were performed in R Studio Version 1.4. Given this study's primary interest in comparing Low and High Compensators,

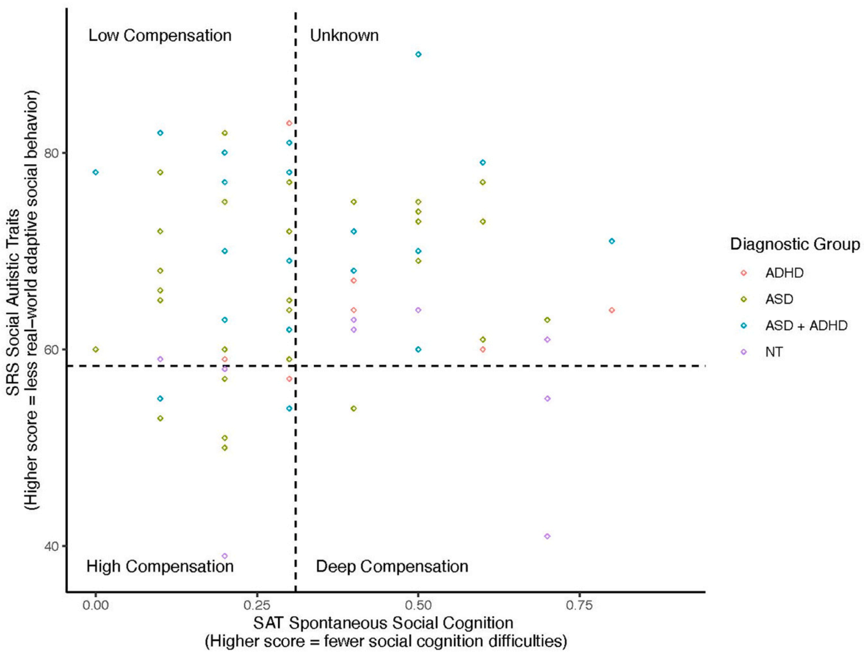

Fig. 2. The relation between spontaneous social cognition performance on the Social Attribution Task (SAT) and real-world adaptive social behavior on the Social Responsiveness Scale (SRS) among children with diagnostic classifications of autism spectrum disorder (ASD), attention-deficit hyperactivity disorder (ADHD), ASD and ADHD, or neurotypical development (NT). Dashed lines represent splits at (a) median spontaneous social cognition score, (b) clinical cut off for SRS SCI (Social Communication/Interaction) t-score, resulting in four quadrants aligned with the approach of Livingston et al. (2018). The two lefthand quadrants (*a priori* High Compensation and Low Compensation groups) were compared for the present study.

separate univariate analysis of covariance (ANCOVA) models statistically controlling for age and SRS SCI t-scores were conducted to examine group differences. Sensitivity analyses were also conducted (Supplemental Materials).

3. Results

Key demographic characteristics are shown in Table 1. The Low and High Compensation groups did not significantly differ in terms of spontaneous social cognition, fulfilling a critical assumption under which the study design lies.

Results of the planned ANCOVA group comparisons between High and Low Compensators while controlling for age and SRS SCI t-scores are shown in Table 2. Effect sizes are reported in terms of partial eta squared. High Compensators had smaller N2 and larger P3 amplitudes in the No-go condition of the cued ERP Go/Nogo task (Fig. 3) and shorter SSRTs in the Stop-Change Task. Groups did not differ significantly in terms of intelligence, internalizing symptoms, externalizing symptoms, or BRIEF behavioral regulation difficulties. The High Compensation and Low Compensation groups did not differ on proportion of diagnostic group classification, $p = .26$, $\chi^2 = 4.0$ (Table 1), although the ADHD subgroup was small ($n = 10$), which limits power to detect diagnostic composition differences.

4. Discussion

Results are consistent with transdiagnostic neurocognitive compensatory strategies in children, advancing the field of neurodiversity to emphasize enhanced cognitive and neural inhibition among social compensators. We demonstrate neurocognitive and electrophysiological evidence of enhanced inhibition in neurodivergent children with real-world adaptive social behavior despite more social cognition difficulties compared to neurodivergent children with equal social cognition difficulties but less real-world adaptive social behavior.

We found inhibition as a potential mechanism underlying real-world social adaptation despite underlying social cognition difficulties. We extend the idea of executive function as a transdiagnostic skill (Zelazo, 2020) to suggest inhibition may be a transdiagnostic compensatory neurocognitive process that may explain the meaningful *discrepancy* between social cognition and social behavior rather than a skill that is universally impaired or enhanced. Indeed, predominant approaches have focused on categorical groups to examine

Table 2
Planned comparisons between low and high compensation groups.

	High Compensation (N = 14)	Low Compensation (N = 29)	Overall (N = 43)	t
CBCL Internalizing T Score				
Mean (SD)	51.5 (8.45)	59.8 (10.8)	57.1 (10.7)	$F(1,39)= 0.63$
Median [Min, Max]	53.0 [34.0, 63.0]	63.0 [34.0, 74.0]	58.0 [34.0, 74.0]	($p = 0.43$); $\eta^2 = .13$
CBCL Externalizing T Score				
Mean (SD)	52.5 (8.86)	56.5 (11.1)	55.2 (10.5)	$F(1,39)= 0.67$
Median [Min, Max]	50.5 [40.0, 70.0]	58.0 [33.0, 74.0]	53.0 [33.0, 74.0]	($p = 0.42$); $\eta^2 = .03$
FSIQ-4				
Mean (SD)	106 (11.5)	106 (15.4)	106 (14.1)	$F(1,39)= 0.002$
Median [Min, Max]	108 [87.0, 126]	106 [80.0, 135]	107 [80.0, 135]	($p = 0.97$); $\eta^2 = .0001$
VCI				
Mean (SD)	106 (14.0)	105 (16.8)	106 (15.8)	$F(1,39)= 0.06$
Median [Min, Max]	103 [79.0, 129]	106 [74.0, 138]	104 [74.0, 138]	($p = 0.82$); $\eta^2 = .0001$
PRI				
Mean (SD)	105 (8.72)	106 (15.6)	106 (13.6)	$F(1,39)= 0.04$
Median [Min, Max]	108 [87.0, 115]	104 [69.0, 139]	105 [69.0, 139]	($p = 0.84$); $\eta^2 = .002$
BRIEF Behavioral Regulation Index				
Mean (SD)	47.6 (12.8)	63.8 (13.6)	58.6 (15.2)	$F(1,39)= 0.11$
Median [Min, Max]	46.0 [30.0, 68.0]	66.0 [29.0, 86.0]	61.0 [29.0, 86.0]	($p = 0.74$); $\eta^2 = .25$
SSRT Mean				
Mean (SD)	198 (74.6)	246 (97.4)	231 (92.8)	$F(1,38)= 11.45$
Median [Min, Max]	200 [31.3, 320]	230 [117, 500]	206 [31.3, 500]	($p = 0.002$)**; $\eta^2 = .06$
Missing	1 (7.1 %)	0 (0 %)	1 (2.3 %)	
N2 Amp GnG				
Mean (SD)	-0.302 (3.28)	-3.02 (2.82)	-2.28 (3.13)	$F(1,22)= 4.68$
Median [Min, Max]	-0.488 [-4.42, 4.57]	-3.26 [-7.48, 1.81]	-1.87 [-7.48, 4.57]	($p = 0.04$)*; $\eta^2 = .15$
Missing	7 (50.0 %)	10 (34.5 %)	17 (39.5 %)	
P3 Amp GnG				
Mean (SD)	6.67 (4.20)	1.52 (2.92)	2.91 (3.97)	$F(1,22)= 8.23$
Median [Min, Max]	5.34 [2.48, 12.6]	1.01 [-2.37, 7.29]	2.69 [-2.37, 12.6]	($p = 0.009$)**; $\eta^2 = .34$
Missing	7 (50.0 %)	10 (34.5 %)	17 (39.5 %)	

CBCL = Child Behavior Checklist, FSIQ = full-scale intelligence quotient, VCI = verbal comprehension index, PRI = perceptual reasoning index, BRIEF = Behavior Rating Inventory of Executive Function, SSRT = stop-signal reaction time, N2 Amp GnG = N2 amplitude on a cued Go/Nogo task, P3 Amp GnG = P3 amplitude on cued Go/Nogo task, η^2 = partial eta squared effect sizes, representing the proportion of variance in the dependent variable (i.e., internalizing symptoms, externalizing symptoms, intelligence, behavioral regulation difficulties, SSRT mean, N2 amplitude, or P3 amplitude) explained by the independent variable (i.e., High Compensators versus Low Compensators) while controlling for covariates (i.e., age and SRS SCI t-scores) in each model, p values: * = <.05, ** = <.01, *** = <.001

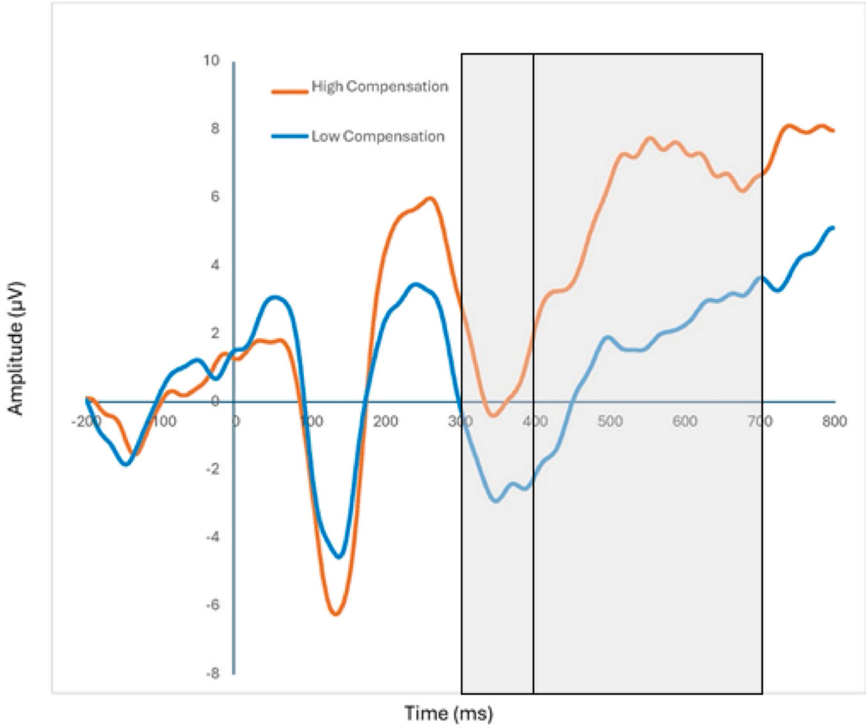

Fig. 3. High Compensation and Low Compensation groups significantly differed in electrophysiological neural inhibition. Event-related potential (ERP) indices of electrophysiological neural inhibition were derived from time windows of 300–400 ms (N2) and 400–700 ms (P3). P3 mean amplitude scores for the High Compensation group ($M = 6.67$) indicated increased neural response inhibition (i.e., better inhibitory control) relative to the Low Compensation group ($M = 1.52$), while N2 mean amplitude scores for the High Compensation group ($M = -.30$) indicated fewer neural resources were required for conflict monitoring relative to the Low Compensation group ($M = -3.02$).

neurodivergent differences in social cognition (e.g., Altschuler et al., 2021; Polónyiová et al., 2024) and unilateral associations between having more observable neurodivergent traits and more difficulties with social cognition (e.g., Altschuler et al., 2018; Moraitopoulou et al., 2024). Our results suggest these approaches may be misguided, obscuring meaningful patterns of individual differences in the discrepancy between observable social behavior and social cognition among children who do not demonstrate the expected social cognition-social behavior association.

While the field of clinical neuroscience has predominantly focused on finding brain-behavior associations (Blanken et al., 2021), future work should prioritize using neurobiological measures to investigate the construct of social compensation (i.e., the clinically meaningful discrepancy between neurobiology and behavior). Our results are the first, to our knowledge, to demonstrate that high compensators have more neural efficiency when responding to conflicting information. We found that high compensators had improved response inhibition at the behavioral level (faster reaction time on a laboratory stop signal task) and at the neural level (stronger neural conflict monitoring as indexed by ERPs on a Go/Nogo task). Indeed, a "more robust science of brain-behavior relationships awaits if research efforts are grounded in alternative assumptions" (Westlin et al., 2023). Our results suggest social compensation is an alternative assumption that is critical for clinical neuroscientists to consider in neurobiological investigations of neurodiversity. Future longitudinal work examining within-person trajectories of neurocognitive compensatory strategies should test the intriguing possibility that compensatory strategies may become well integrated enough across developmental time that they might only be evident at the neural level.

From a developmental framework, "the study of resilience always comes back to these questions: *What makes a difference?* and *What can we do about it?*" (Masten, 2024). Consistent with the view that coordinated socially reciprocal behavior denotes transdiagnostic resilience (Feldman, 2021), our findings suggest key implications for research, practice, and policy. That is, the proposed underlying mechanism of neurodivergent social compensation is one that is malleable through effortful enhancement of inhibition skills. We found that some neurodivergent children may adapt socially at neurocognitive units of analysis even without demonstrating certain aspects of social cognition typically involved in social adaptation. Inhibition can be improved through training, not only for children with behavioral manifestations of categorical neurodivergence (Faja et al., 2021) but also in childhood educational settings more broadly (Honoré et al., 2020), such as through the training of mindfulness skills (Andreu et al., 2023). Our results provide preliminary empirical support for universal design programs that promote inhibition skills as a way to accommodate more neurodivergent ways of thinking (Rao et al., 2014). Focusing on integrating these curricula in health and education systems has potential to improve the wellbeing of more children who may be compensating.

From a neurodiversity inclusive framework, we suggest that underlying social cognition is not needed and that, instead, neurotypical individuals and structural systems in our neuronormative society (Wise, 2023) should ultimately adapt to be more supportive and inclusive of a range of social cognition skills and social behavior patterns. But given the reality of neuronormativity (Wise, 2023), utilizing the skill of inhibition may be helpful for social interactions through allowing neurodivergent children to strategically and effortfully inhibit their prepotent tendencies to engage in behavior considered socially inappropriate, such as expressing negative opinions of certain neurotypical norms. This may facilitate more integrated social adaptation through repeated use of neurocognitive compensatory strategies to inhibit these prepotent tendencies during real-world social interactions until this eventually becomes a more integrated, natural skill across development.

Several limitations should be noted and addressed in future research. First, although proportions of diagnostic classifications did not differ statistically between the High and Low Compensation groups, the sample size was relatively small, which limits power to detect diagnostic composition differences. These similarities in diagnostic group proportions should therefore be interpreted with caution. Second, our sample included relatively few girls (6 of 74 participants), preventing us from examining potential gender/sex differences (Hyde et al., 2019) in compensatory strategies. Recruiting larger and more gender/sex-diverse samples will be important for testing the generalizability of our findings. Third, although ADHD diagnostic status was confirmed using standardized caregiver measures following procedures used in prior work (Cremone-Caira et al., 2021), these children did not receive a comprehensive, independent ADHD clinical diagnostic evaluation as part of the study protocol. Therefore, ADHD classification may not reflect the population of children who receive a clinical diagnosis of ADHD. Future work should incorporate full multi-informant clinical evluations to more accurately characterize ADHD status. Finally, the present study was cross-sectional, which precludes examination of within-person change and limits inferences about the directionality of associations between inhibition, social cognition, and adaptive social behavior. Longitudinal designs following neurodivergent children across development will be important for characterizing trajectories of neurocognitive compensatory strategies and testing whether compensatory processes become increasingly integrated over time.

In summary, we found enhanced neural and cognitive inhibition and less neural effort while conflict monitoring among neurodivergent children who exhibit real-world socially adaptive behavior despite more difficulties with social cognition. Research, practice, and policy efforts should appreciate the nuanced ways in which observable social behavior may not always reflect neurocognitive compensatory strategies. Integrating supportive strategies to enhance inhibition in children across education and mental health care systems may be an effective strategy to promote neurodivergent social adaptation.

CRediT authorship contribution statement

Altschuler Melody: Writing – original draft, Software, Project administration, Methodology, Investigation, Formal analysis, Conceptualization. **Susan Faja:** Writing – review & editing, Visualization, Validation, Supervision, Software, Resources, Project administration, Methodology, Investigation, Funding acquisition, Data curation, Conceptualization.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.reia.2026.202787.

Data availability

Data are available through the NIH NDAR repository.

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