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Associations between retained primitive reflexes and cognitive performance in autism spectrum disorder

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ABSTRACT

Objective: We hypothesized that directing multimodal sensory stimulation to one side of the brain, particularly targeting theoretically underdeveloped networks, would facilitate the integration of hemispheric connectivities, and these changes would be observed alongside improvements in cognitive function in individuals with autism spectrum disorder (ASD).
Method: Transcutaneous Electrical Nerve Stimulation (T.E.N.S.) was applied as the primary somatosensory modality to stimulate the right hemisphere, along with retained primitive reflex stimulation. Neuropsychological testing included behavioral scales, academic achievement measures, and IQ subtest scores. In this study, we sought evidence to identify objective deficits that correlated with retained primitive reflexes and cognitive function. We then compared the existence of retained primitive reflexes and cognitive function in each participant before and after hemispheric stimulation, as well as in comparison to a control group receiving sham treatment.
Results: Support was found for the observation that reduction of retained primitive reflexes following unilateral T.E.N.S. stimulation was associated with concurrent improvements in cognitive performance in ASD. While these findings suggest parallel changes, the present study cannot determine whether reflex integration mediates the cognitive gains or whether both are influenced by broader changes in neural connectivity.

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Introduction

Primitive reflexes: What are they and what is the controversy?

During the second six months of life, primitive-reflex activity is inhibited due to the maturation of the central nervous system (Schott & Rossor, 2003). It has been found that delayed motor skill development and delayed suppression of primordial reflexes are related (Capute et al., 1982; Fiorentino, 2014, Melillo & Leisman, 2009; 2010; 2022a, 2022b; 2023). When RPRs are also asymmetrically present, there is strong support for the hypothesis that this is associated with maturational delays that, in turn, can be associated with deficits in hemispheric network communication, which we refer to as a functional disconnection (Gerber et al., 2010; Fiorentino, 2014; Leisman & Melillo, 2022a; 2023).

Primary reflexes, also known as primitive reflexes, are automatic responses originating from the central nervous system (CNS) that are typically present in infants but not in healthy adults. These reflexes are crucial for survival and development during infancy and are normally inhibited as the brain matures.

The neuroanatomical pathways associated with primary reflexes can be classified based on the level of the CNS at which they are controlled. They include: a) cortical reflexes: these involve higher brain functions and include equilibrium reactions. b) Midbrain reflexes including kinetic labyrinthine reflexes, body righting reflexes, and optical righting reflexes. c) Brainstem reflexes that include tonic neck reflexes (such as the asymmetric tonic neck reflex and symmetric tonic neck reflex), static labyrinthine reflexes, and supporting reactions. d) Spinal reflexes including flexor withdrawal, extensor thrust, crossed extension, plantar grasp, and palmar grasp reflexes (Ref).

Each of these reflexes serves specific functions and is integrated as the infant's motor development progresses. For example, the asymmetric tonic neck reflex (ATNR) helps develop hand-eye coordination, while the symmetric tonic neck reflex (STNR) is crucial for developing crawling patterns and postural control. Schematic of the anatomical pathways involved in primary reflex manifestation may be found in Fig. 1.

Primitive or infantile reflexes are sensory/motor reflexes that are present at birth. It has been known for some time that most of these reflexes are present in utero and that one of their functions is to help the child "birth itself" (Bartlett et al., 1997; Melillo & Leisman, 2010, Leisman et al., 2022a). By that, we mean that the main function of primitive reflexes is to allow the infant to move and react to its environment in the absence of a developed mature motor cortex at birth (Melillo & Leisman, 2010; Leisman & Melillo, 2022a; 2023). The infant needs to be able to move, feed, protect and orient him or herself, to engage their senses and muscles, and create sensory and motor feedback to activate genes that will build the brain from the bottom up. The control of these reflexes is thought to arise from multiple regions of the brainstem (Gieysztor et al., 2018; Leisman & Melillo 2023b; Melillo et al., 2023b). The lower reflexes in the medulla are thought to be active first followed by reflex control associated with the pons and mesencephalon (Vargiami & Zafeiriou, 2019).

It is important to differentiate RPRs from primitive reflexes which reappear in adults. RPRs are adaptive reactions occurring in the

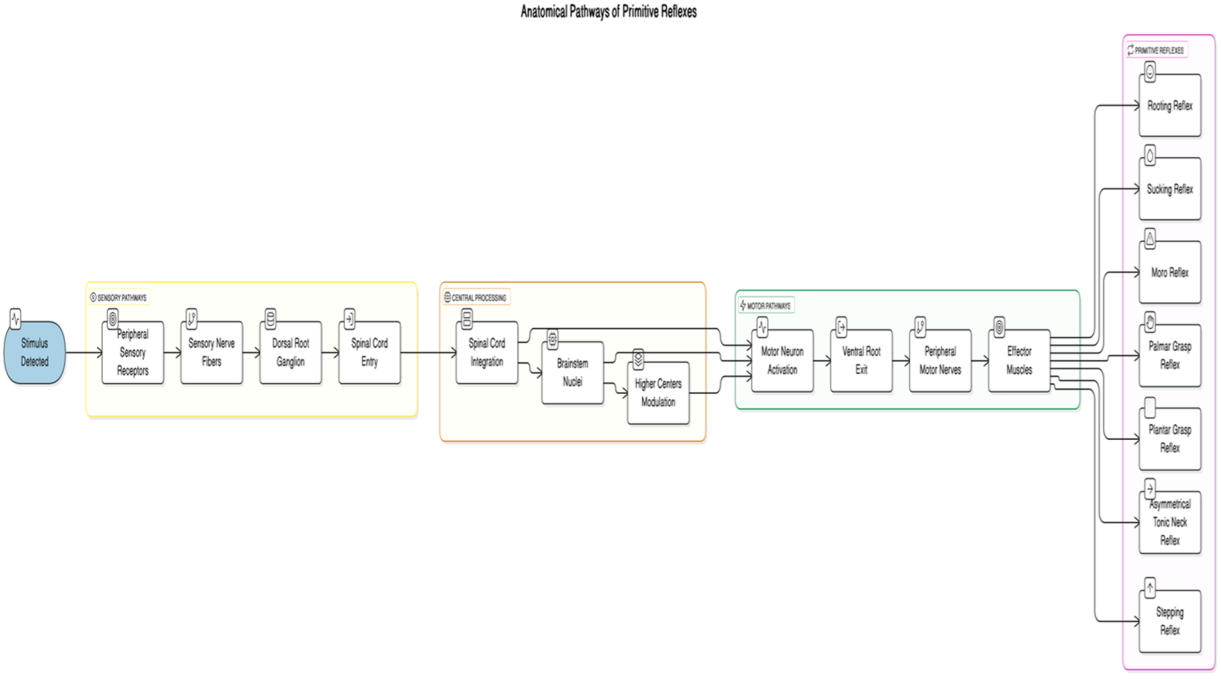


Fig. 1. Schematic of anatomical Pathways of primitive reflexes.

neonatal life, which become integrated as the brain matures. Their reappearance in adulthood returned primitive reflexes usually indicates impairment of cortico-subcortical networks, associated with normal aging as well as with adult-onset neurological compromise. Returned primitive reflexes may be viewed as a release of brainstem activity due to the loss of repressive input from higher levels of the central nervous system. They are often referred to collectively as "frontal release" signs, primitive or archaic reflexes, or atavistic reflexes (Scott & Schoenberg, 2010). They are seen in disorders that affect the frontal lobes, such as dementias, metabolic encephalopathies, closed head trauma, and hydrocephalus (Malloy et al., 1998; Barotono and Press, 2022), but in adults, the presence of frontal release signs reflect deficits in cognitive and executive function (Barotono and Press, 2023; Damasceno et al., 2005) found that in dementia, the highest frontal release sign scores tended to be associated with the lowest cognitive scores and with diminished regional cerebral blood flow in frontal regions. Thus, both the presence of multiple frontal release signs and their scores could be useful predictors of diffuse cerebral dysfunction. In persistent vegetative and minimally conscious states, it is common to find reappearance of primitive reflexes, such as grimacing, startle reflex, etc. (von Wild 2012). The presence of RPRs beyond infancy, on the other hand, may be taken as a sign related to neurological immaturity (Melillo & Leisman, 2010; Vargiami & Zafeiriou, 2019).

As higher levels of the brainstem become active, reflexes controlled by its lower areas become inactive or inhibited (Vargiami & Zafeiriou, 2019). Ultimately, this progression continues into the brain and neocortex. It is then thought that ultimately, the development of the frontal lobes leads to top-down control and inhibition of primitive reflexes (Starosta et al., 2016).

The controversy (Gieysztor et al., 2018) pertaining to RPRs is not whether they exist or not. Primitive reflex testing has been included as part of a normal pediatric neurology examination for decades. They are a well-accepted part of the evaluation of effective child development. The controversy surrounds the inhibition of these reflexes. Most pediatriaians assume that primitive reflexes are all completely inhibited by the end of the first year (Vargiami & Zafeiriou, 2019). The only time it is thought not to happen is if there is a brain injury at birth such as cerebral palsy (Chinello et al., 2018). However, there have been a significant number of studies that have indicated that in a certain percentage of the population, primitive reflexes are not inhibited in the first year of life and persist into middle childhood (Marschik et al., 2017) and even adulthood (Hogan & Ebly, 1995). It has also been documented that in children, adolescents, and adults with RPRs, a neurobehavioral disorder or learning disability coexists (Gieysztor et al., 2018; Chinello et al., 2018). Individuals with ADHD, ASD, Tourette's syndrome, dyslexia, and other neurobehavioral disorders almost always demonstrate RPRs that are thought to relate to maturational delay in the brain and nervous system (Melillo & Leisman, 2010; Gordon, 2013; Leisman et al. 2023; Melillo et al., 2022, 2023a, 2023b).

Cozolino (2017) noted that an individual with early functional neurological impairments or children with subtle developmental delays may still fall within the "normal" range for the majority of tests, yet actually be showing the beginnings of what may later

Table 1
General characteristics of the neurotypical control group (Group A).

Characteristic	Categories	No.	%
Gender	Male	25	83.3
	Female	5	16.7
Apgar score	9*9	23	76.7
	8*9	6	20.0
	7*9	1	3.3
Gestational age	less than 37	3	10.0
	37–42	27	90.0
	more than 42	-	-
Birth	Natural	18	60.0
	Cesarean	12	40.0
Birth Weight (GRAMS)	less than 2500	1	3.33
	2500–4000	26	86.7
	more than 4000	3	10.0
IQ (Intelligence quotient)	140 and more	2	6.7
	120–139	8	26.6
	110–119	2	6.7
	90–109	14	46.6
	less than 90	4	13.4
Overall Health	Normal	30	100
	Abnormal	-	-
Grade Level	Pre School	-	-
	Primary School	12	40.0
	Secondary School	7	23.3
	High School	1	3.4
	Technical	4	13.3
	University	6	20.0
Sidedness	R	24	80.0
	L	6	20.0
Age (years)	5–15	20	66.7
	16–25	6	20.0
	26–35	4	13.3
Hemis. Domin.	R	6	20.0
	L	24	80.0

develop into a pathological state in adulthood such as in the case of schizophrenia (Cozzolino, 2017). Perhaps then these signs can be viewed as early markers for developmental delay or for neurological dysfunction.

Cognitive deficits and RPRs

The relationship between cognitive deficits and RPRs has also been controversial (Koziol et al., 2016). Some authors (Damasceno et al., 2005; Koziol et al., 2016) consider these reflexes as predictive of diffuse cerebral dysfunction, since these signs are significantly correlated with cognitive deficits in a wide age-range of individuals as determined by the Halstead-Reitan neuropsychological test battery (Koziol et al., 2016) and the Woodcock-Johnson Tests of Cognitive Abilities (McWhirter et al., 2022).

If primary reflexes are retained beyond the normal developmental period, they have the potential to disrupt maturational processes and reduce the brain's ability to transmit, process, and integrate sensory information effectively (Blythe, 2011; Sigafoos et al., 2021). In other words, the persistence of primary reflexes beyond the normal timespan (12 months) interferes with subsequent development and is indicative of neurological impairment (McPhillips & Sheehy, 2004) or of maturational delay (Konicarova & Bob, 2013). The findings indicate that independently of the participants' age, the persistence of the primitive reflexes was significantly associated with infant's performance in both the interaction with objects (i.e., actions) and with people (i.e., communicative gestures), meaning that low scores on RPR assessment, correspond to elevated persistence of these reflexes, and correlate with low scores in motor repertoire irrespective of the infants' age. These findings are consistent with previous studies, revealing that the persistence of the asymmetrical tonic neck reflex, another primitive reflex, is associated with reduced fine motor ability (e.g., fingering, shaking, rotating and transferring objects across the midline) (McPhillips et al., 2000), and gross motor abilities (e.g., rolling, creeping, crawling, riding a bicycle and catching or kicking a ball) (McPhillips & Sheehy, 2004).

We theorize that the presence of RPRs can be explained by delayed or "arrested" neurological development of cortical and cortical-subcortical connectivities that should affect efficient intercommunication between processing of many motor and cognitive behaviors (for a more comprehensive overview of the theoretical basis for this contention, *cf*. Leisman et al., 2022a; 2023b). Therefore, this study aims to demonstrate whether hemisphere-specific electrical stimulation interventional protocols to integrate RPRs in ASD individuals can lead to a corresponding improvement of cognitive function.

Methods and methodology

Participants

The characteristics of the participants are reported in Table 1. Sixty male and female participants were recruited from the Institute for Neurology and Neurosurgery in Havana, Cuba, and tested and treated in the Clinical Electrophysiology laboratory. Demographic data of the participants were recorded and included age, gender, Apgar score, birth weight, gestational age, and whether the birth was natural or a Caesarian section. Also recorded were the IQ, overall health of the participants, grade level, and sidedness. Three age groups included 10 ASD participants each (5–10; 11–19; 25–35 years), reflecting different normative stages of development into adulthood. Additional selection criteria may be found below. The participants included 50 males and 10 females whose mean age was 15.8 (S.D. 7.21). The groups' characteristics can be found in a data depository at (https://www.researchgate.net/publication/372345066)

Inclusion criteria

Each participant in both the control and experimental groups was blindly clinically examined by two child neurologists and diagnosed with ASD, based on DSM-V criteria (American Psychiatric Association, 2022) Each of the ASD participants possessed a classical autistic triad of impairments in social interaction, communication, and imagination (Zappella, 2012; Zeidan et al., 2022; Hodges et al., 2020; Bitsika, & Sharpley, 2023) with relatively intact verbal functions and with WISC-IV(Spanish/WAIS) I.Q.s over 85 (Charman et al., 2011a; 2011b).

The following conditions were required for inclusion in the control group: a history of uneventful prenatal, perinatal, and neonatal periods; no disorders of consciousness; no history of central or peripheral nervous system disease; head injury with cerebral symptoms; convulsive episodes; paroxysmal; headache; enuresis or encopresis after the fourth birthday; tics; stuttering; pavor-nocturnus; or any psychiatric, behavioral, or drug-related disorder. Depending on age, school-aged participants demonstrated normal academic achievement (American Psychiatric Association, 2022; Silver & Rapin, 2012). Control group inclusion criteria were based on: a history of uneventful prenatal, perinatal, and neonatal periods; no disorders of consciousness; no history of central or peripheral nervous system disease, head injury with or without cerebral symptoms, convulsive episodes, paroxysmal, headache, enuresis, or encopresis after the fourth birthday, tics, stuttering, pavor-nocturnus, or any psychiatric, behavioral or drug-related disorder. Control group participants were excluded if any spike-wave or paroxysmal activity was present in the EEG.

Exclusion criteria

None of the participants demonstrated a history of epileptic symptoms and neurologic abnormalities other than those directly related to autism, and no history of cerebral palsy or Traumatic Brain Injury (TBI) or brain surgery. None of the participants demonstrated any genetic disorder, metabolic illness, vascular disorder, or history of cancer, and they could not be breastfeeding or pregnant.

None of the control participants demonstrated a history of cerebral palsy, TBI or brain surgery. Participants in the control group did

not demonstrate any genetic or vascular disorder, metabolic illness, or history of cancer and could not be breastfeeding or pregnant. Participants were free of drug treatment. Control group participants showed no manifestations of any RPRs. Depending on age, school-aged participants who demonstrated normal academic achievement (American Psychiatric Association, 2022; Silver & Rapin, 2012) were excluded from the control group as well as individuals demonstrating any spike-wave activity in the EEG.

Informed consent and institutional approval

The Institute of Neurology and Neurosurgery Ethics Committee and the IRB for the University of Haifa research approved the proposed research projects (INN2020–41). The participants' relatives or persons responsible provided informed consent to participate in the study.

Characteristics of participants

Table 1 represents participant data that is purely descriptive and represents the control group (Group A) consisting of 30 individuals whose inclusion/exclusion selection criteria are more fully described in sections 2.1.1 and 2.2.2. above (group A). In this table where the neurotypical control participants are represented, the initial variables are similar, however with IQ, level of schooling, there is a tendency towards a higher IQ, higher levels of schooling, and the other variables. However, the differences validate significant differences between the ASD and Neurotypical groups regardless of the criteria employed to define each group.

Table 2 represents the particularization of the characteristics described for the entire study sample in participants with ASD (Group B). Male participants predominate, Apgar count of 9*9, full-term birth, natural birth, and normal weight at birth are all well within normal limits. However, when focusing on variables such as IQ, level of education, low IQ, low level of education predominates, reflecting a functional disorder. When comparing Tables 1 and 2 these differences are then confirmed.

In Tables 3 and 4 descriptive differences are noted between the neurotypical control group and ASD experimental groups. The tables are illustrative of group differences noting that in ASD individuals, language is acquired later than in the neurotypical individuals, and motor and toileting milestones are reached later in Group B on average than in Group A participants.

Procedure

Reflex testing and stimulation

All individuals had the following reflexes examined clinically and included both symmetric and asymmetric: Asymmetric Tonic

Table 2
General characteristics of participants with ASD (Group B).

Characteristic	Categories	No	%
Gender	Male	25	83.4
	Female	5	16.6
Apgar score	9*9	19	63.3
	8*9	9	30.0
	7*9	2	6.7
Gestational age	less than 37	4	13.3
	37–42	26	86.7
	more than 42	-	-
Birth	Natural	24	80.0
	Cesarean	6	20.0
Birth Weight (GRAMS)	less than 2500	1	3.3
	2500–4000	23	76.7
	more than 4000	6	20.0
IQ (Intelligence quotient)	140 and more	-	-
	120–139	-	-
	110–119	2	6.7
	90–109	10	33.3
	less than 90	18	60.0
Overall Health	Normal	30	100.0
	Abnormal	-	-
Grade Level	Pre School	1	3.4
	Primary School	15	50.0
	Secondary School	8	26.6
	High School	4	13.3
	Technical	2	6.7
	University	-	-
Sidedness	R	25	83.3
	L	5	16.7
Age (years)	5–15	20	66.7
	16–25	7	23.3
	26–35	3	10.0
Hemis. Domin.	R	5	16.7
	L	25	83.3

Table 3
Language, motor, and toilet milestones in neurotypical control participants.

Milestone	Age	No.	%
Language Milestones	10–16 months	25	83.4
	17–24 months	4	13.3
	25–36 months	1	3.3
	37–47 months	-	-
	48 and more months	-	-
Motor Milestones	10–14 months	28	93.4
	15–18 months	1	3.3
	More than 18 months	1	3.3
Toilet Milestones	12–18 months	16	53.4
	19–24 months	7	23.3
	2 years	3	10.0
	3–4 years	3	10.0
	5–6 years	-	-
	7 years	1	3.3

Table 4
Language, motor, and toilet milestones in participants with ASD.

Milestone	Age	No.	%
Language Milestones	10–16 months	2	6.7
	17–24 months	6	20.0
	25–36 months	7	23.3
	37–47 months	12	40.0
	48 and more months	3	10.0
Motor Milestones	10–14 months	6	20.0
	15–18 months	14	46.7
	More than 18 months	10	33.3
Toilet Milestones	12–18 months	3	10.0
	19–24 months	2	6.7
	2 years	1	3.3
	3–4 years	2	6.7
	5–6 years	22	73.3
	7 years	-	-

Neck Reflex (ATNF), Symmetric Tonic Neck Reflex (STNR), Spinal Galant, Babinski, Palmer Grasp, Rooting and Tonic Labyrinthine Neck Reflex (TNR). Abnormality was defined as an individual exhibiting two or greater RPRs. Reflexes were graded on a scale of 0–4 based on clinical judgment (0 =0 fully integrated; 1 =25 %; retained; 2 = 50 % retained; 3 =75 %; 4 =100 % completely retained when Tested by 3 examiners. The testing procedures are described more fully in Table 1 and a detailed procedural guide may be found in Melillo et al. (2023/BMJ).

Cognitive and behavioral testing

The Spanish editions of the following standardized tests were performed: The Wechsler Individualized Achievement Testing-III (WIAT-III) (Vaughan-Jensen et al., 2009), Wechsler Intelligence Scale for Children-Fourth Edition (WISC-IV) (Clinton, 2005), ADI-R (Autism Diagnostic Interview-Revised (Lebersfeld et al. 2021)).

Hearing and vestibular function

All children in Cuba with ASD are routinely tested by brainstem auditory evoked potentials to rule out auditory impairment. Cranial nerve function was also included as part of the evaluation protocol as was the case in the patients evaluated in this report.

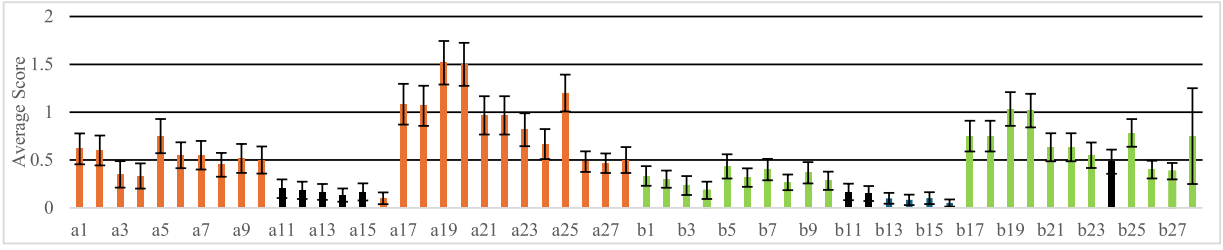


Fig. 2. Pre-reflex integration mean score (**orange**) vs. Post-reflex integration mean score (**green**), not significant (**black**) (Mean and SE). Reflexes are defined in Table A2.

RPR-reduction stimulation procedures

The reflex elicitation techniques and intervention techniques used for all four subjects are shown in the Appendix in Table A1.

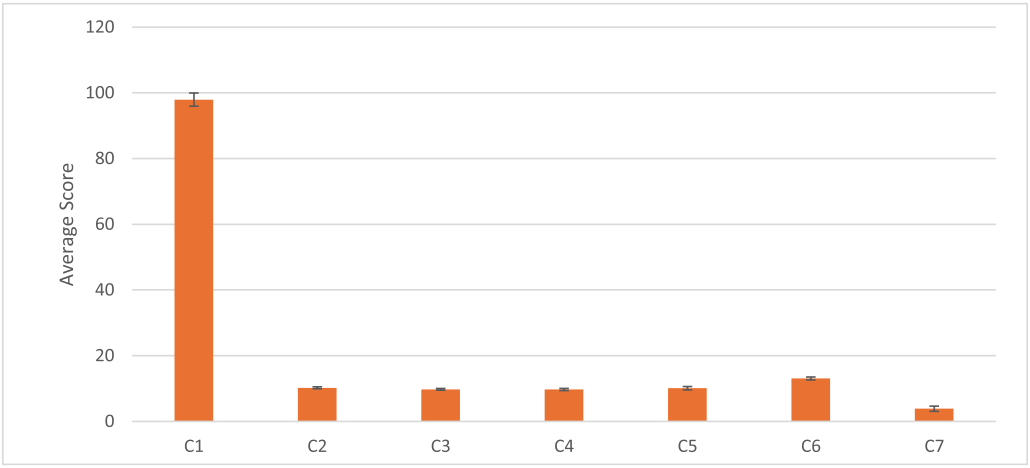
Results

Fig. 2 and 3 report the results of the neuropsychological testing before and after the 12-week treatment program that included RPR integration. The raw data set may be found here. All of the statistical analytical data can be found in the appended Tables A1 and A2.

Examinations for the presence of RPRs were performed both before and after the intervention program. A Z-test for comparison of proportions was performed to determine if the difference in proportions or percentages in terms of the RPRs that disappeared after the intervention was statistically significant. Most of the ASD participants demonstrated a reduction in observed RPRs, and this improvement was statistically significant ($p < .05$). For significant Wilcoxon tests, effect sizes (r) ranged from 0.32 to 0.45, indicating small-to-moderate effects. Bootstrapped 95 % confidence intervals for these effect sizes did not cross zero, supporting the robustness of the observed changes (see Table A2).

As the data were non-parametric, in this study, pre-/post changes in retained primitive reflex (RPR) scores and cognitive performance, and their associations. Significant improvements in cognitive performance were noted on all measures in the ASD individuals between pre- and post-testing among those who received the intervention. Effect sizes for pre/post changes on WISC/WAIS subtests ranged from medium ($d \approx 0.45$) to large ($d \approx 0.80$), with 95 % confidence intervals confirming that nearly all effects were reliable (Table A3). A Bonferroni correction for multiple comparisons, providing greater conservatism of estimate, rendered only one reflex test below significance. Bootstrapped 95 % confidence intervals for Cohen's d estimates did not cross zero, confirming the reliability of

(A)



(B)

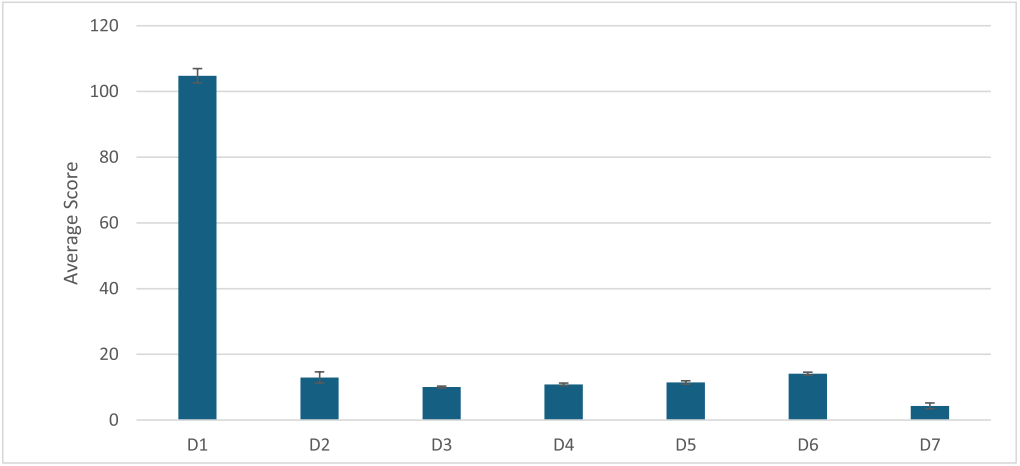


Fig. 3. (A) Pre-reflex integration WISC/WAIS IQ and sub-test scores. (B) Post-reflex integration WISC/WAIS IQ and sub-test scores.

these effects (Table A3). Because this was a pre/post observational design without formal mediation testing, we do not infer that RPR integration mediates cognitive change; we report associations and within-group pre/post differences.

In addition to the pre- and post-intervention performance analyses presented in Table A3, correlation analyses were conducted to examine associations between retained primitive reflex scores and cognitive outcomes. These results are presented in Appendix Tables A4 and A5. Table A4 displays pre-intervention correlations, and Table A5 displays post-intervention correlations, each reporting exact correlation coefficients (r), corresponding significance levels (p), and 95 % confidence intervals calculated using Fisher's *r-to-z* method.

Discussion

Primitive reflexes are involuntary motor responses present at birth, originating from the brainstem, and are essential for an infant's survival. As the nervous system matures, these reflexes should integrate into higher brain functions, allowing for controlled and purposeful movement. When these reflexes remain unintegrated, they can disrupt neurological harmony, affecting everything from motor skills to cognitive processing and emotional regulation. For example, a retained Moro reflex, responsible for an infant's startle response, can cause chronic stress and heightened reactivity to stimuli 2. Similarly, a retained fear paralysis reflex can impair logical thinking and appropriate responses, often resulting in behaviors like screaming or hitting in stressful situations.

Children with ASD demonstrate significant delays in reflex pattern development, which is typically linked to their muscle tone deregulation and hyperactive protective behavior. ASD is a disorder characterized, among other characteristics, by immature and poorly functioning reflex motor patterns. Lack of reflex integration in infancy or within the first two years of life negatively affects neurodevelopment and can interfere with the development of conscious motor-cognitive skills.

We observed significant pre-/post changes in reflex patterns following hemisphere-based T.E.N.S. stimulation. These changes are consistent with improvements in sensory–motor link function, although causal attributions cannot be made from the present design.

In this study, reductions in retained primitive reflexes occurred alongside significant improvements in cognitive performance after

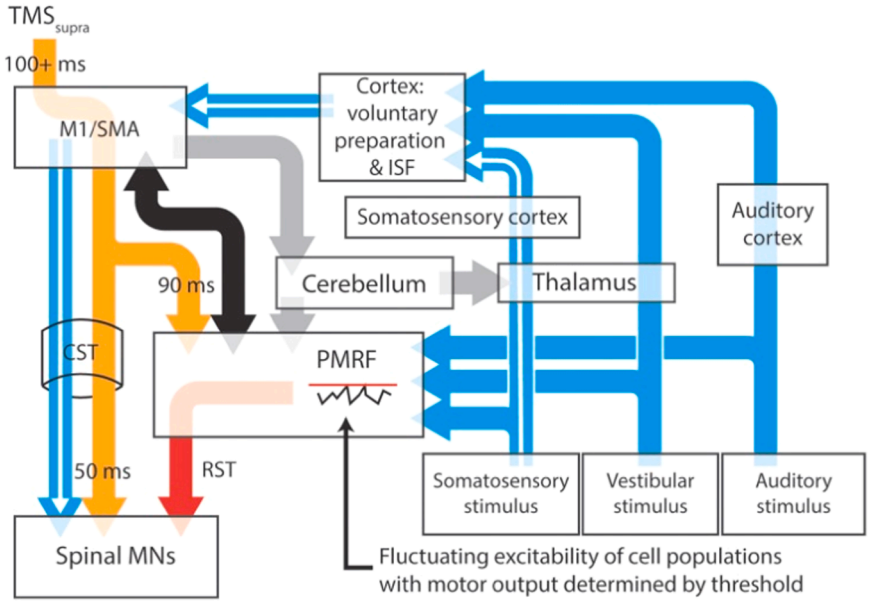


Fig. 4. | A hypothesized model for the brain circuits subserving the expression of long latency startle and stretch reflexes. Transcortical contributions to long latency stretch reflexes are enabled by a pathway that incorporates the primary somatosensory and motor cortices before descending to muscles through the corticospinal tract (white arrows). Stimulation of somatosensory, vestibular, or auditory systems results in the transfer of these signals to the cortex and the pontomedullary reticular formation (blue arrows), each of which has thresholds for activating output cells. Inhibition of the primary motor cortex (or areas involved in movement preparation) by application of suprathreshold TMS (orange arrows) results in almost synchronous inhibition of cells within the spinal cord and PMRF, although for different times. Spinal motoneurons appear to be inhibited for ~50 ms after TMS. Output cells inside the motor cortex, however, can be blocked for up to 200 ms (Strick, 1983). During periods of cortical inhibition, activation of the corticospinal tract is not possible, although a combination of cortico-reticulospinal input (black arrows) and sufficiently large sensory input can still activate reticulospinal tract cells (red arrow) after any TMS-induced inhibition of PMRF ceases. The likelihood of PMRF output (startle) is controlled in this model by the magnitude of the sensory input and the immediate excitability of PMRF cells. A comparable situation is expected to arise at the cortical level (see Alibiglou and MacKinnon, 2012). Task, posture, and stability-dependent modulation of stretch reflex reactions likely entail input from the cerebellum to both the main motor cortex and reticular formation (gray arrows). In this figure, thalamocortical projections reach regions involved for voluntary motor preparation, although projections straight to M1 may also play an essential role in modulating quick responses. M1, Primary motor cortex; SMA, supplementary motor region; PMRF, pontomedullary reticular formation; ISF, intersensory facilitation; MN, motoneuron (adapted from Shemmell, J., 2015).

hemisphere-specific stimulation described in Appendix A. These findings are consistent with the view that both reflex integration and cognitive function are influenced by changes in hemispheric connectivity. However, our design does not allow us to determine whether the disappearance of reflexes directly mediates cognitive improvement, or whether both are parallel outcomes of the same intervention. Accordingly, we interpret the RPR–cognition relationship as correlational, not causal or mediational, within this dataset. By addressing these foundational neurological processes, reductions in retained reflexes were observed in parallel with improved learning and cognitive function (Melillo et al., 2023a; 2023b). The pathways involved in some primitive reflexes that were integrated can be found in Fig. 4. Future work using mediation analysis or longitudinal modeling will be required to determine whether reductions in RPRs are causally responsible for cognitive gains.

RPRs have the potential to impede maturation processes and lessen the brain's capacity to interpret sensory data if they persist after the typical developmental period. Alternatively, the continuation of primary reflexes after the typical period (12 months postpartum) may imply interference with future development and be a sign of neurological malfunction.

The persistence of the primitive reflexes is significantly correlated with an infant's performance in the interaction with objects (i.e., actions) as well as with people (i.e., communicative gestures) regardless of the age of the participants, which means that low scores in the assessment of the primitive reflexes, which correspond to high persistence of the reflexes, correlate with low scores in motor repertoire regardless of the age of the infants (Konicarova & Bob, 2013; Gieysztor et al., 2018; Melillo et al., 2020; 2022; 2023a; 2023b; Leisman et al., 2022a; 2022b; 2023). This result is in line with earlier research, which showed that the persistence of the Asymmetrical Tonic Neck reflex, a different primitive reflex, impairs both fine and gross motor skills. These skills include rolling, creeping, crawling, riding a bicycle, and catching or kicking a ball (McPhillips, 2000; McPhillips & Sheehey, 2004)

Reductions in motor activity, spatial exploration, experience-dependent plasticity, RPRs, and delayed postural reflexes are all signs of cortical networks' overall immaturity in early life (Vogel et al., 2010; Sathyanesan et al., 2019). If RPRs developed in an asymmetric way, a more precise imbalance in maturity would be anticipated. We would anticipate asymmetric brain and nervous system development and maturation if there were unilateral RPRs and unilateral delays of postural reflexes, as this would change muscle tone or produce asymmetry of tone, which would change sensory and muscle feedback, which is believed to be the primary factors influencing brain development (Longman et al., 2017).

According to several investigations, brain connectivities in autistic adults and children varies. Children with autism may exhibit particularly strong connections in several brain networks, whereas autistic adults typically display weaker connections in a number of the same networks (Uddin et al., 2013; Rane et al., 2015; Dajani & Uddin, 2016; Cai et al., 2021).

The "unevenness" of cognitive ability is one of the most fascinating characteristics of individuals with ASD. We have suggested that understanding the condition's basis as a functional disconnection syndrome, like what is seen in sleep (Daneault et al., 2021), minimally conscious states (Porcaro et al., 2022), or as reported in patients with dyslexia (Habib, 2021), will help explain the variety of behavioral effects noted in ASD and nearly all neurobehavioral disorders. (Melillo & Leisman, 2009; 2010; Wang et al., 2020; Siffredi et al., 2021). This point of view has long been known (Leisman & Zenhausern, 1982). Widespread cortical networks may exhibit functional asymmetry, which could lead to decreased temporal coherence in some networks and increased temporal coherence in other functional networks (Gansel, 2022; Leisman et al., 2022a; Kumar et al., 2021).

Childhood and adulthood's retention of infantile reflexes has been linked to brain damage and several developmental issues. (Swapna et al., 2020; Sigafoos et al., 2021; Mohamed et al., 2023). Additionally, they have been reported in childhood functional neurological disorders that are not linked to any particular neurological illness or trauma (McWhirter et al., 2022; Pecuch et al., 2021).

Normally, after the first few months of life, the feedback produced by movement caused by primitive reflexes leads to the inhibition of these reflexes and the activation of more complex postural reflexes, resulting in a more complex interaction with the environment. This in turn is associated with a greater amount of sensory feedback, which activates genes that allow for the creation of integration and coordination between different cortical networks (Leisman et al., 2022a). More areas can be stimulated simultaneously as these cortical networks grow more integrated and coupled. This increases the speed of their interactions and enhances their synchronization (Melillo et al., 2023a; 2023b).

If the child's brain will not continue to expand and develop at a normal rate, the emergence of its more mature functions will be delayed as a consequence. This can happen if cortical maturity and motor coordination are delayed, which may occur because of the abnormal persistence of primitive reflexes (Zafeiriou, 2024; Sigafoos et al., 2021; Leisman et al., 2022a). With the anomalous, asymmetric persistence of primitive reflexes, which affects how the brain's hemispheres develop at distinct rates and times, an imbalance in maturation can result, associated with one hemisphere maturing normally while the other is delayed. Significant imbalances in synchronization and temporal coherence may be associated with this, making it harder for the two hemispheres' cortical networks to link in space and time. A functional disconnection syndrome, which can manifest with a variety of symptoms, may also be associated. We offer clinical support here and elsewhere (Melillo et al., 2023a; 2023b) for the notion that ASD can be, in part, associated with maturational delays as reflected in RPRs and imbalances and not necessarily a result of actual structural damage or pathology. ASD individuals are amenable to remediation (Melillo et al., 2020; 2022). We also support the notion that the presence of RPRs and the developmental milestones that might be delayed or absent as a result may be the earliest markers of developmentally delayed children in general and those with ASD. As a result, we offer support for the notion that hemispheric-specific interventions can significantly reduce the presence of RPRs and will consequently have measurable and significant positive effects on both motor and cognitive function.

Limitations

The study has several limitations. First, the relationship between reductions in RPRs and improvements in cognition should be

interpreted as correlational rather than causal; the present data cannot establish mediation. Although concurrent changes were observed, whether reflex reduction drives cognitive improvements or whether both reflect broader hemispheric changes remains unresolved. Second, the RPR testing scale was narrow (0–4), and reflex scoring relied on clinical judgment, which may introduce variability. Third, while we now report effect sizes and confidence intervals alongside p-values, the modest sample size and use of non-parametric testing may still limit robustness. Finally, generalizability is restricted to ASD individuals without major comorbid neurological disorders. Future work should incorporate mediation analysis, objective reflex testing (e.g., EMG), and a broader range of cognitive measures beyond IQ. RPRs appear to be present at all ages, even though this is not a topic often covered in the literature. Treatment was associated with diminished RPRs and contemporaneous improvements in cognitive performance (Machado et al., 2015; Melillo et al., 2023b). These concurrent changes may reflect alterations in functional connectivity; however, the present data do not establish mediation or causation. After the first year of life, neurodevelopmentalists might wish to consider the examination of primary reflexes post-infancy, given their potential associations with broader cognitive and developmental outcomes.

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CRediT authorship contribution statement

Eli Carmeli: Writing – review & editing, Writing – original draft, Validation, Supervision, Resources, Project administration, Methodology, Funding acquisition, Formal analysis, Conceptualization. **Ty Melillo:** Writing – review & editing, Writing – original draft, Methodology, Conceptualization. **Calixto Machado:** Writing – review & editing, Writing – original draft, Resources, Methodology, Investigation. **Mauricio Chinchilla-Acosta:** Writing – review & editing, Writing – original draft, Validation, Resources, Investigation. **Yanin Machado-Ferrer:** Validation, Resources, Project administration, Investigation, Formal analysis, Data curation. **Gerry Leisman:** Writing – review & editing, Writing – original draft, Visualization, Validation, Supervision, Software, Resources, Project administration, Methodology, Investigation, Funding acquisition, Formal analysis, Data curation, Conceptualization. **Robert Melillo:** Writing – review & editing, Writing – original draft, Visualization, Validation, Resources, Project administration, Methodology, Investigation, Funding acquisition, Formal analysis, Data curation, Conceptualization.

Declaration of Competing Interest

None of the authors have competing interests to declare.

Appendix A

Table A1
The table lists procedures for reflex testing and intervention more fully explained in Appendix 1 (from Melillo et al., 2023a)

Reflex Repetitions			
Reflex	**Method of Evoking Reflex**	**Reflex Stimulation Procedures**	**Frequency of Repetition**
Tonic Labyrinthine (TLR)	Laid on the back with legs flexed up and arms wrapped around legs; head on ground to start. The head flexed, and body rocked forward as far as possible.	Extend head & roll backwards	x 10 × 3/day
Asymmetric Tonic Neck Reflex (ATNR)	Lying on stomach, head turned to one side, arm and left are extended straight on side of head turn, arm and leg on opposite side flexed	Lifts head turning it simultaneously flexing extended arm & leg & opposing arm and leg extended Then return head to original position and arms and legs back to original position	x 10 × 3/day
Symmetric Tonic Reflex (STNR)	On hands and knees, eyes open, bend head back and look upwards then bend head down to look through the knees	Patient on knees while patient chin & forehead held then head moved up & down rapidly through full range	x 10 × 3/day
Babinski	Roll Tennis Ball on bottom outside of foot.	Stroke upwards with hard end of paintbrush laterally & at bottom of foot.	20 Left foot, 10 Right: x 3/day
Rooting	Suck on straw or suck & blow lips.	Brushstroke from cheek to corner of mouth then across both lips x 5 then from chin toward corner of mouth and across lips x 5 x's; repeat on contralateral face.	30 s to 1 min x 3/day
Snout		Press on filtrum (space between nose & upper lip).	x 10 × 3/day
Spinal Galant	Lying on the back, arms at side & legs together. Open arms & legs together slowly as far as possible bringing hands together over head and legs spread	Stroke side of spine downwards with hard side of a brush little more lateral 10 times both sides	x 10 × 3/day

(*continued on next page*)

Table A1 (*continued*)

Reflex Repetitions			
Reflex	**Method of Evoking Reflex**	**Reflex Stimulation Procedures**	**Frequency of Repetition**
	apart simultaneously. Slowly return to original position 10 repetitions		
Moro	Sitting on chair, start from fetal position, hands curled in fist & head bent forward. Right wrist over left wrist and right ankle over left (crisscrossed). Open all the way bending head back with arms and legs stretched then return to fetal position crossing left wrist over right & left ankle over right repeat & to original position for 10 repetitions	Lying face up, hold head & bend it forward quickly moving head downwards approx. 1–2 in. 10 reps. Additionally clap hands loudly behind the individuals head to evoke startle.	x 10 × 3/day
Palmer Grasp	Squeeze tennis ball with hand	Use brush or vibration to draw X on hand with the hard side of the brush.	x 20 left, x 10 right x 3/*day*
Sensory Stimulation			
Vestibular Ocular Reflex (VOR)	VOR exercises to the left (head turn):	While fixating on object (e.g., pencil, finger, or looking in mirror turn head left-ward as far as possible while keeping eyes fixed on object.	x 10 ×2/day
Post-rotational nystagmus testing (PRNG) (Spinning)	Spinning	Spin fast clockwise for 10 rotations of 2 sec/rotation and slow spin counterclockwise @ 6 sec/rotation repeated until nystagmus evoked	10 rotations each direction x 3/day
Optokinetic reflex	Optodrum	Use animals or black & white stripes to right & down only generated on mobile phone	30 sec x 3/day.
Light stimulation	Penlight	Shine light in corner of left eye	x 10 for 3 sec -x 3/day
Combined sensory stimulation	MetroTimer	Flashing light @ 54 beats/min in corner of left eye only for 1 min After one month sound & light together 30 sec after light & sound separately for 30 sec.	x 3 times/day
Tactile stimulation	Brushing	Brush on left arm & legs for right hemisphere dominant or vice versa for left dominant	x10 times on each limb x 3/day
Transcutaneous electrical nerve stimulation (TENS)	Pulsed electrodermal stimulation & Pulsed light stimulation	Wireless pads placed on left upper back between shoulder blade & spine. TENS unit set to mode 6. Starting at 10 min x 2/day. Increase intensity until individual felt light tingling ensuring absence of muscle contraction. Initially administered separately from pulsed light stimulation. At 5th wk. (when both administered for 30 min) then combined for 30 min. simultaneously, increasing by 10 min. every other week up to 60 min. for both simultaneously.	x 2/day (Every other week added 10 min until 60 min) x 2/day

Table A2
Reflex testing pre vs. post primitive reflex integration. Wilcoxon non-parametric tests statistical significance (n = 60). Wilcoxon non-parametric statistical tests were used as the data is not normally distributed. Codes for reflexes are described in the left column (a=pre-reflex integration and b=post-reflex integration)

.**Reflex Code**	**Primitve Reflex**	**Significance**
a1	ATNR-ASYMMT-L	0.004
a2	ATNR-ASYMMT-R	0.004
a3	STNR-ASYMMT-L	0.059
a4	STNR-ASYMMT-R	0.059
a5	SPINAL GALLANT-AYMMT-L	0.004
a6	SPINAL GALLANT-AYMMT-R	0.006
a7	PALMER-ASYMMT-L	0.014
a8	PALMER-ASYMMT-R	0.009
a9	BABINSKI ASYMMT-L	0.034
a10	BABINSKI ASYMMT-R	0.010
a11	ROOTING-ASYMMT-L	0.317 (NS)
a12	ROOTING-ASYMMT-R	0.317 (NS)
a13	LABYRINT ASYMMT-L	0.102 (NS)
a14	LABYRINT ASYMMT-R	0.083 (NS)
a15	MORO-ASYMMT-L	0.102 (NS)
a16	MORO-ASYMMT-R	0.180 (NS)
a17	ATNR-SYMMT-L	0.00001
a18	ATNR-SYMMT-R	0.0001

(*continued on next page*)

Table A2 (*continued*)

.Reflex Code	Primitve Reflex	Significance
a19	STNR-SYMMET-L	0.002
a20	STNR-SYMMET-R	0.020
a21	SPINALGALLANT-SYMMET-L	0.0001
a22	SPINALGALLANT-SYMMET-R	0.059
a23	PALMER-SYMMET	0.059
a24	BABINSKI SYMMET	0.205 (NS)
a25	ROOTING SYMMET	0.002
a26	LABYRINT SYMMET-L	0.002
a27	LABYRINT SYMMET-R	0.006
a28	MORO-SYMMET	0.006
b1	ATNR-ASYMMT-L	0.004
b2	ATNR-ASYMMT-R	0.004
b3	STNR-ASYMMT-L	0.059
b4	STNR-ASYMMT-R	0.059
b5	SPINAL GALLANT-AYMMT-L	0.004
b6	SPINAL GALLANT-AYMMT-R	0.006
b7	PALMER-ASYMMT-L	0.014
b8	PALMER-ASYMMT-R	0.009
b9	BABINSKI ASYMMT-L	0.034
b10	BABINSKI ASYMMT-R	0.010
b11	ROOTING-ASYMMT-L	0.317 (NS)
b12	ROOTING-ASYMMT-R	0.317 (NS)
b13	LABYRINT ASYMMT-L	0.102 (NS)
b14	LABYRINT ASYMMT-R	0.083 (NS)
b15	MORO-ASYMMT-L	0.102 (NS)
b16	MORO-ASYMMT-R	0.180 (NS)
b17	ATNR-SYMMT-L	0.0001
b18	ATNR-SYMMT-R.	0.0001
b19	STNR-SYMMET-L	0.002
b20	STNR-SYMMET-R	0.020
b21	SPINALGALLANT-SYMMET-L	0.0001
b22	SPINALGALLANT-SYMMET-r	0.059
b23	PALMER-SYMMET	0.059
b24	BABINSKI SYMMET	0.205 (NS)
b25	ROOTING SYMMET	0.002
b26	LABYRINT SYMMET-L	0.002
b27	LABYRINT SYMMET-R	0.006
b28	MORO-SYMMET	0.006

Table A3
Raw WISC/WAIS test results and subtests scores (= pre-reflex integration and d = post-reflex integration). All results other than Block-Design significantly changed between pre- and post-reflex integration ($p < 0.001$)

C1	C2	C3	C4	C5	C6	C7	D1	D2	D3	D4	D5	D6	D7
IQ WISC/ WAIS	**Pic Comp**	**Block Des**	**Matrix Reas**	**Info**	**Simil.**	**Digit Span**	**IQ WISC/ WAIS**	**Pic Comp**	**Block Design**	**Matrix Reason**	**Info**	**Similar**	**Digit Span**
85	10	10	10	9	10	10	106	13	15	16	11	12	9
128	12	13	14	18	18	0	144	13	13	17	20	18	0
122	13	11	13	19	10	0	123	13	11	13	19	19	0
96	12	8	10	6	17	0	95	13	9	9	6	12	0
85	8	9	8	6	11	0	92	12	7	7	8	12	0
123	13	12	13	13	20	0	126	13	9	12	16	20	0
85	9	8	5	11	11	9	87	10	8	6	11	11	9
94	7	10	10	8	13	0	104	10	11	11	10	14	0
86	8	5	8	11	9	14	86	8	5	8	11	9	14
85	10	8	7	6	11	0	86	11	7	8	7	11	0
85	11	8	7	4	12	0	93	11	7	5	9	13	0
123	12	11	11	20	17	0	127	14	11	11	19	18	0
86	10	7	10	10	9	0	96	9	8	10	11	11	0
89	5	10	10	4	12	0	95	12	9	10	5	14	0
88	13	8	4	8	10	0	88	11	7	6	10	10	0
115	11	9	11	15	14	19	118	12	9	13	17	14	18
104	10	8	10	9	23	0	109	9	9	15	14	25	0
91	6	10	11	5	14	0	90	7	7	10	4	13	0
120	13	15	14	9	16	0	130	14	12	15	14	16	0
98	15	11	11	13	10	10	89	12	10	9	12	10	10

(*continued on next page*)

Table A3 (*continued*)

C1	C2	C3	C4	C5	C6	C7	D1	D2	D3	D4	D5	D6	D7
134	16	10	12	19	19	16	140	16	11	16	19	19	18
85	10	10	6	5	11	0	85	11	9	6	7	11	0
111	12	17	9	12	14	0	124	13	17	12	14	17	0
96	8	11	12	9	14	0	97	8	11	12	10	14	0
96	8	10	8	8	13	0	118	14	12	13	11	13	0
142	14	13	14	17	20	0	145	14	13	15	18	20	0
85	10	10	6	8	10	0	88	11	8	7	7	12	0
100	11	10	11	8	14	0	119	12	10	15	12	19	0
90	8	10	11	6	13	0	102	9	10	10	10	14	0
129	12	9	13	17	19	19	132	12	9	13	17	19	19
85	10	8	6	8	11	0	85	10	8	6	8	11	0
95	8	9	11	8	12	0	102	8	11	12	10	12	0
95	10	8	8	11	14	12	110	12	9	11	14	16	18
127	12	10	14	15	20	0	138	13	12	15	17	20	0
84	6	9	9	9	17	0	106	11	11	11	11	16	0
81	8	9	7	11	12	7	102	13	11	10	11	14	16
128	9	14	15	11	17	0	125	10	13	14	10	18	0
91	15	11	9	12	12	9	91	15	11	9	12	12	9
88	12	8	7	7	12	0	95	10	9	6	7	13	0
102	9	10	12	13	12	0	109	10	11	12	15	15	0
90	10	9	8	8	13	0	99	13	9	8	9	15	0
96	9	10	11	7	13	0	107	10	10	13	10	14	0
89	12	10	9	12	10	10	98	15	11	11	13	10	9
90	10	9	9	11	6	16	91	10	9	9	11	8	14
112	13	11	13	8	16	0	109	13	11	13	10	16	0
91	9	6	8	14	11	9	115	112	10	11	17	14	19
83	9	7	9	7	10	0	88	10	9	9	8	10	0
108	12	10	10	14	16	14	127	15	11	11	19	18	19
86	7	8	8	6	15	0	123	11	14	16	9	19	0
85	10	10	6	13	8	7	86	10	10	7	13	8	7
93	8	11	13	10	10	0	95	9	11	13	10	11	0
95	12	12	8	7	14	0	96	12	12	8	8	14	0
111	10	10	12	14	16	19	112	12	8	16	15	18	19
93	9	8	10	9	10	12	94	9	8	10	9	11	12
82	10	6	8	12	12	10	93	12	10	9	13	13	10
87	10	10	9	12	9	10	87	10	8	9	11	12	10
88	6	12	11	5	11	0	93	6	12	13	6	12	0
90	11	10	10	8	10	0	95	11	10	11	10	11	0
87	10	10	8	7	10	0	95	10	11	10	7	13	0
88	11	10	6	4	11	0	88	9	9	9	4	13	0

Table A4

Correlation coefficients between retained primitive reflex (RPR) scores and individual WISC/WAIS subtest measures prior to reflex-integration intervention in individuals with autism spectrum disorder (n = 60). The Variable column lists each primitive reflex assessed. The Measure column lists the corresponding WISC/WAIS cognitive subtests examined. The r column presents Pearson correlation coefficients indicating the strength and direction of association between reflex scores and cognitive performance, where positive values denote higher reflex retention associated with higher test scores, and negative values denote inverse relationships. The p column reports significance levels, and 95 % CI (Lower/Upper) columns present Fisher's r-to-z transformed confidence limits for each correlation

Variable	Measure	*r*	*p*	95 % CI (Lower)	95 % CI (Upper)
ATNR-ASYMMT-L	−.295*	−0.295	0.050	−0.511	−0.044
ATNR-ASYMMT-L	−.295*	−0.295	0.050	−0.511	−0.044
ATNR-ASYMMT-L	0.085	0.085		−0.173	0.332
ATNR-ASYMMT-L	−0.199	−0.199		−0.431	0.058
ATNR-ASYMMT-L	−.283*	−0.283	0.050	−0.501	−0.031
ATNR-ASYMMT-L	−0.025	−0.025		−0.277	0.230
ATNR-ASYMMT-L	−0.025	−0.025		−0.277	0.230
ATNR-ASYMMT-L	−.356**	−0.356	0.010	−0.559	−0.112
ATNR-ASYMMT-L	0.161	0.161		−0.097	0.399
ATNR-ASYMMT-R	−.327**	−0.327	0.010	−0.536	−0.080
ATNR-ASYMMT-R	−.327**	−0.327	0.010	−0.536	−0.080
ATNR-ASYMMT-R	0.080	0.080		−0.178	0.327
ATNR-ASYMMT-R	−.236*	−0.236	0.050	−0.462	0.019
ATNR-ASYMMT-R	−.299*	−0.299	0.050	−0.514	−0.049
ATNR-ASYMMT-R	−0.044	−0.044		−0.295	0.212
ATNR-ASYMMT-R	−0.044	−0.044		−0.295	0.212
ATNR-ASYMMT-R	−.363**	−0.363	0.010	−0.565	−0.120

(*continued on next page*)

Table A4 (*continued*)

Variable	Measure	*r*	*p*	95 % CI (Lower)	95 % CI (Upper)
ATNR-ASYMMT-R	0.135	0.135		−0.123	0.376
STNR-ASYMMT-L	−.221*	−0.221	0.050	−0.450	0.035
STNR-ASYMMT-L	−.221*	−0.221	0.050	−0.450	0.035
STNR-ASYMMT-L	−0.097	−0.097		−0.342	0.161
STNR-ASYMMT-L	−0.171	−0.171		−0.407	0.087
STNR-ASYMMT-L	−.251*	−0.251	0.050	−0.475	0.003
STNR-ASYMMT-L	0.025	0.025		−0.230	0.277
STNR-ASYMMT-L	0.025	0.025		−0.230	0.277
STNR-ASYMMT-L	−.367**	−0.367	0.010	−0.568	−0.125
STNR-ASYMMT-L	0.177	0.177		−0.081	0.412
STNR-ASYMMT-R	−.221*	−0.221	0.050	−0.450	0.035
STNR-ASYMMT-R	−.221*	−0.221	0.050	−0.450	0.035
STNR-ASYMMT-R	−0.098	−0.098		−0.343	0.160
STNR-ASYMMT-R	−0.175	−0.175		−0.411	0.083
STNR-ASYMMT-R	−.250*	−0.250	0.050	−0.474	0.004
STNR-ASYMMT-R	0.031	0.031		−0.225	0.283
STNR-ASYMMT-R	0.031	0.031		−0.225	0.283
STNR-ASYMMT-R	−.367**	−0.367	0.010	−0.568	−0.125
STNR-ASYMMT-R	0.184	0.184		−0.073	0.418
SPINAL GALLANT-AYMMT-L	−.285*	−0.285	0.050	−0.503	−0.033
SPINAL GALLANT-AYMMT-L	−.285*	−0.285	0.050	−0.503	−0.033
SPINAL GALLANT-AYMMT-L	0.098	0.098		−0.160	0.343
SPINAL GALLANT-AYMMT-L	−0.114	−0.114		−0.358	0.144
SPINAL GALLANT-AYMMT-L	−.262*	−0.262	0.050	−0.484	−0.009
SPINAL GALLANT-AYMMT-L	0.026	0.026		−0.229	0.278
SPINAL GALLANT-AYMMT-L	0.026	0.026		−0.229	0.278
SPINAL GALLANT-AYMMT-L	−.301**	−0.301	0.010	−0.516	−0.051
SPINAL GALLANT-AYMMT-L	.229*	0.229	0.050	−0.026	0.456
SPINAL GALLANT-AYMMT-R	−.288*	−0.288	0.050	−0.505	−0.037
SPINAL GALLANT-AYMMT-R	−.288*	−0.288	0.050	−0.505	−0.037
SPINAL GALLANT-AYMMT-R	0.094	0.094		−0.164	0.340
SPINAL GALLANT-AYMMT-R	−0.128	−0.128		−0.370	0.130
SPINAL GALLANT-AYMMT-R	−.280*	−0.280	0.050	−0.498	−0.028
SPINAL GALLANT-AYMMT-R	0.033	0.033		−0.223	0.285
SPINAL GALLANT-AYMMT-R	0.033	0.033		−0.223	0.285
SPINAL GALLANT-AYMMT-R	−.318**	−0.318	0.010	−0.529	−0.070
SPINAL GALLANT-AYMMT-R	.233*	0.233	0.050	−0.022	0.460
PALMER-ASYMMT-L	−.257*	−0.257	0.050	−0.480	−0.003
PALMER-ASYMMT-L	−.257*	−0.257	0.050	−0.480	−0.003
PALMER-ASYMMT-L	0.111	0.111		−0.147	0.355
PALMER-ASYMMT-L	0.018	0.018		−0.237	0.271
PALMER-ASYMMT-L	−.257*	−0.257	0.050	−0.480	−0.003
PALMER-ASYMMT-L	0.086	0.086		−0.172	0.333
PALMER-ASYMMT-L	0.086	0.086		−0.172	0.333
PALMER-ASYMMT-L	−.306**	−0.306	0.010	−0.520	−0.056
PALMER-ASYMMT-L	0.205	0.205		−0.052	0.436
PALMER-ASYMMT-R	−.279*	−0.279	0.050	−0.498	−0.027
PALMER-ASYMMT-R	−.279*	−0.279	0.050	−0.498	−0.027
PALMER-ASYMMT-R	0.084	0.084		−0.174	0.331
PALMER-ASYMMT-R	−0.018	−0.018		−0.271	0.237
PALMER-ASYMMT-R	−.237*	−0.237	0.050	−0.463	0.018
PALMER-ASYMMT-R	0.085	0.085		−0.173	0.332
PALMER-ASYMMT-R	0.085	0.085		−0.173	0.332
PALMER-ASYMMT-R	−.302**	−0.302	0.010	−0.516	−0.052
PALMER-ASYMMT-R	0.184	0.184		−0.073	0.418
BABINSKI ASYMMT-L	−.249*	−0.249	0.050	−0.473	0.005
BABINSKI ASYMMT-L	−.249*	−0.249	0.050	−0.473	0.005
BABINSKI ASYMMT-L	0.041	0.041		−0.215	0.292
BABINSKI ASYMMT-L	−0.126	−0.126		−0.368	0.132
BABINSKI ASYMMT-L	−.255*	−0.255	0.050	−0.478	−0.001
BABINSKI ASYMMT-L	−0.039	−0.039		−0.290	0.217
BABINSKI ASYMMT-L	−0.039	−0.039		−0.290	0.217
BABINSKI ASYMMT-L	−.250*	−0.250	0.050	−0.474	0.004
BABINSKI ASYMMT-L	0.187	0.187		−0.070	0.421
BABINSKI ASYMMT-R	−.249*	−0.249	0.050	−0.473	0.005
BABINSKI ASYMMT-R	−.249*	−0.249	0.050	−0.473	0.005
BABINSKI ASYMMT-R	0.105	0.105		−0.153	0.350
BABINSKI ASYMMT-R	−0.184	−0.184		−0.418	0.073
BABINSKI ASYMMT-R	−.255*	−0.255	0.050	−0.478	−0.001
BABINSKI ASYMMT-R	−0.040	−0.040		−0.291	0.216

(*continued on next page*)

Table A4 (*continued*)

Variable	Measure	*r*	*p*	95 % CI (Lower)	95 % CI (Upper)
BABINSKI ASYMMT-R	−0.040	−0.040		−0.291	0.216
BABINSKI ASYMMT-R	−.364**	−0.364	0.010	−0.566	−0.121
BABINSKI ASYMMT-R	0.177	0.177		−0.081	0.412
ROOTING-ASYMMT-L	−0.081	−0.081		−0.328	0.177
ROOTING-ASYMMT-L	−0.081	−0.081		−0.328	0.177
ROOTING-ASYMMT-L	0.049	0.049		−0.208	0.299
ROOTING-ASYMMT-L	0.032	0.032		−0.224	0.284
ROOTING-ASYMMT-L	−0.066	−0.066		−0.315	0.191
ROOTING-ASYMMT-L	0.176	0.176		−0.082	0.412
ROOTING-ASYMMT-L	0.176	0.176		−0.082	0.412
ROOTING-ASYMMT-L	−.365**	−0.365	0.010	−0.566	−0.122
ROOTING-ASYMMT-L	.332**	0.332	0.010	0.085	0.540
ROOTING-ASYMMT-R	−0.079	−0.079		−0.326	0.179
ROOTING-ASYMMT-R	−0.079	−0.079		−0.326	0.179
ROOTING-ASYMMT-R	0.050	0.050		−0.207	0.300
ROOTING-ASYMMT-R	0.031	0.031		−0.225	0.283
ROOTING-ASYMMT-R	−0.066	−0.066		−0.315	0.191
ROOTING-ASYMMT-R	0.176	0.176		−0.082	0.412
ROOTING-ASYMMT-R	0.176	0.176		−0.082	0.412
ROOTING-ASYMMT-R	−.364**	−0.364	0.010	−0.566	−0.121
ROOTING-ASYMMT-R	.332**	0.332	0.010	0.085	0.540
LABYRINT ASYMMT-L	−0.128	−0.128		−0.370	0.130
LABYRINT ASYMMT-L	−0.128	−0.128		−0.370	0.130
LABYRINT ASYMMT-L	−0.109	−0.109		−0.353	0.149
LABYRINT ASYMMT-L	−0.151	−0.151		−0.390	0.107
LABYRINT ASYMMT-L	−0.197	−0.197		−0.429	0.060
LABYRINT ASYMMT-L	0.079	0.079		−0.179	0.326
LABYRINT ASYMMT-L	0.079	0.079		−0.179	0.326
LABYRINT ASYMMT-L	−.303**	−0.303	0.010	−0.517	−0.053
LABYRINT ASYMMT-L	0.187	0.187		−0.070	0.421
LABYRINT ASYMMT-R	−0.129	−0.129		−0.371	0.129
LABYRINT ASYMMT-R	−0.129	−0.129		−0.371	0.129
LABYRINT ASYMMT-R	−0.109	−0.109		−0.353	0.149
LABYRINT ASYMMT-R	−0.153	−0.153		−0.392	0.105
LABYRINT ASYMMT-R	−0.200	−0.200		−0.432	0.057
LABYRINT ASYMMT-R	0.084	0.084		−0.174	0.331
LABYRINT ASYMMT-R	0.084	0.084		−0.174	0.331
LABYRINT ASYMMT-R	−.303**	−0.303	0.010	−0.517	−0.053
LABYRINT ASYMMT-R	0.187	0.187		−0.070	0.421
MORO-ASYMMT-L	−0.150	−0.150		−0.389	0.108
MORO-ASYMMT-L	−0.150	−0.150		−0.389	0.108
MORO-ASYMMT-L	−0.069	−0.069		−0.317	0.188
MORO-ASYMMT-L	−0.147	−0.147		−0.387	0.111
MORO-ASYMMT-L	−0.054	−0.054		−0.304	0.203
MORO-ASYMMT-L	−0.004	−0.004		−0.258	0.250
MORO-ASYMMT-L	−0.004	−0.004		−0.258	0.250
MORO-ASYMMT-L	−.357**	−0.357	0.010	−0.560	−0.113
MORO-ASYMMT-L	0.110	0.110		−0.148	0.354
MORO-ASYMMT-R	−0.115	−0.115		−0.358	0.143
MORO-ASYMMT-R	−0.115	−0.115		−0.358	0.143
MORO-ASYMMT-R	−0.071	−0.071		−0.319	0.186
MORO-ASYMMT-R	−0.185	−0.185		−0.419	0.072
MORO-ASYMMT-R	−0.002	−0.002		−0.256	0.252
MORO-ASYMMT-R	0.064	0.064		−0.193	0.313

(*continued on next page*)

Table A4 (*continued*)

Variable	Measure	*r*	*p*	95 % CI (Lower)	95 % CI (Upper)
MORO-ASYMMT-R	0.064	0.064		−0.193	0.313
MORO-ASYMMT-R	−.321**	−0.321	0.010	−0.532	−0.073
MORO-ASYMMT-R	0.172	0.172		−0.086	0.408
ATNR-SYMMT-L	−.339**	−0.339	0.010	−0.546	−0.093
ATNR-SYMMT-L	−.339**	−0.339	0.010	−0.546	−0.093
ATNR-SYMMT-L	−.217*	−0.217	0.050	−0.446	0.039
ATNR-SYMMT-L	−0.196	−0.196		−0.429	0.061
ATNR-SYMMT-L	−.415**	−0.415	0.010	−0.605	−0.180
ATNR-SYMMT-L	−.387**	−0.387	0.010	−0.584	−0.148
ATNR-SYMMT-L	−.387**	−0.387	0.010	−0.584	−0.148
ATNR-SYMMT-L	−0.140	−0.140		−0.380	0.118
ATNR-SYMMT-L	−0.151	−0.151		−0.390	0.107
ATNR-SYMMT-R	0.004	0.004		−0.250	0.258
ATNR-SYMMT-R	0.004	0.004		−0.250	0.258
ATNR-SYMMT-R	0.048	0.048		−0.208	0.298
ATNR-SYMMT-R	0.066	0.066		−0.191	0.315
ATNR-SYMMT-R	0.000	0.000		−0.254	0.254
ATNR-SYMMT-R	0.001	0.001		−0.253	0.255
ATNR-SYMMT-R	0.001	0.001		−0.253	0.255
ATNR-SYMMT-R	0.144	0.144		−0.114	0.384
ATNR-SYMMT-R	0.125	0.125		−0.133	0.367
ATNR-SYMMT-R	−.345**	−0.345	0.010	−0.551	−0.100
ATNR-SYMMT-R	−.345**	−0.345	0.010	−0.551	−0.100
ATNR-SYMMT-R	−0.212	−0.212		−0.442	0.044
ATNR-SYMMT-R	−0.187	−0.187		−0.421	0.070
ATNR-SYMMT-R	−.422**	−0.422	0.010	−0.611	−0.188
ATNR-SYMMT-R	−.392**	−0.392	0.010	−0.587	−0.153
ATNR-SYMMT-R	−.392**	−0.392	0.010	−0.587	−0.153
ATNR-SYMMT-R	−0.130	−0.130		−0.372	0.128
ATNR-SYMMT-R	−0.169	−0.169		−0.406	0.089
STNR-SYMMET-L	−.505**	−0.505	0.010	−0.673	−0.288
STNR-SYMMET-L	−.505**	−0.505	0.010	−0.673	−0.288
STNR-SYMMET-L	−0.109	−0.109		−0.353	0.149
STNR-SYMMET-L	−.270*	−0.270	0.050	−0.490	−0.017
STNR-SYMMET-L	−.527**	−0.527	0.010	−0.689	−0.315

(*continued on next page*)

Table A4 (*continued*)

Variable	Measure	*r*	*p*	95 % CI (Lower)	95 % CI (Upper)
STNR-SYMMET-L	−.393**	−0.393	0.010	−0.588	−0.154
STNR-SYMMET-L	−.393**	−0.393	0.010	−0.588	−0.154
STNR-SYMMET-L	−.276*	−0.276	0.050	−0.495	−0.024
STNR-SYMMET-L	−0.122	−0.122		−0.365	0.136
STNR-SYMMET-R	−.513**	−0.513	0.010	−0.679	−0.298
STNR-SYMMET-R	−.513**	−0.513	0.010	−0.679	−0.298
STNR-SYMMET-R	−0.100	−0.100		−0.345	0.158
STNR-SYMMET-R	−.258*	−0.258	0.050	−0.480	−0.004
STNR-SYMMET-R	−.535**	−0.535	0.010	−0.695	−0.325
STNR-SYMMET-R	−.398**	−0.398	0.010	−0.592	−0.160
STNR-SYMMET-R	−.398**	−0.398	0.010	−0.592	−0.160
STNR-SYMMET-R	−.267*	−0.267	0.050	−0.488	−0.014
STNR-SYMMET-R	−0.142	−0.142		−0.382	0.116
SPINAL GALLANT-SYMMET-L	−.312**	−0.312	0.010	−0.524	−0.063
SPINAL GALLANT-SYMMET-L	−.312**	−0.312	0.010	−0.524	−0.063
SPINAL GALLANT-SYMMET-L	−0.184	−0.184		−0.418	0.073
SPINAL GALLANT-SYMMET-L	−.294*	−0.294	0.050	−0.510	−0.043
SPINAL GALLANT-SYMMET-L	−.420**	−0.420	0.010	−0.609	−0.186
SPINAL GALLANT-SYMMET-L	−.386**	−0.386	0.010	−0.583	−0.146
SPINAL GALLANT-SYMMET-L	−.386**	−0.386	0.010	−0.583	−0.146
SPINAL GALLANT-SYMMET-L	−.226*	−0.226	0.050	−0.454	0.030
SPINAL GALLANT-SYMMET-L	−0.184	−0.184		−0.418	0.073
SPINAL GALLANT-SYMMET-R	−.312**	−0.312	0.010	−0.524	−0.063
SPINAL GALLANT-SYMMET-R	−.312**	−0.312	0.010	−0.524	−0.063
SPINAL GALLANT-SYMMET-R	−0.184	−0.184		−0.418	0.073
SPINAL GALLANT-SYMMET-R	−.294*	−0.294	0.050	−0.510	−0.043
SPINAL GALLANT-SYMMET-R	−.420**	−0.420	0.010	−0.609	−0.186
SPINAL GALLANT-SYMMET-R	−.386**	−0.386	0.010	−0.583	−0.146
SPINAL GALLANT-SYMMET-R	−.386**	−0.386	0.010	−0.583	−0.146
SPINAL GALLANT-SYMMET-R	−.226*	−0.226	0.050	−0.454	0.030
SPINAL GALLANT-SYMMET-R	−0.184	−0.184		−0.418	0.073
PALMER-SYMMET	−.402**	−0.402	0.010	−0.595	−0.165
PALMER-SYMMET	−.402**	−0.402	0.010	−0.595	−0.165
PALMER-SYMMET	−0.183	−0.183		−0.418	0.074
PALMER-SYMMET	−.276*	−0.276	0.050	−0.495	−0.024
PALMER-SYMMET	−.466**	−0.466	0.010	−0.644	−0.241
PALMER-SYMMET	−.492**	−0.492	0.010	−0.663	−0.272
PALMER-SYMMET	−.492**	−0.492	0.010	−0.663	−0.272
PALMER-SYMMET	−0.173	−0.173		−0.409	0.085

(*continued on next page*)

Table A4 (*continued*)

Variable	Measure	*r*	*p*	95 % CI (Lower)	95 % CI (Upper)
PALMER-SYMMET	−.252*	−0.252	0.050	−0.476	0.002
BABINSKI SYMMET	−.332**	−0.332	0.010	−0.540	−0.085
BABINSKI SYMMET	−.332**	−0.332	0.010	−0.540	−0.085
BABINSKI SYMMET	−0.098	−0.098		−0.343	0.160
BABINSKI SYMMET	−0.174	−0.174		−0.410	0.084
BABINSKI SYMMET	−.422**	−0.422	0.010	−0.611	−0.188
BABINSKI SYMMET	−.460**	−0.460	0.010	−0.639	−0.233
BABINSKI SYMMET	−.460**	−0.460	0.010	−0.639	−0.233
BABINSKI SYMMET	−0.098	−0.098		−0.343	0.160
BABINSKI SYMMET	−.270*	−0.270	0.050	−0.490	−0.017
ROOTING SYMMET	−.539**	−0.539	0.010	−0.697	−0.330
ROOTING SYMMET	−.539**	−0.539	0.010	−0.697	−0.330
ROOTING SYMMET	−0.116	−0.116		−0.359	0.142
ROOTING SYMMET	−.302**	−0.302	0.010	−0.516	−0.052
ROOTING SYMMET	−.588**	−0.588	0.010	−0.733	−0.393
ROOTING SYMMET	−.502**	−0.502	0.010	−0.670	−0.284
ROOTING SYMMET	−.502**	−0.502	0.010	−0.670	−0.284
ROOTING SYMMET	−0.195	−0.195		−0.428	0.062
ROOTING SYMMET	−.257*	−0.257	0.050	−0.480	−0.003
LABYRINT SYMMET-L	−.265*	−0.265	0.050	−0.486	−0.012
LABYRINT SYMMET-L	−.265*	−0.265	0.050	−0.486	−0.012
LABYRINT SYMMET-L	−0.064	−0.064		−0.313	0.193
LABYRINT SYMMET-L	−0.134	−0.134		−0.375	0.124
LABYRINT SYMMET-L	−.258*	−0.258	0.050	−0.480	−0.004
LABYRINT SYMMET-L	−.252*	−0.252	0.050	−0.476	0.002
LABYRINT SYMMET-L	−.252*	−0.252	0.050	−0.476	0.002
LABYRINT SYMMET-L	−0.198	−0.198		−0.430	0.059
LABYRINT SYMMET-L	−0.116	−0.116		−0.359	0.142
LABYRINT SYMMET-R	−.263*	−0.263	0.050	−0.485	−0.010
LABYRINT SYMMET-R	−.263*	−0.263	0.050	−0.485	−0.010
LABYRINT SYMMET-R	−0.064	−0.064		−0.313	0.193
LABYRINT SYMMET-R	−0.136	−0.136		−0.377	0.122
LABYRINT SYMMET-R	−.257*	−0.257	0.050	−0.480	−0.003
LABYRINT SYMMET-R	−.257*	−0.257	0.050	−0.480	−0.003
LABYRINT SYMMET-R	−.257*	−0.257	0.050	−0.480	−0.003
LABYRINT SYMMET-R	−0.193	−0.193		−0.426	0.064
LABYRINT SYMMET-R	−0.122	−0.122		−0.365	0.136
MORO-SYMMET	−0.196	−0.196		−0.429	0.061
MORO-SYMMET	−0.196	−0.196		−0.429	0.061
MORO-SYMMET	−0.044	−0.044		−0.295	0.212
MORO-SYMMET	−0.137	−0.137		−0.378	0.121
MORO-SYMMET	−.339**	−0.339	0.010	−0.546	−0.093
MORO-SYMMET	−.342**	−0.342	0.010	−0.548	−0.096
MORO-SYMMET	−.342**	−0.342	0.010	−0.548	−0.096
MORO-SYMMET	−0.006	−0.006		−0.260	0.248
MORO-SYMMET	−.298*	−0.298	0.050	−0.513	−0.048

Table A5

Correlation coefficients between retained primitive reflex (RPR) scores and individual WISC/WAIS subtest measures following reflex-integration intervention in individuals with autism spectrum disorder (n = 60). The Variable column lists each primitive reflex assessed. The Measure column lists the corresponding WISC/WAIS cognitive subtests examined. The r column presents Pearson correlation coefficients indicating the strength and direction of associations observed after intervention, where positive values denote higher reflex retention associated with higher test scores, and negative values denote inverse relationships. The p column reports significance levels derived from the original asterisk coding ($p < 0.05$, $p < 0.01$, $p < 0.001$). The 95 % CI (Lower/Upper) columns provide Fisher's *r-to-z* transformed confidence limits for each correlation

Variable	Measure	*r*	*p*	95 % CI (Lower)	95 % CI (Upper)
ATNR-ASYMMT-L	IQ WISC/ WAIS				
ATNR-ASYMMT-L	IQ WISC/ WAIS	−0.300	0.050	−0.515	−0.050
ATNR-ASYMMT-L	PIC COMP	−0.021		−0.273	0.234
ATNR-ASYMMT-L	BLOCK DESIGN	−0.186		−0.420	0.071
ATNR-ASYMMT-L	MATRIX REASON	−0.341	0.010	−0.548	−0.095
ATNR-ASYMMT-L	INFO	−0.080		−0.327	0.178
ATNR-ASYMMT-L	SIMILAR	−0.330	0.010	−0.539	−0.083
ATNR-ASYMMT-L	DIGIT SPAN	−0.330	0.010	−0.539	−0.083
ATNR-ASYMMT-L	DIGIT SPAN	0.245	0.050	−0.010	0.470

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Table A5 (*continued*)

Variable	Measure	*r*	*p*	95 % CI (Lower)	95 % CI (Upper)
ATNR-ASYMMT-R	IQ WISC/ WAIS				
ATNR-ASYMMT-R	IQ WISC/ WAIS	−0.278	0.050	−0.497	−0.026
ATNR-ASYMMT-R	PIC COMP	0.019		−0.236	0.272
ATNR-ASYMMT-R	BLOCK DESIGN	−0.233	0.050	−0.460	0.022
ATNR-ASYMMT-R	MATRIX REASON	−0.343	0.010	−0.549	−0.098
ATNR-ASYMMT-R	INFO	−0.031		−0.283	0.225
ATNR-ASYMMT-R	SIMILAR	−0.326	0.010	−0.536	−0.079
ATNR-ASYMMT-R	DIGIT SPAN	−0.326	0.010	−0.536	−0.079
ATNR-ASYMMT-R	DIGIT SPAN	0.254	0.050	0.000	0.477
STNR-ASYMMT-L	IQ WISC/ WAIS				
STNR-ASYMMT-L	IQ WISC/ WAIS	−0.266	0.050	−0.487	−0.013
STNR-ASYMMT-L	PIC COMP	−0.112		−0.356	0.146
STNR-ASYMMT-L	BLOCK DESIGN	−0.181		−0.416	0.076
STNR-ASYMMT-L	MATRIX REASON	−0.209		−0.440	0.047
STNR-ASYMMT-L	INFO	−0.067		−0.316	0.190
STNR-ASYMMT-L	SIMILAR	−0.250	0.050	−0.474	0.004
STNR-ASYMMT-L	DIGIT SPAN	−0.250	0.050	−0.474	0.004
STNR-ASYMMT-L	DIGIT SPAN	0.224	0.050	−0.032	0.452
STNR-ASYMMT-R					
STNR-ASYMMT-R	IQ WISC/ WAIS				
STNR-ASYMMT-R	IQ WISC/ WAIS	−0.267	0.050	−0.488	−0.014
STNR-ASYMMT-R	PIC COMP	−0.081		−0.328	0.177
STNR-ASYMMT-R	BLOCK DESIGN	−0.240	0.050	−0.466	0.015
STNR-ASYMMT-R	MATRIX REASON	−0.208		−0.439	0.048
STNR-ASYMMT-R	INFO	−0.001		−0.255	0.253
STNR-ASYMMT-R	SIMILAR	−0.261	0.050	−0.483	−0.008
STNR-ASYMMT-R	DIGIT SPAN	−0.261	0.050	−0.483	−0.008
STNR-ASYMMT-R	DIGIT SPAN	0.279	0.050	0.027	0.498
SPINAL GALLANT-AYMMT-L	IQ WISC/ WAIS				
SPINAL GALLANT-AYMMT-L	IQ WISC/ WAIS	−0.279	0.050	−0.498	−0.027
SPINAL GALLANT-AYMMT-L	PIC COMP	0.114		−0.144	0.358
SPINAL GALLANT-AYMMT-L	BLOCK DESIGN	−0.059		−0.308	0.198
SPINAL GALLANT-AYMMT-L	MATRIX REASON	−0.250	0.050	−0.474	0.004
SPINAL GALLANT-AYMMT-L	INFO	−0.006		−0.260	0.248
SPINAL GALLANT-AYMMT-L	SIMILAR	−0.286	0.050	−0.503	−0.035
SPINAL GALLANT-AYMMT-L	DIGIT SPAN	−0.286	0.050	−0.503	−0.035
SPINAL GALLANT-AYMMT-L	DIGIT SPAN	0.297	0.050	0.047	0.512
SPINAL GALLANT-AYMMT-R	IQ WISC/ WAIS				
SPINAL GALLANT-AYMMT-R	IQ WISC/ WAIS	−0.273	0.050	−0.493	−0.020
SPINAL GALLANT-AYMMT-R	PIC COMP	0.142		−0.116	0.382
SPINAL GALLANT-AYMMT-R	BLOCK DESIGN	−0.089		−0.335	0.169
SPINAL GALLANT-AYMMT-R	MATRIX REASON	−0.254	0.050	−0.477	−0.000
SPINAL GALLANT-AYMMT-R	INFO	0.042		−0.214	0.293
SPINAL GALLANT-AYMMT-R	SIMILAR	−0.279	0.050	−0.498	−0.027
SPINAL GALLANT-AYMMT-R	DIGIT SPAN	−0.279	0.050	−0.498	−0.027
SPINAL GALLANT-AYMMT-R	DIGIT SPAN	0.324	0.010	0.076	0.534
PALMER-ASYMMT-L	IQ WISC/ WAIS				
PALMER-ASYMMT-L	IQ WISC/ WAIS	−0.272	0.050	−0.492	−0.019
PALMER-ASYMMT-L	PIC COMP	−0.007		−0.260	0.247
PALMER-ASYMMT-L	BLOCK DESIGN	−0.053		−0.303	0.204
PALMER-ASYMMT-L	MATRIX REASON	−0.274	0.050	−0.494	−0.022
PALMER-ASYMMT-L	INFO	0.031		−0.225	0.283
PALMER-ASYMMT-L	SIMILAR	−0.286	0.050	−0.503	−0.035
PALMER-ASYMMT-L	DIGIT SPAN	−0.286	0.050	−0.503	−0.035
PALMER-ASYMMT-L	DIGIT SPAN	0.220	0.050	−0.036	0.449
PALMER-ASYMMT-R	IQ WISC/ WAIS				

(*continued on next page*)

Table A5 (*continued*)

Variable	Measure	*r*	*p*	95 % CI (Lower)	95 % CI (Upper)
PALMER-ASYMMT-R	IQ WISC/ WAIS	−0.226	0.050	−0.454	0.030
PALMER-ASYMMT-R	PIC COMP	0.019		−0.236	0.272
PALMER-ASYMMT-R	BLOCK DESIGN	−0.047		−0.297	0.209
PALMER-ASYMMT-R	MATRIX REASON	−0.245	0.050	−0.470	0.010
PALMER-ASYMMT-R	INFO	0.098		−0.160	0.343
PALMER-ASYMMT-R	SIMILAR	−0.293	0.050	−0.509	−0.042
PALMER-ASYMMT-R	DIGIT SPAN	−0.293	0.050	−0.509	−0.042
PALMER-ASYMMT-R	DIGIT SPAN	0.242	0.050	−0.013	0.467
BABINSKI ASYMMT-L	IQ WISC/ WAIS				
BABINSKI ASYMMT-L	IQ WISC/ WAIS	−0.293	0.050	−0.509	−0.042
BABINSKI ASYMMT-L	PIC COMP	−0.041		−0.292	0.215
BABINSKI ASYMMT-L	BLOCK DESIGN	−0.155		−0.393	0.103
BABINSKI ASYMMT-L	MATRIX REASON	−0.306	0.010	−0.520	−0.056
BABINSKI ASYMMT-L	INFO	−0.089		−0.335	0.169
BABINSKI ASYMMT-L	SIMILAR	−0.259	0.050	−0.481	−0.005
BABINSKI ASYMMT-L	DIGIT SPAN	−0.259	0.050	−0.481	−0.005
BABINSKI ASYMMT-L	DIGIT SPAN	0.164		−0.094	0.401
BABINSKI ASYMMT-R	IQ WISC/ WAIS				
BABINSKI ASYMMT-R	IQ WISC/ WAIS	−0.261	0.050	−0.483	−0.008
BABINSKI ASYMMT-R	PIC COMP	0.000		−0.254	0.254
BABINSKI ASYMMT-R	BLOCK DESIGN	−0.205		−0.436	0.052
BABINSKI ASYMMT-R	MATRIX REASON	−0.306	0.010	−0.520	−0.056
BABINSKI ASYMMT-R	INFO	−0.015		−0.268	0.240
BABINSKI ASYMMT-R	SIMILAR	−0.336	0.010	−0.544	−0.090
BABINSKI ASYMMT-R	DIGIT SPAN	−0.336	0.010	−0.544	−0.090
BABINSKI ASYMMT-R	DIGIT SPAN	0.204		−0.053	0.435
ROOTING-ASYMMT-L	IQ WISC/ WAIS				
ROOTING-ASYMMT-L	IQ WISC/ WAIS	−0.309	0.010	−0.522	−0.060
ROOTING-ASYMMT-L	PIC COMP	−0.161		−0.399	0.097
ROOTING-ASYMMT-L	BLOCK DESIGN	−0.158		−0.396	0.100
ROOTING-ASYMMT-L	MATRIX REASON	−0.211		−0.441	0.045
ROOTING-ASYMMT-L	INFO	0.037		−0.219	0.288
ROOTING-ASYMMT-L	SIMILAR	−0.326	0.010	−0.536	−0.079
ROOTING-ASYMMT-L	DIGIT SPAN	−0.326	0.010	−0.536	−0.079
ROOTING-ASYMMT-L	DIGIT SPAN	0.308	0.010	0.059	0.521
ROOTING-ASYMMT-R	IQ WISC/ WAIS				
ROOTING-ASYMMT-R	IQ WISC/ WAIS	−0.309	0.010	−0.522	−0.060
ROOTING-ASYMMT-R	PIC COMP	−0.159		−0.397	0.099
ROOTING-ASYMMT-R	BLOCK DESIGN	−0.155		−0.393	0.103
ROOTING-ASYMMT-R	MATRIX REASON	−0.212		−0.442	0.044
ROOTING-ASYMMT-R	INFO	0.038		−0.218	0.289
ROOTING-ASYMMT-R	SIMILAR	−0.328	0.010	−0.537	−0.081
ROOTING-ASYMMT-R	DIGIT SPAN	−0.328	0.010	−0.537	−0.081
ROOTING-ASYMMT-R	DIGIT SPAN	0.307	0.010	0.058	0.520
LABYRINT ASYMMT-L	IQ WISC/ WAIS				
LABYRINT ASYMMT-L	IQ WISC/ WAIS	−0.123		−0.366	0.135
LABYRINT ASYMMT-L	PIC COMP	−0.074		−0.322	0.183
LABYRINT ASYMMT-L	BLOCK DESIGN	−0.035		−0.286	0.221
LABYRINT ASYMMT-L	MATRIX REASON	−0.126		−0.368	0.132
LABYRINT ASYMMT-L	INFO	−0.008		−0.261	0.246
LABYRINT ASYMMT-L	SIMILAR	−0.187		−0.421	0.070
LABYRINT ASYMMT-L	DIGIT SPAN	−0.187		−0.421	0.070
LABYRINT ASYMMT-L	DIGIT SPAN	0.231	0.050	−0.024	0.458
LABYRINT ASYMMT-R	IQ WISC/ WAIS				
LABYRINT ASYMMT-R	IQ WISC/ WAIS	−0.108		−0.352	0.150
LABYRINT ASYMMT-R	PIC COMP	−0.027		−0.279	0.228
LABYRINT ASYMMT-R	BLOCK DESIGN	−0.090		−0.336	0.168

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Table A5 (*continued*)

Variable	Measure	*r*	*p*	95 % CI (Lower)	95 % CI (Upper)
LABYRINT ASYMMT-R	MATRIX REASON	−0.118		−0.361	0.140
LABYRINT ASYMMT-R	INFO	0.088		−0.170	0.334
LABYRINT ASYMMT-R	SIMILAR	−0.196		−0.429	0.061
LABYRINT ASYMMT-R	DIGIT SPAN	−0.196		−0.429	0.061
LABYRINT ASYMMT-R	DIGIT SPAN	0.314	0.010	0.065	0.526
MORO-ASYMMT-L	IQ WISC/WAIS				
MORO-ASYMMT-L	IQ WISC/WAIS	−0.180		−0.415	0.077
MORO-ASYMMT-L	PIC COMP	−0.266	0.050	−0.487	−0.013
MORO-ASYMMT-L	BLOCK DESIGN	−0.290	0.050	−0.507	−0.039
MORO-ASYMMT-L	MATRIX REASON	−0.117		−0.360	0.141
MORO-ASYMMT-L	INFO	−0.039		−0.290	0.217
MORO-ASYMMT-L	SIMILAR	−0.229	0.050	−0.456	0.026
MORO-ASYMMT-L	DIGIT SPAN	−0.229	0.050	−0.456	0.026
MORO-ASYMMT-L	DIGIT SPAN	0.166		−0.092	0.403
MORO-ASYMMT-R	IQ WISC/WAIS				
MORO-ASYMMT-R	IQ WISC/WAIS	−0.197		−0.429	0.060
MORO-ASYMMT-R	PIC COMP	−0.199		−0.431	0.058
MORO-ASYMMT-R	BLOCK DESIGN	−0.235	0.050	−0.461	0.020
MORO-ASYMMT-R	MATRIX REASON	−0.109		−0.353	0.149
MORO-ASYMMT-R	INFO	−0.059		−0.308	0.198
MORO-ASYMMT-R	SIMILAR	−0.171		−0.407	0.087
MORO-ASYMMT-R	DIGIT SPAN	−0.171		−0.407	0.087
MORO-ASYMMT-R	DIGIT SPAN	0.256	0.050	0.002	0.479
ATNR-SYMMT-L	IQ WISC/WAIS				
ATNR-SYMMT-L	IQ WISC/WAIS	−0.314	0.010	−0.526	−0.065
ATNR-SYMMT-L	PIC COMP	0.037		−0.219	0.288
ATNR-SYMMT-L	BLOCK DESIGN	−0.203		−0.435	0.054
ATNR-SYMMT-L	MATRIX REASON	−0.346	0.010	−0.551	−0.101
ATNR-SYMMT-L	INFO	−0.402	0.010	−0.595	−0.165
ATNR-SYMMT-L	SIMILAR	−0.241	0.050	−0.466	0.014
ATNR-SYMMT-L	DIGIT SPAN	−0.241	0.050	−0.466	0.014
ATNR-SYMMT-L	DIGIT SPAN	−0.146		−0.386	0.112
ATNR-SYMMT-R					
ATNR-SYMMT-R	IQ WISC/WAIS				
ATNR-SYMMT-R	IQ WISC/WAIS	−0.314	0.010	−0.526	−0.065
ATNR-SYMMT-R	PIC COMP	0.037		−0.219	0.288
ATNR-SYMMT-R	BLOCK DESIGN	−0.203		−0.435	0.054
ATNR-SYMMT-R	MATRIX REASON	−0.346	0.010	−0.551	−0.101

(*continued on next page*)

Table A5 (*continued*)

Variable	Measure	*r*	*p*	95 % CI (Lower)	95 % CI (Upper)
ATNR-SYMMT-R	INFO	−0.402	0.010	−0.595	−0.165
ATNR-SYMMT-R	SIMILAR	−0.241	0.050	−0.466	0.014
ATNR-SYMMT-R	DIGIT SPAN	−0.241	0.050	−0.466	0.014
ATNR-SYMMT-R	DIGIT SPAN	−0.146		−0.386	0.112
STNR-SYMMET-L	IQ WISC/WAIS				
STNR-SYMMET-L	IQ WISC/WAIS	−0.461	0.010	−0.640	−0.235
STNR-SYMMET-L	PIC COMP	0.042		−0.214	0.293
STNR-SYMMET-L	BLOCK DESIGN	−0.266	0.050	−0.487	−0.013
STNR-SYMMET-L	MATRIX REASON	−0.550	0.010	−0.705	−0.344
STNR-SYMMET-L	INFO	−0.381	0.010	−0.579	−0.141
STNR-SYMMET-L	SIMILAR	−0.428	0.010	−0.615	−0.195
STNR-SYMMET-L	DIGIT SPAN	−0.428	0.010	−0.615	−0.195
STNR-SYMMET-L	DIGIT SPAN	−0.071		−0.319	0.186
STNR-SYMMET-R	IQ WISC/WAIS				
STNR-SYMMET-R	IQ WISC/WAIS	−0.454	0.010	−0.635	−0.226
STNR-SYMMET-R	PIC COMP	0.056		−0.201	0.306
STNR-SYMMET-R	BLOCK DESIGN	−0.254	0.050	−0.477	−0.000
STNR-SYMMET-R	MATRIX REASON	−0.548	0.010	−0.704	−0.342
STNR-SYMMET-R	INFO	−0.376	0.010	−0.575	−0.135
STNR-SYMMET-R	SIMILAR	−0.417	0.010	−0.607	−0.182
STNR-SYMMET-R	DIGIT SPAN	−0.417	0.010	−0.607	−0.182
STNR-SYMMET-R	DIGIT SPAN	−0.084		−0.331	0.174
SPINAL GALLANT-SYMMET-L	IQ WISC/WAIS				
SPINAL GALLANT-SYMMET-L	IQ WISC/WAIS	−0.304	0.010	−0.518	−0.054
SPINAL GALLANT-SYMMET-L	PIC COMP	−0.049		−0.299	0.208
SPINAL GALLANT-SYMMET-L	BLOCK DESIGN	−0.302	0.010	−0.516	−0.052
SPINAL GALLANT-SYMMET-L	MATRIX REASON	−0.408	0.010	−0.600	−0.172
SPINAL GALLANT-SYMMET-L	INFO	−0.443	0.010	−0.626	−0.213
SPINAL GALLANT-SYMMET-L	SIMILAR	−0.274	0.050	−0.494	−0.022
SPINAL GALLANT-SYMMET-L	DIGIT SPAN	−0.274	0.050	−0.494	−0.022
SPINAL GALLANT-SYMMET-L	DIGIT SPAN	−0.211		−0.441	0.045
SPINALGALLANT-SYMMET-R	IQ WISC/WAIS				
SPINALGALLANT-SYMMET-R	IQ WISC/WAIS	−0.304	0.010	−0.518	−0.054
SPINALGALLANT-SYMMET-R	PIC COMP	−0.049		−0.299	0.208
SPINAL GALLANT-SYMMET-R	BLOCK DESIGN	−0.302	0.010	−0.516	−0.052
SPINAL GALLANT-SYMMET-R	MATRIX REASON	−0.408	0.010	−0.600	−0.172
SPINAL GALLANT-SYMMET-R	INFO	−0.443	0.010	−0.626	−0.213

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Table A5 (*continued*)

Variable	Measure	*r*	*p*	95 % CI (Lower)	95 % CI (Upper)
SPINAL GALLANT-SYMMET-R	SIMILAR	−0.274	0.050	−0.494	−0.022
SPINAL GALLANT-SYMMET-R	DIGIT SPAN	−0.274	0.050	−0.494	−0.022
SPINAL GALLANT-SYMMET-R	DIGIT SPAN	−0.211		−0.441	0.045
PALMER-SYMMET	IQ WISC/ WAIS				
PALMER-SYMMET	IQ WISC/ WAIS	−0.329	0.010	−0.538	−0.082
PALMER-SYMMET	PIC COMP	0.063		−0.194	0.312
PALMER-SYMMET	BLOCK DESIGN	−0.262	0.050	−0.484	−0.009
PALMER-SYMMET	MATRIX REASON	−0.397	0.010	−0.591	−0.159
PALMER-SYMMET	INFO	−0.484	0.010	−0.657	−0.262
PALMER-SYMMET	SIMILAR	−0.243	0.050	−0.468	0.012
PALMER-SYMMET	DIGIT SPAN	−0.243	0.050	−0.468	0.012
PALMER-SYMMET	DIGIT SPAN	−0.226	0.050	−0.454	0.030
BABINSKI SYMMET	IQ WISC/ WAIS				
BABINSKI SYMMET	IQ WISC/ WAIS	−0.302	0.050	−0.516	−0.052
BABINSKI SYMMET	PIC COMP	0.102		−0.156	0.347
BABINSKI SYMMET	BLOCK DESIGN	−0.195		−0.428	0.062
BABINSKI SYMMET	MATRIX REASON	−0.363	0.010	−0.565	−0.120
BABINSKI SYMMET	INFO	−0.474	0.010	−0.650	−0.250
BABINSKI SYMMET	SIMILAR	−0.213		−0.443	0.043
BABINSKI SYMMET	DIGIT SPAN	−0.213		−0.443	0.043
BABINSKI SYMMET	DIGIT SPAN	−0.257	0.050	−0.480	−0.003
ROOTING SYMMET	IQ WISC/ WAIS				
ROOTING SYMMET	IQ WISC/ WAIS	−0.388	0.010	−0.584	−0.149
ROOTING SYMMET	PIC COMP	0.139		−0.119	0.380
ROOTING SYMMET	BLOCK DESIGN	−0.234	0.050	−0.461	0.021
ROOTING SYMMET	MATRIX REASON	−0.485	0.010	−0.658	−0.264
ROOTING SYMMET	INFO	−0.471	0.010	−0.647	−0.247
ROOTING SYMMET	SIMILAR	−0.309	0.010	−0.522	−0.060
ROOTING SYMMET	DIGIT SPAN	−0.309	0.010	−0.522	−0.060
ROOTING SYMMET	DIGIT SPAN	−0.220	0.050	−0.449	0.036
LABYRINT SYMMET-L	IQ WISC/ WAIS				
LABYRINT SYMMET-L	IQ WISC/ WAIS	−0.228	0.050	−0.456	0.028
LABYRINT SYMMET-L	PIC COMP	0.044		−0.212	0.295
LABYRINT SYMMET-L	BLOCK DESIGN	−0.126		−0.368	0.132
LABYRINT SYMMET-L	MATRIX REASON	−0.164		−0.401	0.094
LABYRINT SYMMET-L	INFO	−0.280	0.050	−0.498	−0.028
LABYRINT SYMMET-L	SIMILAR	−0.209		−0.440	0.047
LABYRINT SYMMET-L	DIGIT SPAN	−0.209		−0.440	0.047
LABYRINT SYMMET-L	DIGIT SPAN	−0.105		−0.350	0.153
LABYRINT SYMMET-R	IQ WISC/ WAIS				
LABYRINT SYMMET-R	IQ WISC/ WAIS	−0.226	0.050	−0.454	0.030
LABYRINT SYMMET-R	PIC COMP	0.047		−0.209	0.297
LABYRINT SYMMET-R	BLOCK DESIGN	−0.123		−0.366	0.135
LABYRINT SYMMET-R	MATRIX REASON	−0.163		−0.400	0.095
LABYRINT SYMMET-R	INFO	−0.283	0.050	−0.501	−0.031
LABYRINT SYMMET-R	SIMILAR	−0.209		−0.440	0.047
LABYRINT SYMMET-R	DIGIT SPAN	−0.209		−0.440	0.047
LABYRINT SYMMET-R	DIGIT SPAN	−0.109		−0.353	0.149
MORO-SYMMET	IQ WISC/ WAIS				
MORO-SYMMET	IQ WISC/ WAIS	−0.187		−0.421	0.070
MORO-SYMMET	PIC COMP	−0.043		−0.294	0.213
MORO-SYMMET	BLOCK DESIGN	−0.122		−0.365	0.136
MORO-SYMMET	MATRIX REASON	−0.235	0.050	−0.461	0.020
MORO-SYMMET	INFO	−0.446	0.010	−0.629	−0.217

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Table A5 (*continued*)

Variable	Measure	r	p	95 % CI (Lower)	95 % CI (Upper)
MORO-SYMMET	SIMILAR	−0.082		−0.329	0.176
MORO-SYMMET	DIGIT SPAN	−0.082		−0.329	0.176
MORO-SYMMET	DIGIT SPAN	−0.348	0.010	−0.553	−0.103

Data availability

Raw data is available at website indicated in the ms.

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