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<th>Autism spectrum disorder and early motor abnormalities: connected or coincidental companions?</th>
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Review article

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abstract

Research in the past decade has produced a growing body of evidence showing that motor abnormalities in individuals with autism spectrum disorder (ASD) are the rule rather than the exception. The paper by Chinello and colleagues furthers our understanding of the importance of studying motor functions in ASD by testing a non-clinical population of parents-infant triads. Chinello and colleagues’ findings seem to suggest that subclinical motor impairments may exist in the typical population with inherited non-clinical ASD traits. Chinello and colleagues’ discovery also urges us to ask why motor abnormalities exist in typically developing infants when their parents present some subclinical ASD traits. We believe that there are at least two possibilities. In the first possible scenario, motor impairments and ASD traits form a single cluster of symptoms unique to a subgroup of individuals with autism. A second possible scenario is that motor atypicalities are the first warning signs of vulnerability often associated with atypical development. In conclusion, Chinello et al.’s findings inform us that subclinical atypical phenotypes such as sociocommunica- tive anomalies may be related to subclinical motor performances in the next generation. This adds to our knowledge by shedding some light on the relation of vulnerability in one domain with vulnerability in another domain.
Research in the past decade has produced a growing body of evidence showing that motor abnormalities in individuals with autism spectrum disorder (ASD) are the rule rather than the exception. Early motor functions have been reported to precede socio-communicative deficits (e.g., Leary & Hill, 1996). ASD-related motor abnormalities include an extensive variety of impairments affecting gross and fine motor functions, the inability to execute a sequence of actions (apraxia, difficulties in imitation), atypical eye movements, and deficiencies in motor learning (Dziuk et al., 2007; Mostofsky, Goldberg, Landa, & Denckla, 2000; Takarae, Minshew, Luna, Krisky, & Sweeney, 2004). The earliest mentions of motor abnormalities can be found in Kanner (1943) and Asperger (1944) studies, in which they report on ‘clumsiness in gait and motor performances’ (Asperger, 1944, 1979; Kanner & Lesser, 1958). Subsequent research described deviant motor phenomena in ASD as dyskinetic and dystonic (Damasio and Maurer, 1978), a parkinsonian (Vilensky, Damasio, & Maurer, 1981) or ataxic-cerebellar gait (Hallett et al., 1993; see also Nayate et al., 2012), an asymmetrical alignment of body at rest and in motion (Teitelbaum, Teitelbaum, Nye, Fryman, & Maurer, 1998; Teitelbaum et al., 2004), having asymmetric postures (Esposito, Venuti, Maestro, & Muratori, 2009; Esposito, Venuti, Apicella, & Muratori, 2011; Esposito, Yoshida, Venuti, & Kuroda, 2012), head lag (Flanagan, Landa, Bhat, & Bauman, 2012), and abnormal spontaneous general movements during the prodromal period of motor development (Einspieler et al., 2014; Zappella et al., 2015). However, the evidence and the interpretation of data so far is mixed. No study to date has found any set of motor symptomatology as a potential identifier for ASD (Yirmiya & Charman, 2010). Nonetheless, more and more evidence points towards the utility and impact of motor assessments for diagnostics and/or outcome measurements in ASDs. Motor dysfunctions may serve as early indicators of an increased risk to develop ASD because they commonly are persistent and may be part of a broad ASD phenotype. Within different age groups, ASD and motor anomalies are somehow linked. Hence, motor dysfunctions could potentially play a central role in elucidating pathophysiology.

The paper by Chinello and colleagues furthers our understanding of the importance of studying motor functions in ASD by testing a non-clinical population of parents-infant triads. They evaluated the association between 12- to 17- month-olds’ persistence of primitive reflexes (grasping, rooting and sucking), their motor repertoire in terms of interactions with objects and people, and their parents’ autistic traits as measured by the Autism-Spectrum Quotient questionnaire. Chinello et al. found that primitive reflexes decreased with infants’ increasing age, however, regardless of infants’ age, the persistence of their primitive reflexes was related to their reduced motor repertoire. Parents who had higher levels of subclinical autistic traits tended to have infants who displayed more persistent primitive reflexes than parents with lower levels of subclinical autistic traits. Chinello et al. postulate that within the typically developing population, parents’ subclinical autistic traits can be predictive of their infants’ persistent reflexes. They go on to suggest that this relates to the infants’ reduced motor repertoire. In particular, they found that low social and communication skills for male parents, and low social skills for female parents give rise to the endophenotype of subclinical motor impairment in their child. This is a striking finding because, although past studies have linked gross motor functions and social skills (see for example: Pusponegoro et al., 2016), such a link has not been explored within the typically developing population. Chinello and colleagues’ findings seem to suggest that subclinical motor impairments may exist in the typical population with inherited non-clinical ASD traits. This hypothesis remains to be confirmed by longitudinal studies following up typically developing infants who display motor impairments at a young age to find out if they develop some subclinical ASD traits in their later years. It also remains to be determined whether these results can be replicated with a larger sample, and across a wider age range of infants. In addition, it
would have been ideal if Chinello et al. investigated the parents’ motor skills to see how it interacted with their autistic traits. However, this new finding is consistent with previous work on the heritability of motor difficulties as part of the broader autism phenotype. For example, delayed motor development is common among siblings of individuals with ASD and it is predictive of later communication deficits (e.g., Bhat, Galloway, & Landa, 2012). However, it should be noted that there are studies which have failed to find motor delays in typically developing siblings of children with ASD (Hilton, Zhang, White, Klohr, & Constantino, 2011).

Chinello and colleagues’ discovery also urges us to ask why motor abnormalities exist in typically developing infants when their parents present some sub-clinical ASD traits. We believe that there are at least two possibilities. In the first possible scenario, motor impairments and ASD traits form a single cluster of symptoms unique to a subgroup of individuals with autism (as suggested by previous studies such as Teitelbaum et al., 1998, 2004). This view would be in line with the idea of a common neural substrate relating ASD and motor development (Fatemi et al., 2012; Sears et al., 1993). A second possible scenario is that motor atypicalities are the first warning signs of vulnerability often associated with atypical development (Hartman et al., 2012; Piek et al., 2004). This vulnerability is evinced as motor deficits because the motor domain is ubiquitously implicated across development. This also suggests that if there is something wrong with neural wiring in the early stages, it may lead to peculiarities in the motor domain because it is among the first parameters to develop in early infancy (Piek, Dawson, Smith, & Gasson, 2008). The assumption of possibility two is that atypical motor development may be observable in a variety of neurodevelopmental and psychiatric disorders, including Rett syndrome, Tourette syndrome, obsessive-compulsive disorder, language impairment, attention deficit hyperactivity disorder, depression, disruptive behavior disorder, etc. (e.g., Einspieler et al., 2014; McPhillips, Finlay, Bejerot, & Hanley, 2014; Van Damme, Simons, Sabbe, & van West, 2015).

In conclusion, Chinello et al.’s findings do not provide a solution addressing which of our two proposed options is more accurate. However, their findings inform us that subclinical atypical phenotypes such as socio-communicative anomalies may be related to subclinical motor performances in the next generation. This adds to our knowledge by shedding some light on the relation of vulnerability in one domain with vulnerability in another domain. There is however more to learn in order to understand the stability of these findings and the causal relations of this potential cross domain effect.

References


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