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A review of physical growth in children and adolescents with Autism Spectrum Disorder

Cherie Green, Cheryl Dissanayake *, Danuta Loesch

Olga Tennison Autism Research Centre (OTARC), School of Psychological Science, Faculty of Science, Technology and Engineering, La Trobe University, Bundoora, Vic. 3086, Australia

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ABSTRACT

Head circumference growth in individuals with an Autism Spectrum Disorder (ASD) has been well characterized in the first two to three years of life and reflects a period of acceleration followed by a period of deceleration when compared with their typically developing (TD) peers. While this altered growth trajectory has been consistently found for head circumference, it is less clear if an abnormal growth trajectory also exists across measures of height and weight. Moreover, most studies have focused on infancy and early childhood, and no longitudinal data have been collected in older children with ASD. This review focuses on the physical growth trajectory of individuals with ASD, and proposes that a general growth dysregulation is present in ASD, and that an endophenotype within ASD may exist that is characteristic of extreme overgrowth. Two possible explanations for a general growth dysregulation are suggested: (1) a connective tissue disorder, which is frequently associated with increased height and disproportionate body ratios; and (2) a dysregulation of the hypothalamic–pituitary–adrenal (HPA) axis, which regulates growth hormones. The existence of a general growth dysregulation, and possible endophenotype, may serve as a potential biological marker in ASD.

* Corresponding author. Olga Tennison Autism Research Centre, School of Psychological Science, La Trobe University, Bundoora, Vic. 3086, Australia. Fax: +61 3 9479 1956.
E-mail address: c.dissanayake@latrobe.edu.au (C. Dissanayake).

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Background

Autism Spectrum Disorder (ASD) is first evident in infancy and early childhood, and individuals with ASD have deficits in social communication and a repertoire of restricted interests and repetitive behaviours (American Psychiatric Association, 2013). Until recently, ASD comprised Autistic Disorder (AD), Asperger's Disorder (AspD), and Pervasive Developmental Disorder – Not Otherwise Specified (PDD-NOS) (American Psychiatric Association, 2000). The specific diagnosis given depended on the severity and quantity of symptoms in each of the affected domains. Individuals diagnosed with AD had pronounced deficits across the domains, while individuals diagnosed with AspD had fewer deficits across the domains and did not have delays in language development and no cognitive impairments. PDD-NOS encompassed what was sometimes referred to as "atypical autism" – where the individual presented with deficits characteristic of AD, but had fewer symptoms than required for a clinical diagnosis of AD. Individuals diagnosed with AD but who have normal cognitive functioning (IQ ≥ 70) are referred to as having high-functioning autism (HFA). With recent changes to the Diagnostic and Statistical Manual of Mental Disorders (DSM-5), these diagnostic categories are no longer used for newly diagnosed clients, and are now encompassed within the diagnostic label of ASD (American Psychiatric Association, 2013).

Autism Spectrum Disorder occurs in three to four times as many males than females (Fombonne, 2003) and affects approximately 1% of the population (Centers for Disease Control and Prevention, 2012). Recent studies have provided overwhelming evidence for genetic involvement in ASD (Bishop, Maybery, Wong, Maley, & Hallmayer, 2006; Constantino et al., 2006; Constantino, Zhang, Frazier, Abbacchi, & Law, 2010b; Rosenberg et al., 2009; Taniai, Nishiyama, Miyachi, Imaeda, & Sumi, 2008; Veenstra-VanderWeele, Christian, & Cook, 2004). The risk of having a child with ASD increases with a family history of ASD, with heritability estimates ranging between 37% and 70% (Constantino et al., 2013; Hallmayer et al., 2011). First degree relatives of children with ASD often have traits of ASD themselves, known as the Broader Autism Phenotype (BAP); these features are more prevalent in the parents of children who are more severely affected, suggesting intergenerational transmission (Sasson, Lam, Parlier, Daniels, & Piven, 2013). Rare de novo copy variants (CNVs) account for a smaller portion of ASD cases. These CNVs are more common in simplex families (families with only one member affected by ASD) compared with multiplex families (families with multiple members affected) (Iossifov et al., 2012; Luo et al., 2012; Sebat et al., 2007). However, despite advances in our understanding of ASD, the biological bases of these disorders remain unknown. One area which may provide some insight into the biological bases of ASD is physical growth.

Growth in ASD

Few studies have focused on physical growth in individuals with ASD, thus little is known about their growth in stature and their body proportions, although there have been many studies on size and growth in head circumference (HC), and corresponding studies on brain size and growth. The current review aimed to elucidate the growth trajectory of not only head growth, but growth in height and weight, from infancy throughout adolescence in ASD, in order to determine if physical overgrowth in ASD is limited to head and brain growth, or if an overall dysregulation of growth is present. The review also aimed to determine the extent to which subgroups exist within ASD based on growth parameters and characteristics of ASD. Peer-reviewed studies investigating physical growth of individuals diagnosed with an ASD (AD, AspD, and PDD-NOS) were included in the current review. Studies were not excluded based on specific criteria, but rather were included in the review, with limitations discussed, and were excluded from the list of studies used to synthesize the overall results. Table 1 presents a summary of the most methodologically sound studies (11 out of the 34 studies identified) used to synthesize the results. Relevant journal articles were identified using La Trobe University's online library database, which includes, but is not limited to, ProQuest Central, PsychINFO, and Medline. Reference lists from articles, as well as key journals (e.g., Journal of Autism and Developmental Disorders) were also used to identify relevant articles. Key search terms included, but were not restricted to: Autism, Autistic Disorder, Autism Spectrum Disorder, growth, head circumference, height, and weight.