FEATURES OF THE DEVELOPMENT OF NEURAL CONNECTIONS (WHITE MATTER) IN CHILDREN WITH AUTISM

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Abstract. *White matter development is one of the most important neurobiological dimensions in autism spectrum disorder because it reflects how distant brain regions become structurally connected during infancy and childhood. In children with autism, diffusion MRI studies do not support a single fixed abnormality. Instead, they point to an atypical developmental trajectory. The earliest findings suggest that some infants who later develop autism show relatively increased fractional anisotropy at 6 months, followed by a slower pace of white matter maturation during the second year of life. In preschool and school-age samples, this early advantage is often replaced by reduced or region-specific compromise in major tracts related to interhemispheric transfer, language, social communication, and executive regulation.*

Key words: *autism spectrum disorder, white matter, neural connections, diffusion tensor imaging, fractional anisotropy, radial diffusivity.*

Introduction

Autism spectrum disorder is a neurodevelopmental condition in which difficulties of social communication, sensory processing, and behavioral flexibility emerge against the background of atypical brain maturation. One of the most informative systems for studying this process is white matter, because white matter tracts support communication between cortical and subcortical regions. In neuroimaging studies, white matter development is usually examined with diffusion tensor imaging, which provides indirect microstructural measures such as fractional anisotropy, mean diffusivity, radial diffusivity, and axial diffusivity. These measures do not identify a single cellular mechanism by themselves, but they are useful for tracking maturation-related changes in fiber organization, axonal properties, and myelination-related processes. In autism research, the major question is not whether white matter differs at one time point, but how its developmental trajectory diverges from the expected course from infancy into childhood [1], [3], [4].

Materials and methods

One of the most influential findings comes from the prospective infant work of Wolff and colleagues. In a high-risk sibling cohort scanned at 6, 12, and 24 months, infants who were later diagnosed with autism showed significantly different fractional anisotropy trajectories in 12 of 15 fiber tracts. For most tracts, the autism group had higher FA at 6 months, but then showed slower change over time, so that by 24 months lower FA values became evident relative to infants who did not develop autism [1]. This finding is important for two reasons. First, it suggests that white matter divergence begins before full behavioral syndrome expression. Second, it indicates that autism-related white matter development cannot be described as a simple delay. The pattern is more complex: in early infancy some pathways may appear relatively advanced or atypically compact, but the subsequent rate of maturation is reduced. That sequence has shaped much of the later literature on autism and white matter development [1], [3], [4].

Results and discussion

Studies in early childhood extended this picture and showed that abnormalities are already visible in major tracts by the preschool years. In young children with autism, Weinstein and colleagues identified white matter abnormalities in the genu and body of the corpus callosum, the left superior longitudinal fasciculus, and the bilateral cingulum. In the abnormal clusters, increased FA was driven by reduced radial diffusivity, while axial diffusivity and mean diffusivity did not differ significantly. The authors suggested that such atypical integrity may affect connectivity among brain regions and contribute to behavioral impairments [2]. Although the direction of change differed from many later childhood findings, this is precisely what makes the study valuable. It supports the idea that early autism is associated with a distinct microstructural profile that may not persist unchanged over time. White matter in autism seems to develop through a shifted trajectory rather than through a uniform deficit that looks the same at all ages [2], [3].

A broader tract-level picture was provided by Ouyang and colleagues, who examined boys aged 2 to 7 years across 48 major white matter tracts. Their results showed that children with autism had higher FA and lower radial diffusivity in most major tracts before about 4 years of age, but then displayed reduced rates of FA and RD change afterward. The authors concluded that widespread white matter in autism may reach a relatively advanced microstructural level early and then mature more slowly, producing the later pattern of lower FA and higher RD seen in older cohorts [3]. This helps reconcile apparently conflicting findings in the literature. Rather than assuming that one study is “correct” and another is “incorrect,” the more plausible explanation is that the biological picture shifts with age. In autism, the timing of measurement matters enormously. A tract examined at 3 years may look different from the same tract examined at 7 or 12 years, not because the disorder changes identity, but because its developmental curve is atypical [3].

This age-sensitive interpretation is also supported by school-age data. Mak-Fan and colleagues studied children with autism between 6 and 14 years and found age-by-group interactions for frontal, long-distance, interhemispheric, and posterior tracts in longitudinal, radial, and mean diffusivity, but not in FA. Diffusivity measures decreased

with age in typically developing children, whereas the autism group showed little or no change. The authors concluded that white matter in autism follows an abnormal developmental trajectory that may influence neural connectivity and atypical cognitive development [4]. This result is especially useful because it shows that by middle childhood the main issue is not necessarily an isolated tract lesion, but reduced developmental change itself. Typical maturation involves continuing refinement of long-range and interhemispheric pathways. When that refinement becomes weaker or flatter over time, the child may enter later developmental stages with less efficient structural integration across brain systems [4].

Conclusion

The development of neural connections in children with autism is characterized less by one fixed abnormality than by an atypical developmental course of white matter maturation. The most consistent pattern across infant and childhood diffusion studies is early divergence followed by slower or altered maturation of major long-range tracts. In infancy and early preschool years, some pathways may show relatively higher FA or other signs of altered early organization, but across later childhood this often gives way to weaker developmental change and, in some tracts, lower apparent integrity compared with typical development.

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