

Neurodevelopmental disorders and the cerebellum

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ABSTRACT

The cerebellum has been dubbed "a neuronal learning machine", but its importance in development and developmental disorders has only lately been recognised. The cerebellum is assumed to facilitate implicit/procedural learning via the construction and error-driven adjustment of internal models of behaviour during development, which is a period of fast skill acquisition. The cerebellum develops in a similar way to the cerebral cortex, with parts supporting more basic sensorimotor skills (such as the cerebellar anterior lobe

(lobules I-V)) maturing earlier than those supporting higher-level cognitive activities (e.g. posterolateral lobule VII). The structural foundation via which the cerebellum can affect activity patterns in distant regions is the closed-loop circuits between the cerebellum and the cerebral cortex. Based on these relationships, it's been hypothesized that cerebellar malfunction or disruption early in development could have a significant impact on the structure and function of the cortical regions into which it projects.

Key Words: *Neurodevelopmental disorders; Cerebellar dysfunction; Autism Spectrum Disorder (ASD); Attention Deficit-Hyperactivity Disorder (ADHD)*

EDITORIAL

The evidence suggesting cerebellar dysfunction plays a significant role in neurodevelopmental disorders is reviewed here, as well as the cerebellum's possible contribution to typical and atypical development. We concentrate on three developmental illnesses in which cerebellar abnormalities have been well-documented (Autism Spectrum Disorder (ASD), attention deficit-hyperactivity disorder (ADHD), and developmental dyslexia [1], as well as the consequences of cerebellar damage in children.

ASD is defined by communication and social interaction difficulties, as well as repetitive behaviours and limited interests. The cerebellum is one of the most consistently reported abnormalities in ASD, with findings in genetic, animal model, post-mortem, and neuroimaging investigations (for reviews, see [2]). Reduced cerebellar cortical volume is a key classifier for ASD brains, and hypoplasia of the posterior vermis was one of the first identified brain abnormalities in ASD. Grey matter reductions have been reported in the right Crus I, left lobule VIII, and medial IX across studies, which could have functional implications for specific cerebro-cerebellar circuits; for example, reduced functional connectivity between the right Crus I and left-hemisphere language regions has been observed in language-impaired children with ASD [3]. Disruption in both the input and output channels of cerebro-cerebellar circuits is indicated by structural abnormalities in the cerebellar peduncles. These

anatomical and functional abnormalities in the cerebellum are linked to basic autism symptoms.

ADHD, which is characterised by inattention, hyperactivity, and impulsivity, has also been linked to cerebellar abnormalities. Cerebellar results, like those in ASD, were among the first reported variations in ADHD, with reduced cerebellar volumes reported in the first quantitative investigation of brain morphometry. In children with ADHD, methylphenidate, one of the most commonly used pharmaceutical therapies for ADHD, causes changes in cerebellar activity [4]. In children treated with methylphenidate, posterior vermal reductions were not observed, but cerebellar structural changes were the most significant observation in medication-naive adults with ADHD. A recent meta-analysis showed consistent grey matter decreases in lobule IX in ADHD patients on both sides. Differences in anatomical (e.g. middle cerebellar peduncles) cerebellar connections have also been identified in ADHD.

Finally, developmental dyslexia is characterised as a reading learning issue that is not caused by a general intellectual deficiency or a lack of educational opportunities [5]. Dyslexia is linked to a core phonological processing deficiency as well as delayed, laborious reading. Dyslexia has been linked to poor performance on a variety of "cerebellar" motor tasks, including balance, quick pointing, peg-moving, and eye movement control. These observations, together with the lack of fluent, automatic reading in dyslexia, led to the hypothesis that dyslexia is caused by cerebellar dysfunction [6].

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Because dyslexia is a distinct learning disability, cerebellar dysfunction resulting in a procedural learning deficit could be a plausible explanation for dyslexic readers' poor reading skills acquisition. Cerebellar abnormalities have been reported in both structural and functional neuroimaging investigations in dyslexic children and adults, with diminished grey matter in right lobule VI being the most important biomarker for adult dyslexic brain classification. In a meta-analysis of voxel-based morphometry studies in dyslexia, reduced grey matter in the left VI was also reported. Recent neuroimaging research reveals that the fluency component of dyslexia, which is characterised by quick naming, is linked to aberrant right lobule VI activations [7]. Finally, right anterior cerebellar GM increased in response to successful dyslexia remediation, supporting the hypothesis that the cerebellum aids skill development.

Different cerebellar subregions are impacted in each illness, suggesting that disruption of distinct cerebro-cerebellar circuits may be relevant to the development of ASD, ADHD, or dyslexic behavioural characteristics. Cerebellar injury can also cause symptoms and, in some cases, diagnoses of these illnesses.

Early cerebellar injury is linked to a variety of motor, cognitive, and affective effects in a location-dependent manner, according to data from clinical populations. Attention deficits in children following cerebellar damage often fall under the overarching executive function impairments associated with the Cerebellar Cognitive Affective Syndrome. Cerebellar damage has been directly linked to ASD diagnoses, and attention deficits in children following cerebellar damage often fall under the overarching executive function impairments associated with the Cerebellar Cognitive Affective Syndrome. While verbal injury and malformations have been linked to ASD symptoms, right cerebellar damage has been linked to poorer language outcomes and left cerebellar damage to poorer visuospatial performance (e.g. due to the cerebellum's contra-lateral connections to the cerebral cortex. Importantly, unlike injury to the cerebral cortex, the consequences of cerebellar damage in childhood can be far worse than the consequences of cerebellar damage in maturity [8,9].

Multiple regions of the brain reveal neurological abnormalities in ASD, ADHD, and dyslexia, thus it's crucial to investigate the cerebellar contribution to the aetiology of these diseases. The cerebellum's cytoarchitecture is fairly uniform; therefore it's assumed that whatever information it gets is processed in the same way. The realm of procedural learning is one overarching potential mechanism of the cerebellum contribution to developmental disorders (for example, explicit/declarative processes are assumed to be intact in dyslexia and used for compensating).

The cerebellum's role in procedural learning could entail honing and improving cerebro-cerebellar circuits that support a wide range of behaviours, from social skill acquisition to attention regulation to the formation of the left-hemispheric "reading" network as children learn to read. This optimization of performance is enabled through the creation of internal models inside the cerebellum, probably through modulation of activity in regions targeted by cerebellar output. When this modulation is lost, behaviour becomes erroneous and poorly calibrated, disrupting procedural skill learning [10].

CONCLUSION

Given that the cerebellum is thought to be most active during the early stages of learning and least active during the retention of

learned behaviours, it's possible that the cerebellum is more important early in life when cerebro-cortical networks are forming and less important later in life when behaviours have been properly set up in distributed cortical networks. As a result of the disruption of circuits supporting language and social cognition, attention, and literacy acquisition, cerebellar dysfunction may have an impact on behaviours linked with autism, ADHD, and dyslexia, depending on the exact subregions where developmental anomalies originate.

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