

Interval timing deficits and abnormal cognitive development

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Time perception deficits represent an aspect of cognitive malfunctioning shared by developmental disorders, which otherwise seem distinct with respect to their individual primary symptom clusters, such as autism spectrum disorders (ASD) and attention-deficit/hyperactivity disorder (ADHD). Multiple impairments of social interaction, communication, and restricted interests constitute the diagnostic criteria for ASD, whereas behavioral symptoms of ADHD comprise inattention, hyperactivity, and impulsivity (DSM-IV-TR; American Psychiatric Association, 2000). Both ASD and ADHD are additionally associated with non-diagnostic secondary symptoms in perception and cognition. A secondary symptom observed in both ASD and ADHD is abnormal interval timing, i.e., processing of stimulus duration (for a review see Falter and Noreika, accepted). For instance, it has been reported that reproduction of auditory and visual intervals of 1-5.5 s was impaired in individuals with ASD (Szelag et al., 2004). Similarly, Barkley et al. (2001) found that individuals with ADHD were impaired in reproducing intervals of 2-60 s.

It is difficult to assess whether secondary symptoms in general and interval timing abnormalities in particular play a causal role in developmental disorders. There are several possible relationships. First, a direct causal relationship would characterize a case in which impairment of an interval timing system could directly disrupt (otherwise possibly unimpaired) cognitive functions relying on accurate timing information, resulting in the known primary symptoms. Although such a direct causal relationship might be found in adult neuropsychology, it is not applicable to the study of developmental disorders, which are characterized by atypical neurogenetic pathways of cognitive development. Rather, as a second option, an ontogenetic causal relationship is conceivable in which an impaired interval timing system could affect the development of all processes downstream, which rely on accurate timing information. Finally, a third

option would be that abnormal interval timing could be an epiphenomenon of developmental disorders without bearing a causal relationship to other symptoms.

The idea of an ontogenetic causal relationship raises the question of why the phenomenological outcomes of the disorders differ so strongly. The apparent lack of specificity of interval timing deficits to ASD and ADHD raises the question of their explanatory relevance for the ontogenesis of a particular developmental disorder. A further challenge for the causality assumption is the lack of universality of interval timing abnormalities. A few studies report null findings (Wallace and Happe, 2008; Jones et al., 2009), and in studies showing group differences not all individuals with ASD or ADHD perform atypically. Indeed, the range of reproduced time intervals can be quite variable and the variability is often increased in ASD and ADHD compared to typically developing controls (Toplak et al., 2003; Martin et al., 2010). At the current stage of knowledge, therefore, it seems premature to suggest that interval timing abnormalities can be the sole ontogenetic cause of ASD or

Nevertheless, before we discard any causal account in favor of the alternative view of interval timing as a mere epiphenomenon of atypical cognitive development, we suggest that a more associative view of the role of interval timing in cognitive development be adopted. Indeed, there is strong evidence for the association between temporal processing and other typical cognitive functions such as social cognition (Trevarthen and Daniel, 2005; Striano et al., 2006), language processing (Tallal et al., 1993), and understanding of causality (Freeman, 2008). Therefore, it is difficult to conceive how an impairment of interval timing would have no relevance for developmental disorders, which show deficits in cognitive functions relying on accurate timing. Furthermore, the incidence of interval timing abnormalities is increased in developmental disorders, as shown by group differences in perfor-

mance even for relatively small sample sizes (Szelag et al., 2004). Thus, in spite of the lack of universality of interval timing deficits, the increased incidence rate needs to be explained. Moreover, it has been suggested that ADHD and ASD share some susceptibility genes (Castellanos and Tannock, 2002), which makes it likely that some dysfunctions are shared between them or their subtypes. We propose that the focus of research needs to be on the association of interval timing abnormalities and other functional deficits. For instance, although a Theory of Mind deficit can hamper the understanding of social situations in its own right, an additional interval timing deficit could result in a lack of precise perception of temporal cues of eye gaze, and thereby increase misinterpretations of social situations. It has been proposed that different symptoms can be independent dimensions of impairment, which nevertheless interact with and modulate one another, leading to the characteristic phenomenology of an individual with a developmental disorder (Happe et al., 2006). In this line of thought, interval timing abnormalities might interact with primary dysfunctions.

In fact, several ADHD studies confirmed significant associations between duration perception and other cognitive functions. Toplak and Tannock (2005) reported significant correlations between time discrimination thresholds and working memory measures in participants with ADHD, but not in healthy individuals. Rubia and colleagues argued persuasively that the primary ADHD symptom of impulsiveness is based on poor inhibition and attention functions, as well as on poor interval timing (Rubia, 2002; Rubia et al., 2009). Even though interaction between interval timing and other cognitive functions is much less investigated in ASD than in ADHD, preliminary findings show significant correlation between timing measures in a temporal bisection task and primary ASD symptoms in the language and communication domain (Allman et al., 2011). Taken together, these reports demonstrate complex associations between abnormally developing cognitive functions, and suggest that interval timing might play an important yet under-investigated role in developmental disorders by interacting with and modulating primary symptoms.

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