



Dysfunction of sensory oscillations in Autism Spectrum Disorder



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ABSTRACT

Autism Spectrum Disorder (ASD) is a highly prevalent developmental disability characterized by deficits in social communication and interaction, restricted interests, and repetitive behaviors. Recently, anomalous sensory and perceptual function has gained an increased level of recognition as an important feature of ASD. A specific impairment in the ability to integrate information across brain networks has been proposed to contribute to these disruptions. A crucial mechanism for these integrative processes is the rhythmic synchronization of neuronal excitability across neural populations; collectively known as oscillations. In ASD there is believed to be a deficit in the ability to efficiently couple functional neural networks using these oscillations. This review discusses evidence for disruptions in oscillatory synchronization in ASD, and how disturbance of this neural mechanism contributes to alterations in sensory and perceptual function. The review also frames oscillatory data from the perspective of prevailing neurobiologically-inspired theories of ASD.

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1. Introduction

Autism Spectrum Disorder (ASD) is a developmental disability characterized by persistent deficits in social communication and interaction, restricted interests, and repetitive behaviors (American Psychiatric Association, 2013). An estimated 1 in 68 children born in the United States will receive a diagnosis of ASD, and the disorder carries enormous social and economic costs (Buescher et al., 2014; Developmental Disabilities Monitoring Network Surveillance Year Principal et al., 2014; Karst and Van Hecke, 2012). This high prevalence and socioeconomic cost have motivated numerous investigations to better understand the brain bases of ASD. Studies utilizing functional magnetic resonance imaging (fMRI) have consistently indicated that patterns of structural (Shukla et al., 2010) and functional (Dinstein et al., 2011) connectivity are significantly altered in individuals with ASD. Postmortem anatomical inquiries have likewise indicated that the microstructure of cortical circuitry is fundamentally altered in ASD (Casanova et al., 2006; McKavanagh et al., 2015). Investigations examining connectivity on more rapid time scales utilizing electroencephalography (EEG) (Coben et al., 2014) and magnetoencephalography (MEG) (Ye et al., 2014) have similarly indicated that connectivity alterations are a characteristic feature of ASD. These connectivity alterations have been proposed as both a leading biomarker and the origin of the behavioral dysfunction characteristic of the disorder (Geschwind and Levitt, 2007). Network-based analyses have revealed that the nature of connectivity differences among individuals with ASD is highly individualized (Hahamy et al., 2015). However, how these changes in network structure impact neural processing and emerge as the collection of phenotypes that characterize ASD is poorly understood, and consequently has become an area of important investigation. Studies using EEG and MEG have uncovered differences in rhythmically modulated networks known as oscillators. This oscillatory dysfunction in ASD may form the bridge between dysfunction at the cellular and local levels, changes in large-scale network organization, and the sensory and perceptual processing differences that represent a core feature of the disorder.

2. Sensory and perceptual function in ASD

Alterations in sensory and perceptual processes have long been recognized to be present in ASD (Marco et al., 2011). Recent revisions to diagnostic criteria have now acknowledged that these sensory and perceptual dysfunctions constitute a core feature of ASD (American Psychiatric Association, 2013). Intriguingly, investigations focused on sensory function in ASD have revealed that, even within a single sensory modality such as vision, both strengths and weaknesses can be present. For example, individuals with ASD consistently outperform their typically developing (TD) peers in terms of accuracy and response speed in visual search tasks (O’Riordan et al., 2001; Shah and Frith, 1983), and similarly excel at visuospatial tasks (Caron et al., 2006). In other visual tasks, such as discrimination of visual motion (David et al., 2010; Milne et al., 2002) or gestalt perception (Grinter et al., 2010), individuals with ASD show a pattern of significant deficits. This dichotomy between impaired and enhanced processing is also found in other sensory modalities. For example, in auditory tasks, individuals with ASD excel at detection of pitch change (Bonnell et al., 2003; Foxton et al., 2003), but are impaired in the ability to utilize gaps in noise to assist with speech comprehension (Groen et al., 2009). Tactile discrimination thresholds may also be superior in ASD (Blakemore et al., 2006), although this is more debated (Puts et al., 2014). Collectively, this complex pattern of strengths and weaknesses define sensory and perceptual function in ASD as an area of *difference* rather than one of *deficit*.

An account of perceptual differences that has gained increasing support is that individuals with ASD have deficits in *perceptual integration*. In other words, they may possess normal or even superior processing of stimulus characteristics, but fail to integrate sensory information into a coherent perceptual whole (Dakin and Frith, 2005). Tasks such as discriminating visual motion within a cloud of moving dots require integration of localized evidence and are frequently impaired in ASD, despite their seemingly simplistic sensory composition. In contrast, visual search of complex stimuli does not require combining disparate pieces of sensory information. Indeed, reduced integration may result in enhanced performance on certain tasks (Motttron et al., 2006). This hypothesis receives support from experimental manipulations that focus upon the perceptual complexity of visual stimuli. In these tasks, the performance of individuals with ASD continuously degrades as the need for feature integration increases (Bertone et al., 2005). Impaired processing is also notable when the available evidence spans multiple sensory systems and thus requires integration for the formation of correct multisensory perceptual representations. In these multisensory tasks individuals with ASD exhibit perceptual deficits even when working with relatively simplistic sensory stimuli (Kwakye et al., 2011). The level of impairment in ASD further rises with the increased need for perceptual integration associated with processing complex naturalistic stimuli such as speech (Stevenson et al., 2014). Investigators have increasingly turned to non-invasive neuroimaging and neurophysiological techniques to investigate the neural bases of these differences. These investigations have uncovered that harmonic neural synchronization, collectively referred to as oscillations, is altered in ASD (Uhlhaas and Singer, 2012).

3. Oscillatory contributions to sensory encoding

The rhythmic nature of neural activity has been recognized since the earliest attempts at non-invasive measurement (Berger, 1929). These rhythmic fluctuations are referred to as oscillations, and have been characterized over a large range of frequencies (here denoted as delta: δ , 1–4 Hz, theta: θ , 4–8 Hz, alpha: α , 8–14 Hz, beta: β , 15–30 Hz, and gamma: γ , >30 Hz, although the exact ranges vary in the literature). The role of these oscillations in neural computation is of great interest and has motivated studies designed to establish their neurophysiological origin and functional significance. At the cellular level, these studies have indicated that oscillations index fluctuations of the local field potential (LFP; a measure of voltage change in proximity to a recording electrode), and are primarily a result of synchronized postsynaptic activity (Buzsaki et al., 2012) (Fig. 1A). These studies have also found that neurons have biophysical properties that facilitate synchronization, such as intrinsic resonance (Hutcheon and Yarom, 2000; Llinas, 1988) and a mixture of predictable harmonic and responsive relaxation properties (Glass, 2001). At the circuit level, this harmonic synchronization appears to be an optimal mechanism of network organization, allowing for modulation of responses and synchronization of outputs at low energetic cost (Buzsaki and Draguhn, 2004). The optimal nature of oscillatory synchronization is also supported by modelling studies in the field of network science, which indicate that forming small world networks (Bullmore and Sporns, 2009) through harmonization and network hubs is more efficient (Strogatz, 2001) and flexible (Bullmore and Sporns, 2012) than direct structural connections. Neurons participating in these synchronized assemblies experience temporally aligned fluctuations in membrane potential that correspond with the observed oscillatory phase (Wang and Buzsaki, 1996). This synchronized phasic modulation of neuronal excitability and spike timing represents an effective method of selectively shaping the nature of network interactions and multiplexing signals (Akam and Kullmann, 2014).

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