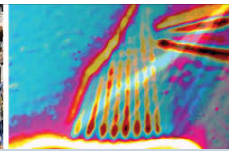
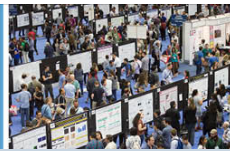


2018

Atypical excitatory-inhibitory balance in feedforward and feedback circuits in autism

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Session 456 - Autism: Cellular Mechanisms

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456.01 / A18 - Atypical excitatory-inhibitory balance in feedforward and feedback circuits in autism

November 6, 2018, 8:00 AM - 12:00 PM

SDCC Halls B-H

Presenter at Poster

Tue, Nov. 6, 2018, 8:00 AM - 9:00 AM

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Abstract

Interactions between excitatory and inhibitory elements in neural circuits are crucially altered in autism and contribute to its central symptoms. Functionally and neurochemically distinct classes of inhibitory neurons, which express the calcium-binding proteins calbindin (CB), calretinin (CR), and parvalbumin (PV), are distributed differentially between different cortical areas and cortical layers. These neurons modify the influence of the excitatory pathways, carried by myelinated axons, that project to those layers. Excitatory connections that terminate in the middle or deep cortical layers behave similarly to the feedforward pathways that are defined in sensory areas, and provide driving input to the cortex. Pathways that terminate in superficial layers behave as feedback pathways and modulate the activity of the cortex. In order to study excitatory-inhibitory balance in the human cortex we studied the distribution of the three classes of inhibitory neurons and the density of myelinated axons in post-mortem tissue from medial, cingulate, and lateral prefrontal cortices of typically developing individuals and individuals with autism. We separately studied superficial and middle/deep cortical layers in order to distinguish changes that influence feedback and feedforward pathways. Adults with autism had a significant reduction in the density of CR-expressing inhibitory neurons in both superficial and middle/deep cortical layers in lateral prefrontal cortices. There was a similar trend in medial prefrontal cortices in adults with autism. CR-expressing inhibitory neurons in superficial cortical layers serve a disinhibitory role while those in deep layers may provide modulatory inhibition. We found no significant change in the density of PV-expressing or CB-expressing interneurons in adults with autism in these regions. In individuals with autism there was also a steeper rate of increase in the density of small myelinated axons, which are representative of short-range pathways, across layers during development. These parallel structural changes likely produce opposite functional effects: enhanced short-range feedback pathways terminate in an environment with increased inhibition, while enhanced short-range feedforward pathways terminate in an environment with reduced inhibition. Together, these changes in laminar structure may significantly affect information processing in the prefrontal cortex in autism.

Abstract Citation