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Sommario/riassunto

The Centers for Disease Control and Prevention estimate that 1 in 68 children in the United states is afflicted with autism spectrum disorders (ASD), yet at this time, there is no cure for the disease. Autism is characterized by delays in the development of many basic skills, most notably the ability to socialize and adapt to novelty. The condition is typically identified in children around 3 years of age, however the high heritability of autism suggests that the disease process begins at conception. The identification of over 500 ASD risk genes, has enabled the molecular genetic dissection of the pathogenesis of the disease in model organisms such as mice. Despite the genetic heterogeneity of ASD etiology, converging evidence suggests that these disparate genetic lesions may result in the disruption of a limited number of key biochemical pathways or circuits. Classification of patients into groups by pathogenic rather than etiological categories, will likely aid future therapeutic development and clinical trials. In this set of papers, we explore the existing evidence supporting this view. Specifically, we focus on biochemical cascades such as mTOR and ERK signaling, the mRNA network bound by FMRP and UBE3A, dorsal and ventral striatal circuits, cerebellar circuits, hypothalamic projections, as well as prefrontal and anterior cingulate cortical circuits. Special attention will be given to studies that demonstrate the necessity and/or sufficiency of genetic disruptions (e.g. by molecular deletion and/or replacement) in these pathways and circuits for producing characteristic behavioral

features of autism. Necessarily these papers will be heavily weighted towards basic mechanisms elucidated in animal models, but may also include investigations in patients.