Rett syndrome is a delayed-onset childhood disorder, typically found in girls, that causes a broad range of severe neurological disabilities, including loss of speech and socialization skills and the development of tremors, ataxia, seizures, autonomic dysfunction, and stereotypic hand-wringing movements. We discovered that mutations in the gene MECP2 cause Rett syndrome, and before long it became clear that mutations in MECP2 can also cause other neuropsychiatric phenotypes—ranging from autism to bipolar disorders. Using genetically-engineered mice, we learned that the brain is acutely sensitive to MECP2 levels; both decreases and increases in the amount of MECP2 protein can lead to neurological problems that are also observed in humans. Normalizing MECP2 levels can reverse disease-like features in a mouse model of the human MECP2 duplication syndrome, a disorder that is usually found in boys and results from excess MECP2. In addition, we have been gradually pinpointing the neurons and circuit abnormalities that mediate various symptoms. Building on this understanding of the relationship between neural circuits and the features of Rett syndrome, we collaborated with the laboratory of Dr. Jianrong Tang and showed that deep brain stimulation of a specific neural network improved learning and memory in a Rett syndrome mouse model.

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