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Brennan Beeler
Washington University in St. Louis

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Brennan Beeler

Mentor: Michael Wong

Tuberous sclerosis complex (TSC) is a genetic condition resulting from mutation in the *TSC1* or *TSC2* genes. TSC1 and TSC2 normally form a complex which downregulates the mammalian target of rapamycin (mTOR) pathway, a regulator of cellular proliferation, but in their absence, mTOR hyperactivation occurs. This causes the development of benign tumors throughout multiple organ systems, leading to systemic problems, the most concerning of which are neurological symptoms like seizures and autism spectrum disorder.

A major downstream target of mTOR is autophagy, a catabolic process which breaks down cellular waste, and whose dysregulation may contribute to TSC pathology. As mTOR normally suppresses autophagy, in TSC autophagy may be excessively inhibited. Rapamycin (a direct mTOR inhibitor) can induce autophagy and has demonstrated beneficial effects in TSC mouse models, however, the mTOR pathway regulates many cellular processes, making side effects of rapamycin numerous. This study was designed to: 1) determine whether autophagy is dysregulated in a mouse model of TSC, and 2) test the disaccharide trehalose as a potential mTOR independent regulator of autophagy. Evidence has shown trehalose may have the capacity to increase autophagy levels, which could help reduce symptoms of TSC.

To investigate this hypothesis, *Tsc1*^{GFAP} KO mice were treated with 3% trehalose, and autophagy activity was assessed using western blotting of autophagy pathway markers. These markers were compared to rapamycin treated mice, vehicle-treated wild-type, and KO control mice. The results demonstrated that autophagy was inhibited in KO mice compared to control mice, and rapamycin was effective in increasing autophagy levels, however, trehalose did not show a significant shift in autophagy levels relative to controls. Future work will assess whether this negative result with trehalose was due to a mechanistic compensation in the *Tsc1*^{GFAP} KO mouse model, insufficient bioavailability, or some other factor.