**Specific Aims**

Tuberous sclerosis complex (TSC) is a disorder that results in the formation of benign tumors throughout the body, including in the brain[1]. TSC is caused by inactivating mutations in either the TSC1 (hamartin) or TSC2 (tuberin) tumor suppressor genes. Previous studies have found evidence that hamartin and tuberin form a heterodimer that has GTPase-activating protein activity toward Rheb, a negative regulator of mTORC1[2]. Loss of this regulatory activity results in hyperactive mTOR and aberrant growth. Loss of tuberin, in particular, is correlated with more severe phenotypes[1]. TSC is also the leading known genetic cause of epilepsy, which affects 80-90% of TSC patients. Interestingly, some of these patients do not display cortical tubers, suggesting additional mechanisms for seizure induction[3]. *The mechanism of epileptogenesis in TSC is not completely understood*[4][5].

**References**

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