# Effects of 50 mg/kg EGCG Treatment of Ts65Dn Down Syndrome Mice on Novel Object Recognition

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Down syndrome (DS) is caused by trisomy of chromosome 21, and affects 1/700 live births. *DYRK1A*, a gene found in three copies in humans with DS and Ts65Dn DS mice, has been linked to alterations in morphology and function of the brain resulting in cognitive impairment. Epigallocatechin-3-gallate (EGCG), an inhibitor of DYRK1A activity, has been proposed as a possible treatment. Using the Ts65Dn DS mouse model, we examined the effects of EGCG treatment on on hippocampal dependent learning and memory using a novel object recognition task (NOR). A previous study analyzing the effects of EGCG at a concentration 30mg/kg/day showed that there was no genotype or treatment effect in the NOR task when treatment is continuous through testing. In this study, the mice were given 50 mg/kg/day EGCG or water via their drinking water starting at 3 weeks of age. The mice were handled two days before testing and then underwent a series of behavioral tests including NOR. They underwent testing at 3 weeks and 7 weeks of treatment. Treatment was continuous throughout behavioral testing. NOR consists of a box with the objects placed diagonally from each other. The mice underwent 3 days of testing with 15 minute sessions per day consisting of habituation, exposure, and test day, all of which were recorded and analyzed to determine time spent exploring novel object in relation to familiar. The amount of time spent at each object was scored by three independent scorers, blind to genotype and treatment. We observed no genotype or treatment effect at either the 3 or 7 week test results, which is consistent with our past results. A higher dose, along with a more sensitive test of recognition memory, may be needed in order to show a treatment effect on the Ts65Dn mice.

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