

IMPROVED COGNITION IN STREPTOZOTOCIN-RAT MODEL OF SPORADIC ALZHEIMER DISEASE AFTER ORAL GALACTOSE TREATMENT

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OBJECTIVES

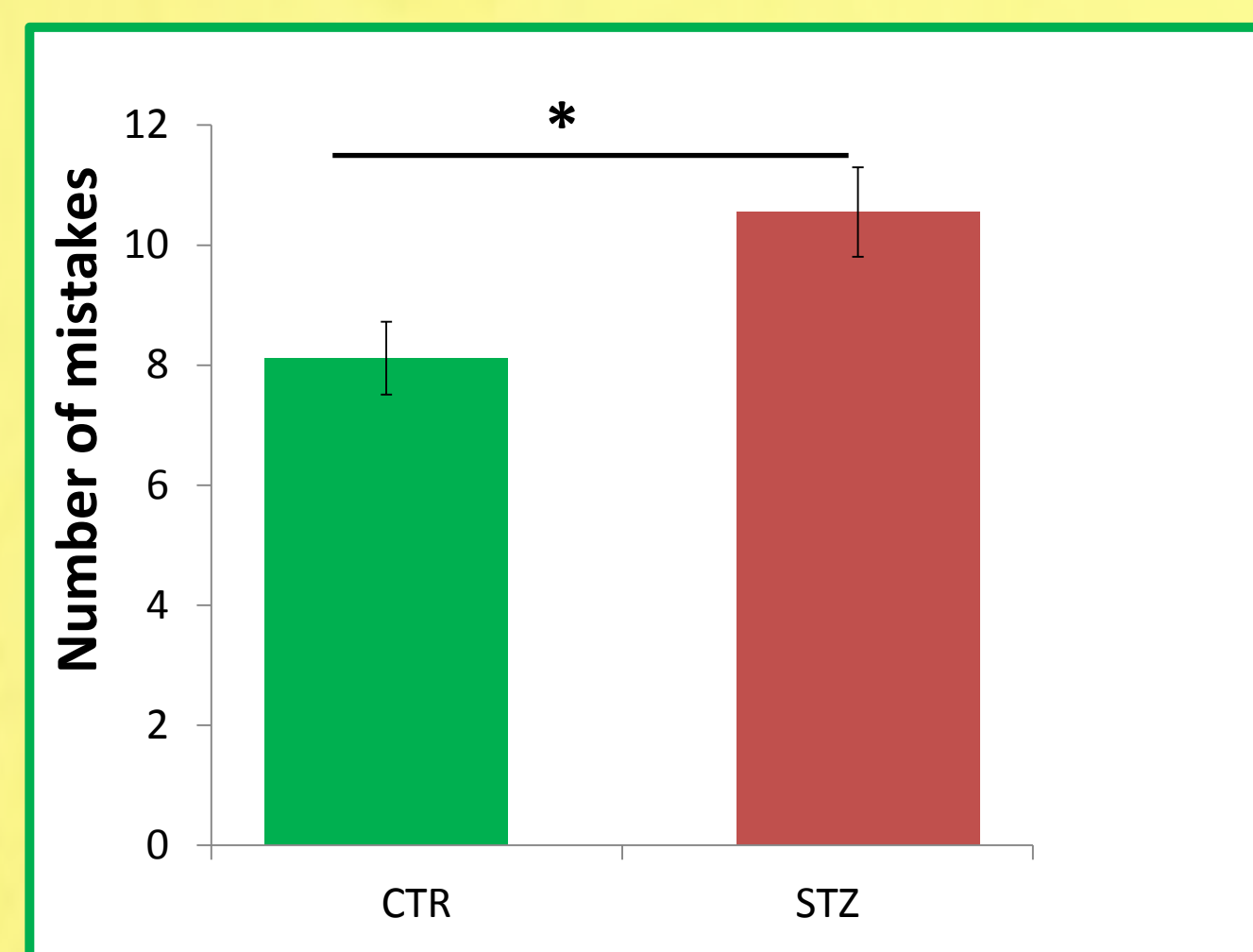
Streptozotocin intracerebroventricularly-treated rats (STZ-icv) have been proposed as a non-transgenic model of sporadic Alzheimer disease (sAD) which demonstrate cerebral glucose hypometabolism and cognitive deficits. Galactose can serve as an alternative energy source and our previous research showed that its oral administration prevented STZ-icv-induced cognitive deficits. We aimed to explore whether chronic oral galactose treatment can improve already developed cognitive deficits in STZ-icv rat model.

MATERIAL AND METHODS

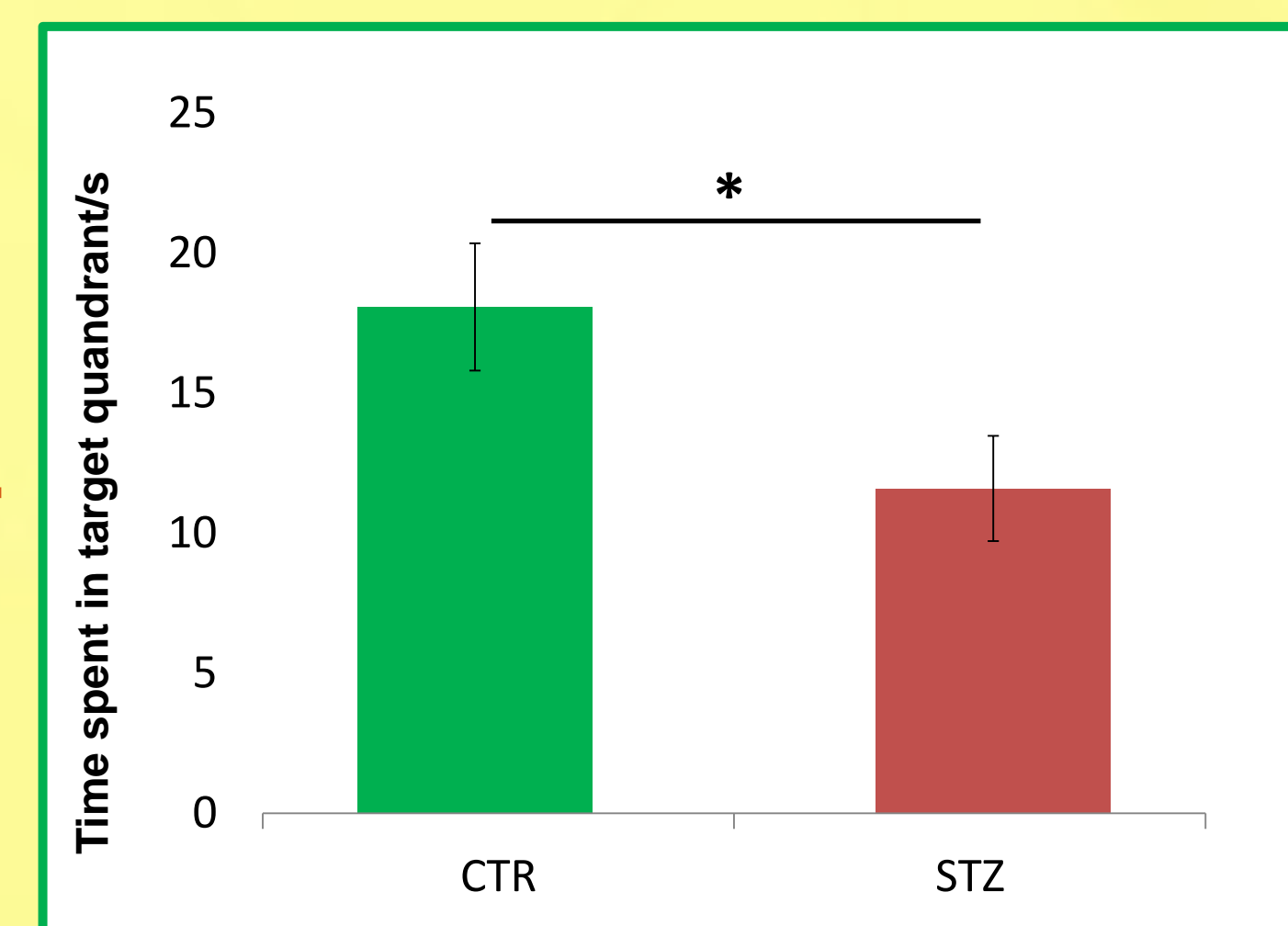
Adult male Wistar rats were given STZ-icv (3 mg/kg) while controls received vehicle only. Oral galactose treatment (200 mg/kg) was initiated 1 month after icv injections and continued for 2 months on daily basis until sacrifice (long-term). Cognitive functions were tested by Morris Water Maze (MWM, before and after galactose treatment) and Passive Avoidance (PA, after galactose treatment) tests at the end of galactose treatment. Data were analyzed by Kruskal-Wallis/Mann-Whitney U-test ($p < 0.05$).

RESULTS

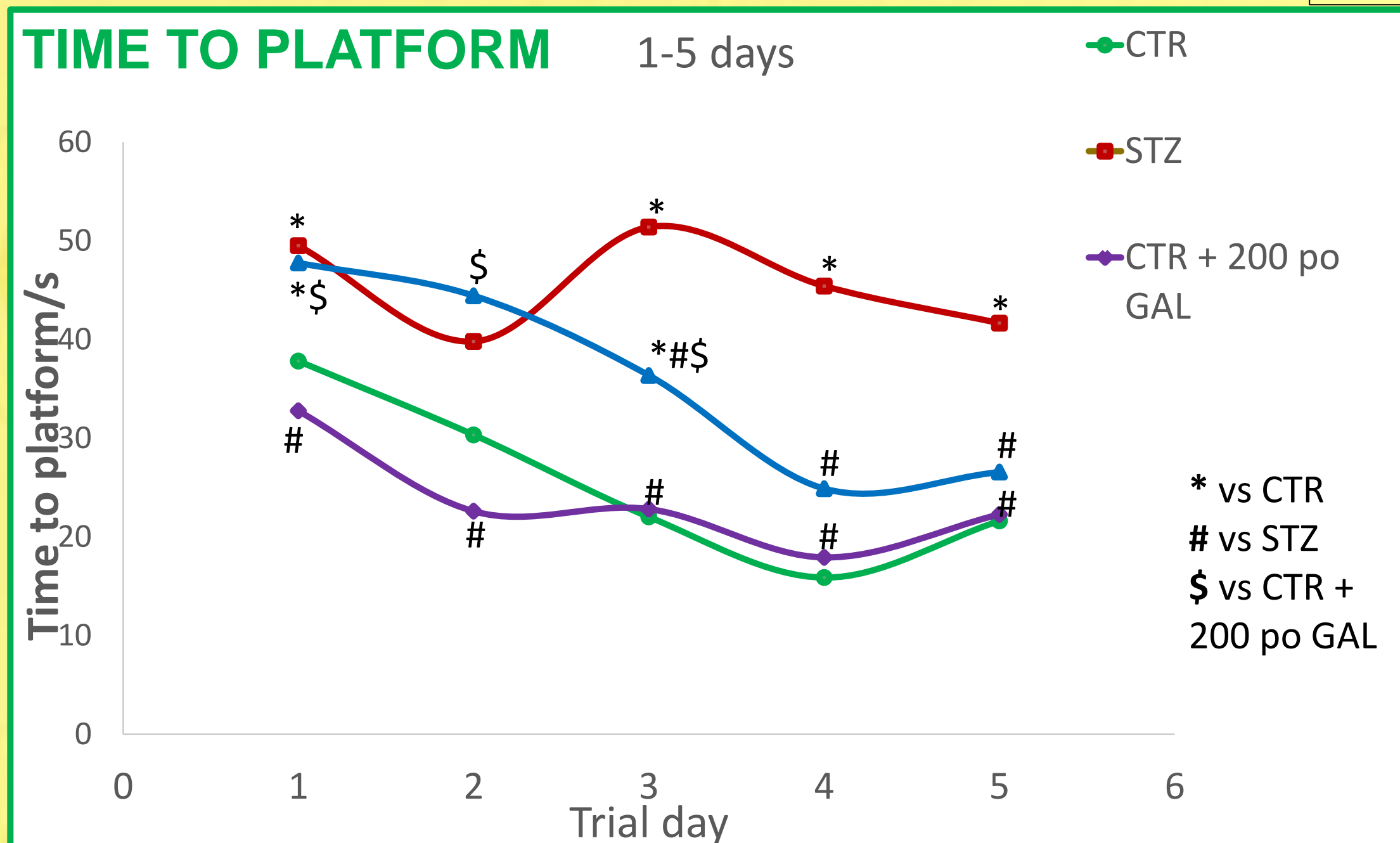
BASAL COGNITION



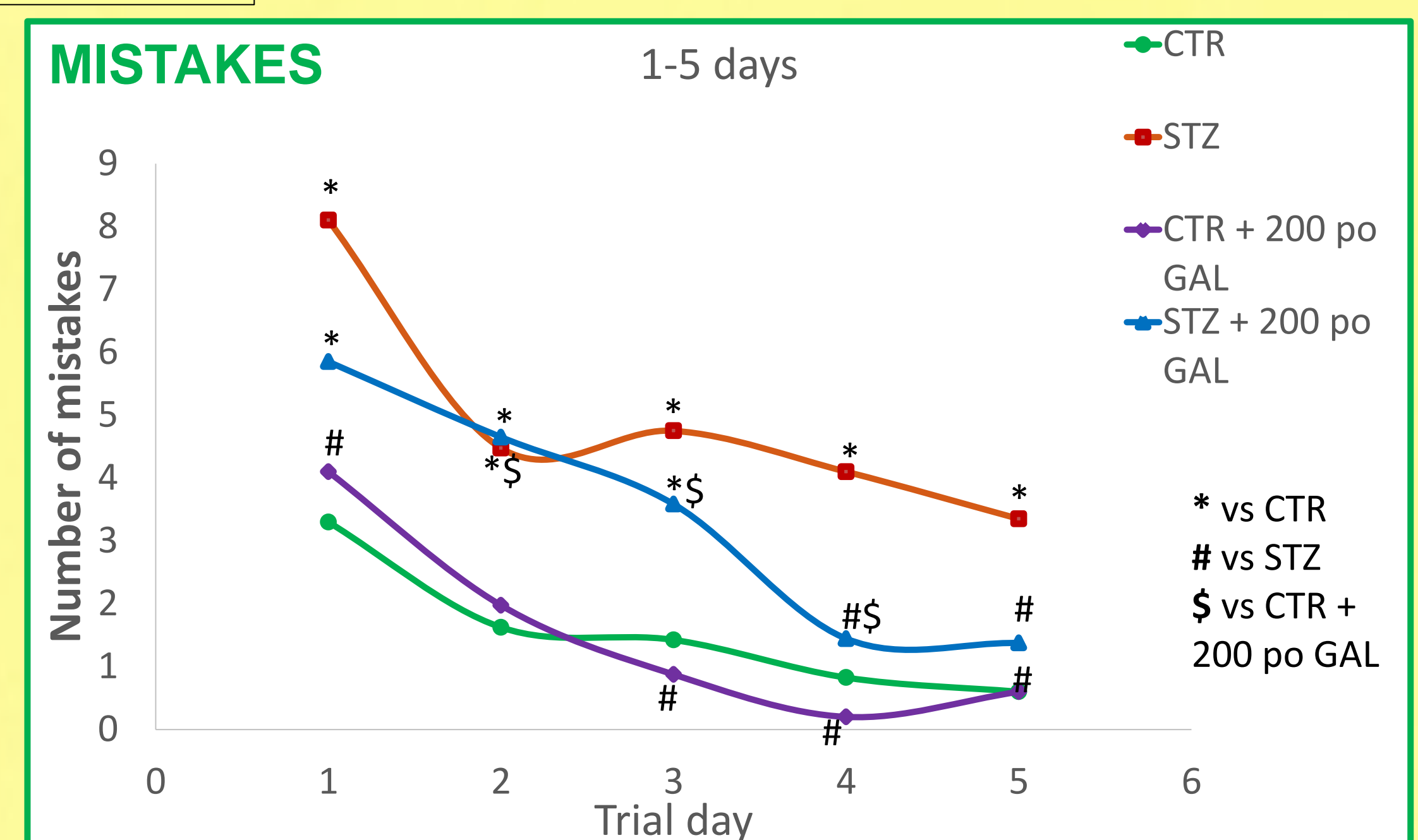
MWM BEFORE GALACTOSE TREATMENT



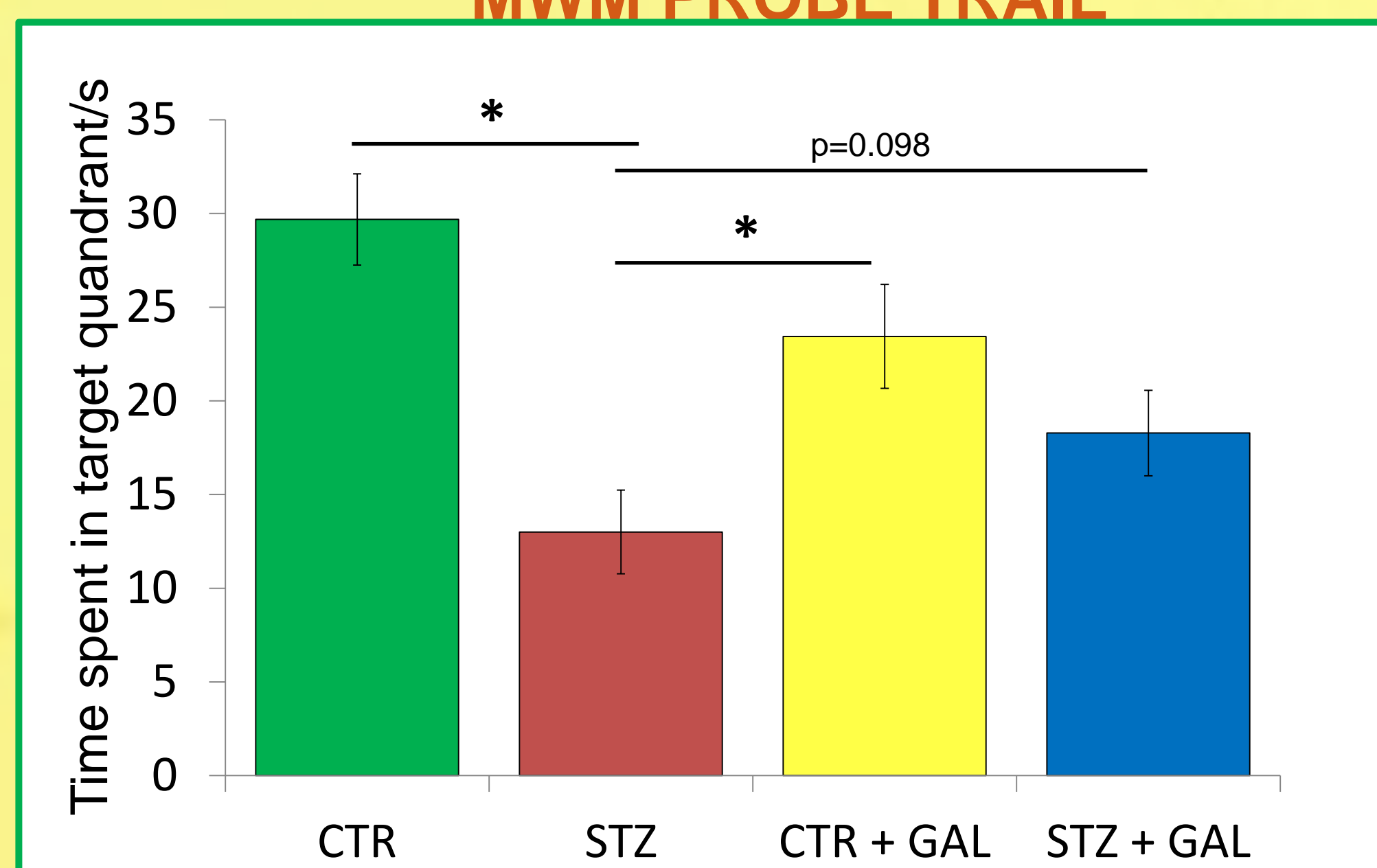
AFTER GALACTOSE TREATMENT



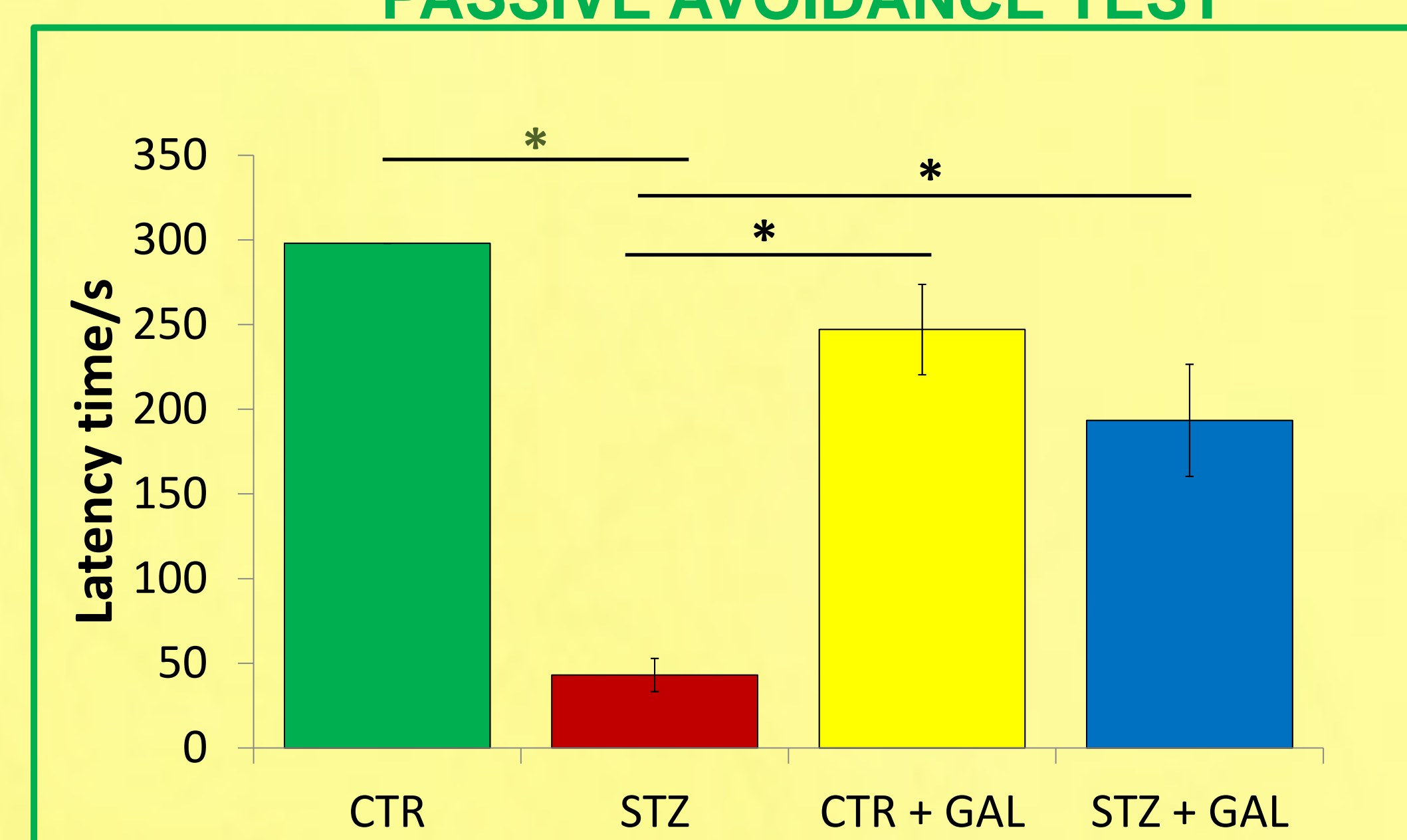
MWM LEARNING TRIALS



MWM PROBE TRIAL



PASSIVE AVOIDANCE TEST



CONCLUSION

Chronic oral galactose treatment normalized STZ-icv induced cognitive deficits, which should be further explored as a possible new strategy in nutrient-dependent sAD treatment.