

Ts65Dn

The Ts65Dn and Dp(16)1Yey [mouse models](#) of DS are both used for [translational research](#) ¹⁾.

Mounting [evidence](#) implicates dysfunctional [GABAA receptor](#)-mediated [neurotransmission](#) as one of the underlying causes of [learning](#) and [memory](#) deficits observed in the [Ts65Dn](#) mouse model of [Down syndrome](#) (DS). The specific origin and nature of such dysfunction are still under investigation, which is an issue with practical consequences to preclinical and clinical research, as well as to the care of individuals with DS and [anxiety](#) disorder or those experiencing seizures in the emergency room settings.

Victorino et al. investigated the effects of GABAAR positive allosteric modulation (PAM) by [diazepam](#) on brain activity, synaptic plasticity, and behavior in Ts65Dn mice. They found Ts65Dn mice to be less sensitive to diazepam, as assessed by [electroencephalography](#), long-term potentiation, and elevated plus-maze. Still, diazepam pre-treatment displayed typical effectiveness in reducing susceptibility and severity to picrotoxin-induced seizures in Ts65Dn mice. These findings fill an important gap in the understanding of GABAergic function in a key model of DS ²⁾.

¹⁾

Liu, C., Belichenko, P. V., Zhang, L., Fu, D., Kleschevnikov, A. M., Baldini, A., et al. (2011). Mouse models for Down syndrome-associated developmental cognitive disabilities. Dev. Neurosci. 33, 404–413. doi: 10.1159/000329422

²⁾

Victorino DB, Pinheiro DJLL, Scott-McKean JJ, Barker S, Stasko MR, Faber J, Scorza CA, Costa ACS. Atypical electrophysiological and behavioral responses to diazepam in a leading mouse model of Down syndrome. Sci Rep. 2021 May 4;11(1):9521. doi: 10.1038/s41598-021-89011-y. PMID: 33947925.

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