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 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 [1].

Memory deficit is the cardinal feature, of Dementia however, the DSM-IV definition requires impairment in at least one other domain (language, perception, visuospatial function, calculation, judgment, abstraction, problem-solving skills) [2].

1)
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2)
Fleming KC, Adams AC, Petersen RC. Dementia: Diagnosis and Evaluation. Mayo Clin Proc. 1995; 70:1093–1107