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The mice with exonic *Reln* deletion show cognitive dysfunction but not attention impairment in the touchscreen operant tasks

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Reelin protein, an extracellular matrix protein, plays an important role in embryonic neuronal migration and layer formation in cerebral cortex. In the adult brain, reelin is produced by GABA-ergic interneurons and affect synaptic plasticity, dendritic morphology and cognitive function. We have recently found a schizophrenia patient carrying a novel exonic deletion in *RELN* gene by CNV analysis on a mainly Japanese population. Meanwhile touchscreen-based operant tasks have been developed for rodents to provide a better translational approach across species for further understanding of the cognition and attention impairment observed in various neuropsychiatric disorders. In the present study, we aimed to explore the performance of *RELN* deletion mice (*Reln*-del), using touchscreen-based visual discrimination (VD) and 5-choice serial reaction time (5-CSRT) tasks. Mice were initially trained to discriminate stimuli simultaneously displayed on the screen and received a liquid reward. The cognitive impairment in *Reln*-del mice was demonstrated by an increase in the number of total trials, total errors trials, total correction trials and total sessions in complex VD task and in complex reversal learning. On the other hand, they showed no significant impairments in 5-CSRT task. These results suggest that the exonic *Reln* deletion lead to an impairment in cognitive function but not in attentional processes.