Alzheimer's disease (AD) is a neurodegenerative disease characterized by extracellular beta-amyloid plaques in regions like hippocampus and frontal cortex, regions highly involved in memory, and by a progressive cognitive decline. The amyloid precursor protein in AD is cleaved by beta-secretase and gamma-secretase releasing abeta42, a highly toxic fragment. In recent years, a link between diabetes type-II and AD was observed: high insulin resistance and increased activity in cell tissue growth factor (CTGF) stimulated gamma-secretase in its release of abeta42. Also, in our lab we observed that tauroursodeoxycholic acid (TUDCA), a bile acid, downregulated CTGF by >10-fold in hepatocytes. Combining these two findings, we hypothesized an effect of TUDCA on abeta42 release. APP/PS1 male mice were fed with TUDCA-containing food pellets for a period of 6 months and were tested at an age of 8 months on several behavioural tasks measuring memory (social recognition, Morris water maze, contextual fear learning and passive avoidance). We observed a clear rescue of different memory types in TUDCA-treated transgenic mice compared to control transgenic mice. No differences were measured in control wild type and TUDCA-treated wild type mice. These findings suggest that TUDCA interfered in gamma-secretase activity, leaving memory formation in TUDCA-treated transgenic mice fairly intact. The brains of these mice, currently being analyzed in immunohistochemistry, will hopefully further strengthen our behavioural findings.