α-synuclein(α-syn) is a major component of Lewy bodies, which are pathological hallmarks in Parkinson's disease (PD). The point mutations (PARK1) as well as multiplications (PARK4) in α-syn gene cause familial form of PD. Recently α-syn gene is also identified as the most susceptible gene for idiopathic PD by genome-wide association study. To make mice model for both familial(PARK4) and sporadic PD, we generated genome-based human α-syn BAC (bacterial artificial chromosome) transgenic (tg) mice harboring both entire human α-syn gene and it's expression regulatory elements. These mice are unique in that human α-syn is overexpressed in an inherent pattern as in normal human brain both temporally and spatially, and they exhibited decreased anxiety-like behavior and attenuated response to methamphetamine in open field test. The non-motor symptoms in PD including autonomic failure, sleep abnormalities, and mood change such as anhedonia and depression sometimes emerge before the motor symptoms, and these have been brought growing attention. These behavioral changes in tg mice possibly reflect the non-motor symptoms and abnormalities of dopaminergic system in the preclinical stage of PD. In conclusion, α-syn BAC tg mice may represent a valuable tool for the analysis of the α-syn gene dosage effects in vivo and pathological mechanisms underlying PD especially in the early preclinical stage.