The leucine-rich repeat kinase 2 (LRRK2) autosomal dominant mutations are responsible for PARK8-linked PD. Lrrk2 gene contains 51 exons and its encoded protein is unusually large (2527 amino acids). Lrrk2 mRNA is expressed throughout the brain and in some other organs, but little is known about the biological substrates and true physiological role of LRRK2. Recently, a number of groups have started to report data on LRRK2 transgenic mice that express various pathological (G2019S or R1441C) forms of LRRK2 using BAC, Thy-1, or endogenous promoters. In the present studies, G2019S knock-in (KI) mutation which is expressed under the control of endogenous regulatory elements has been generated. We then tested the G2019S KI mice (3-4, 12-13, 18-19 months) in a battery of motor tests (basal or stimulant induced locomotor activity, grip strength, pole test, balance beam and static rods). A second cohort of mice at 14-16 months was evaluated using additional behavioural tests (spontaneous alternation in Y maze and psycho vigilance task in operant chamber, rotarod, SHIRPA). This detailed behavioural phenotyping indicated that G2019S KI mice had no functional deficits on any of the basal behavioural tests at any age. Only at 18 months of age, these mice showed an increase of locomotor response after amphetamine challenge. These data indicate that knock-in mutations in LRRK2 do not appear to effect basal motor function or more complex behaviour in C57Bl6 mice. However, G2019S mice appear to develop abnormalities in striatal function at 18 months and we are further exploring this using microdialysis.