Dominant mutations in the LRRK2 gene are the most common cause of familial Parkinson’s disease (PD). Studies show that LRRK2 functions in various cellular processes including regulation of protein translation, endocytosis, and autophagy. How PD associated LRRK2 mutations interfere with these functions is unknown. To gain more insight into LRRK2 function and how mutations lead to PD, it is important to determine the exact localization of LRRK2 in the brain. Although sub-cellular localization studies indicate that LRRK2 is associated with membranes at synapses, localization of LRRK2 in situ has so far been hampered by the lack of verifiable LRRK2 specific antibodies. Here we demonstrate by immunohistochemistry (IHC) that LRRK2 is widely expressed in neurons of various mouse brain regions including cortex, cerebellum, and striatum. Expression is undetectable in LRRK2 knockout mice. Strikingly, expression of LRRK2 in the striatum is enriched in specific compartments identified as striosomes, by co-staining with striosome specific antibodies and absence of co-staining with matrix specific antibodies. Furthermore, LRRK2 expression is not required for initiating or maintaining striosome/matrix compartmentalization. First, compartmentalization can be detected in LRRK2 KO mice. Second, developmental studies indicate that striatal compartmentalization is already present before the initiation of LRRK2 expression. Interestingly, LRRK2 protein expression in substantia nigra pars compacta (SNpc) is low to undetectable. Future work will investigate the role of LRRK2 in the striosome-SNpc pathway and its relevance to PD.