Mutations in the **PARK8** gene, encoding LRRK2, are the most frequent known cause of Parkinson’s disease and are associated with diverse neuropathological features. LRRK2 is a cytosolic multifunctional protein that belongs to the ROCO family of proteins which are characterised by a Roc domain with intrinsic GTPase activity and a COR domain. The modification of LRRK2 GTPase and kinase activity by familial Parkinson’s disease mutations in the Roc, COR and kinase domains is believed to lead to neuronal cell death but the pathways involved remain elusive. We previously characterised a direct interaction between LRRK2 and dishevelled (DVL) family phosphoproteins (DVL1-3), key signalling proteins in *Wingless/Int* (Wnt) signalling pathways. Wnt signalling cascades play an important role in axon guidance, neurite outgrowth/branching, synapse formation, differentiation of dopaminergic cells in the ventral midbrain, release and recycling of dopaminergic vesicles at presynaptic sites and have also been linked to neurodegeneration. We demonstrated that pathogenic mutations in the LRRK2 Roc and COR domains modulate interactions with DVL proteins and now present compelling evidence that LRRK2 modulates Wnt signalling pathways. The LRRK2 RocCOR tandem domain was sufficient to regulate Wnt signalling, but both LRRK2 GTPase and kinase activity had an effect on the magnitude of the modulatory influence. We further demonstrate the association of LRRK2 with Wnt signalling components and the influence of pathogenic LRRK2 mutations on Wnt signalling cascades.