**Conclusion**

Parkinson's disease is a chronic progressive neurodegenerative movement disorder characterized by a profound and selective loss of nigrostriatal dopaminergic neurons. Our findings demonstrated that glutamatergic system is impaired during PD. The evaluations of these damages have important implications in understanding the molecular mechanism underlying motor, cognitive and memory deficits in PD. Our results showed a significant increase of glutamate content in the brain regions of 6-OHDA infused rat compared to control. This increased glutamate content caused an increase in glutamatergic and NMDA receptors function. Glutamate receptor subtypes- NMDAR1, NMDA2B and mGluR5 have differential regulatory role in different brain regions during PD. The second messenger studies confirmed that the changes in the receptor levels alter the IP3, cAMP and cGMP content. The alteration in the second messengers level increased the expression of pro-apoptotic factors - Bax and TNF-α, intercellular protein - α-synuclein and reduced the expression of transcription factor - CREB. These neurofunctional variations are the key contributors to motor and cognitive abnormalities associated with PD. Nestin and GFAP expression study confirmed that 5-HT and GABA induced the differentiation and proliferation of the BMC to neurons and glial cells in the SNpc of rats. We also observed that activated astrocytes are playing a crucial role in the proliferation of transplanted BMC which makes them significant for stem cell-based therapy. Our molecular and behavioural results showed that 5-HT and GABA along with BMC potentiates a restorative effect by reversing the alterations in glutamate receptor binding, gene expression and behaviour abnormality that occur during PD. The therapeutic significance in Parkinson’s disease is of prominence.