

direct pathway drive. Although this, as expected, directly inhibits hyperkinesia, it can never account for the neuronal cell death in the striatum. Huntington's patients therefore will always have a tendency to persist in erratic movement.

Unlike Parkinsonism, Huntington's has a proven genetic factor – glutamine repeat expansion in the Huntington gene. Although this very precise genetic determinant has been discovered, the molecular pathophysiology of Huntington's has yet to be elucidated. Discovery of these molecular mechanisms is crucial to the understanding of selective striatum dopaminergic neuron death. Ahmed et al (2015) showed a strong negative correlation between the Huntington gene and inositol polyphosphate multikinase (IPMK) concentration in cell culture. Although IPMK is intrinsically involved in the synthesis pathways of inositol polyphosphatases (of which IP3 regulates calcium influx), Ahmed et al could detect no measureable calcium aberrations. Therefore Ahmed was unable to describe causality, only correlation, and the molecular mechanisms of Huntington's remain anonymous.

In conclusion, experimental evidence produced thus far demonstrates the significance of basal ganglia lesions in causing neurodegenerative diseases like Parkinsonism and Huntington's. Although many details have yet to be discovered, methods of treatment and clinical presentations appear to fit the basal ganglia model. The systems of the direct and indirect pathway appear to be correctly established and provide a substantial tool to model disease states. By further understanding the molecular mechanisms (and possible further sub types of dopaminergic neurons) there is the hope that curative treatment could be produced for Huntington and Parkinson patients rather than simply palliative care.

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