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Pathogenesis of Tourette Syndrome: clues from clinical phenotypes

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Tourette Syndrome (TS) is a neurodevelopmental disorder affecting 1% of children and it is often mis-understood and under-diagnosed. TS is highly heritable yet genetically heterogeneous. The genetic heterogeneity also links to clinical heterogeneity and this session will trace the pathogenesis of TS from genotypes to clinical phenotypes including the commonly occurring co-morbidities such as ADHD and OCD. The role of fronto-striatal pathways will be discussed to illustrate how these neuronal circuits serve as the final common pathway in translating genetic vulnerability to tics and related behaviours. Evidence from genetic, neuroimaging and phenomenological data sets suggest that there are different subtypes of TS and OCD and that some forms of OCD are alternative phenotypic expressions of the putative TS gene(s) with gender dependent differences in the phenotypic expression. Newly emerging data including neurophysiological findings suggest that the improvement in tic symptoms with age may be the result of frontal compensatory responses, with frontal cortices becoming more efficiently connected to the striatum and to the motor and sensorimotor cortices. Thus research exploring the neuronal circuitry in relation to sensorimotor gating, procedural learning, and habit formation as well as its genetic underpinnings has implications for understanding the genesis, course and outcome as well as the management.

Learning objectives:

- Be able to recognize TS including the common co-morbidities
- Understand the genetic and neurobiological factors that underpin the translation of biological vulnerability to clinically significant symptoms
- Integrate information in relation to the links between neuronal substrates and circuitry to clinical symptoms and implications for management.