

Parkinson's gene mutated in cancer

Date: 29/11/2009 18:00:52

PARK2, a gene commonly mutated in early-onset Parkinson's disease, is also mutated in glioblastoma, colon cancer and lung cancer, according to a new study published online in this week's *Nature Genetics*. This suggests that while germline mutations in PARK2 cause neuronal defects that lead to Parkinson's disease, somatic mutations in PARK2 contribute to cancer.

Parkinson's disease is a common neurodegenerative disorder that affects more than four million people worldwide. Most cases of Parkinson's disease occur in people with no familial history of the disorder. However, 15% of cases occur in patients with familial history of the disease, many of whom carry mutations in known genes, including PARK2. Germline mutations in PARK2 lead to early-onset loss of specific neurons in the brain, but PARK2 is also known to be expressed in other tissues, including the lung, colon and testes.

Timothy Chan and colleagues report that they found deletions within both alleles of PARK2 in about 2% of glioblastoma – an aggressive type of brain tumour – and 6% of colon cancer samples. The scientists also identified mutations in the PARK2 gene in glioblastoma and lung cancer. Functional experiments show that these cancer-specific PARK2 mutations affect the ability of PARK2 protein to suppress cell growth.

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Source Article DOI: [10.1038/ng.491](https://doi.org/10.1038/ng.491)
