Environmental Factors in Parkinson's Disease

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Idiopathic Parkinson's Disease (IDP) is traditionally regarded as a sporadic condition with possible environmental etiologies. A large number of epidemiological surveys have further demonstrated influence on disease incidence by such external influences as smoking, caffeine consumption, pesticide exposure, heavy metals, and dairy products, and internal influences such as cholesterol metabolism, uric acid levels, and hormone status. With rapid expansion of genetic data and genome wide association studies, Mendelian forms of Parkinson's disease and putative susceptibility loci have been uncovered and may shed some light on differences in disease incidence across populations, as well as risk factors in those populations. Nevertheless, the etiologic basis for sporadic disease is unclear, with mitochondrial dysfunction and oxidative stress figuring prominently in the pathogenesis. In this respect, is interesting to note that proteins affected by all germline mutations in familial Parkinson's disease have to date been shown to have a physiological role as an anti-oxidant and/or in mitochondrial metabolism, a finding that parallels other neurodegenerative diseases (amyloid precursor protein in Alzheimer' disease, prion protein in prion disease, Cu-Zn superoxide dismutase in amyotrophic lateral sclerosis). Taken together, IDP is an age-related acquired process, albeit with genetic susceptibilities that are as yet incompletely characterized. Whereas genetic constructs tend to target downstream events (e.g. alpha-synuclein aggregation) and treatment of fully developed disease, a better understanding of the non-genetic influences, and post-translational processes such as oxidative stress and mitochondrial dysfunction, may be more critical to disease prevention per se.