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Characterization of the metabolome in brain following deep brain stimulation: Uncovering the workings and role of antioxidants

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Metabolomics is the newest division of the "omics" sciences and involves holistic analysis of the metabolites contained in a biological sample allowing for quantification and classification the physiological state of cells by analysing endogenous metabolites present. Currently, metabolomic studies are used to discover biomarkers of pathologies and to understand disease etiology through overarching metabolic pathways. Deep-brain stimulation (DBS) is now an accepted therapy for the treatment of patients with movement disorders including Parkinson's Disease (PD). Despite clinical successes, little is known about the mechanisms underlying DBS. Majority of studies are focused on changes in neuronal activity in either the immediate stimulated target area (inhibition) or excitation of more distant targets and circuits. One of the leading hypotheses for DBS action is that it locally inactivates the targeted neuron populations. However, DBS has also been shown to have influence on and affect brain regions at quite distal sites from the point of stimulation and to affect, not only, neurons in stimulated targets, but also astrocytes, microglia and possibly have longer distance effects resulting in recruitment of progenitor cells from ventricular zones into stimulation sites. We hypothesize that recruitment and functional changes in these cell types contribute at least in part to the therapeutic effect of DBS and are critical to long-term DBS treatment efficacy. The UF DBS Brain Bank repository currently has 64 brains, which are available for study. We postulate that these changes are mediated in part by unique anti-inflammatory metabolites resulting from the action of DBS, which restore microglia to their more normal state, hence protecting dopaminergic death via oxidative stress. Therefore, a comprehensive understanding of DBS-related changes in brain cell function and organization will be critical to fill a knowledge gap. This information can be used to develop improved and novel strategies for the long-term treatment of PD.