Voxel-based morphometry indicates relative preservation of the limbic prefrontal cortex in early Huntington disease.

Abstract:
In Huntington disease (HD), both the genetic defect and mutant gene product huntington are known but the exact mechanisms that lead to neuronal loss are poorly understood. Until now, the distribution of tissue loss throughout the brain has been investigated intensively. Here we searched for areas that, antipodal to the striatum, display grey-matter (GM) preservation. We performed high resolution T1-weighted magnetic resonance imaging and voxel-based morphometry in 46 patients in early HD and 46 healthy controls. We applied an analysis of covariance (ANCOVA) model with the total GM volume of each participant as covariate. In accordance with earlier reports, group comparisons revealed GM decrease in the striatum, insula, and thalamus as well as in dorsolateral frontal and occipital areas. In contrast, the limbic prefrontal cortex displayed GM preservation. Our findings support hypotheses that postulate differential involvement of frontosubcortical circuits in the pathophysiology of HD.