EVALUATION OF OXIDATIVE STRESS AND MITOCHONDRIAL DYSFUNCTION IN ANIMAL MODELS AND PATIENTS OF HUNTINGTON’S DISEASE USING CU(II)-ATSM PET.

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Country

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EVALUATION OF OXIDATIVE STRESS AND MITOCHONDRIAL DYSFUNCTION IN ANIMAL MODELS AND PATIENTS OF HUNTINGTON’S DISEASE USING CU(II)-ATSM PET.

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Huntington's disease

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Research Abstract
Huntington’s disease (HD) is a hereditary neurological disorder characterized by a distinctive degeneration of the striatum. The genetic defect is an expansion in the number of CAG codon repeats located in the coding region of HD gene, which codes for a 350 kDa protein, huntingtin. Several pathological mechanisms have been proposed for HD neurodegeneration, including oxidative stress and mitochondrial dysfunction. Cu(II)-diacetyl-bis(N4-methylthiosemicarbazone) (Cu(II)-ATSM) are a group of radiopharmaceuticals used for positron emission tomography (PET) and they have been previously applied in the visualization of regional oxidative stress produced by mitochondrial dysfunction in patients of other neurological disorders, namely Parkinson’s disease. In this study, we will evaluate the oxidative stress in brains of living wild-type, HD transgenic YAC128 mice, expressing full-length human mutant huntingtin using Cu(II)-ATSM PET. Cu(II)-ATSM brain imaging will be extended to symptomatic and asymptomatic HD patients, to clarify the role of mitochondrial dysfunction in HD pathogenesis.

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