FIG. 3 (1065)

factor 2 signaling in Parkinson disease: a promising multi therapeutic target against oxidative stress, neuroinflammation and cell death., CNS Neurol. Disord. Drug Targets. 11 (2012)

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Assessing Tele-Health Outcomes in Multiyear Extensions of Parkinson's Disease Trials (AT-HOME PD): An Update on Recruitment

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Objective: To leverage technology to develop a new model for the long-term follow-up of Parkinson's disease (PD) clinical trial cohorts and investigate novel disease measures.

Background: Long-term follow-up of early PD clinical trial cohorts may generate novel insights about disease progression and heterogeneity. However, infrastructure, personnel, and financial demands render multi-year in-person follow-up studies infeasible.

Methods: AT-HOME PD is a 24-month, remote observational follow-up study of two multi-center phase 3 clinical trials of potential disease-modifying therapeutics for PD (STEADY-PDIII and SURE-PD3) with three platforms: virtual research visits (real-time videoconferencing), mPower 2.0 smartphone application, and Fox Insight (an online survey-based clinical research study). Data from the three platforms is deposited and aggregated in Synapse, a cloud-based data management and research collaboration platform, and then transferred to the Parkinson's Disease Biomarkers Program's (PDBP) Data Management Resource, where it will be integrated with data from STEADY-PDIII and SURE-PD3 and made available to the broader research community.

Results: 262/336 (78%) STEADY-PDIII and 234/298 (79%) SURE-PD3 participants have agreed to be contacted regarding participation in AT-HOME PD. We have conducted 208 screening visits and 192 baseline visits for participants located in 40 U.S states and Canada. 147 (77%) are participating in Fox Insight and 152 (79%) are participating in mPower 2.0. Whole genome sequencing of DNA from over 90% of the parent study participants has been initiated through the Accelerated Medicine Partnership-PD (AMP-PD) and PDBP programs, and is expected to enhance analysis of genetic determinants of AHPD tele-health outcomes.

Conclusions: Over 9 months, we have enrolled approximately 30% of STEADY-PDIII and SURE-PD3 participants into AT-HOME PD. Recruitment may have been hampered by the delayed initiation of AT-HOME PD relative to the conclusion of STEADY-PDIII and SURE-PD3 study visits and negative results of the parent studies.

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Increased lysosphingolipids levels in blood of patients with multiple system atrophy

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Objective: To estimate activity of lysosomal enzymes and lysosphingolipids concentration in dry blood spot of patients with synuleinopathies: Parkinson's disease (PD), dementia with Lewy bodies (DLB) and multiple system atrophy (MSA).

Background: Synucleinopathies are group of neurodegenerative diseases characterized by the abnormal accumulation of alpha-synuclein aggregates which leads to neuronal death. Mutations in the gene (GBA) encoding for lysosomal enzyme glucocerebrosidase proofed to be high risk for PD and DLB. In recent years, the contribution of other genes that encode lysosomal enzymes to the pathogenesis of synucleinopathies has been discovered.

Methods: Our study included 111 PD patients, 13 MSA patients, 11 DLB patients, and 108 controls. Lysosphingolipids (hexosylsphingosine HexSph (glucosylsphingosine (GlcSph)+ galactosylsphingosine (GalSph)), LysoGB3, LysoSM, Lyso509) levels and activity of lysosomal enzymes (Glucocerebrosidase (GCase), Galactosylceramidase (GALC), Alpha-galactosidase (GLA), Alpha-L-iduronidase (IDUA), Acid sphingomyelinase (ASM)) were measured by liquid chromatography-tandem mass spectrometry (LC-MS/MS) in dry blood spots.

Results: The concentration of HexSph (GalSph + GlcSph) was significantly increased in MSA patients compared with all the studied groups ($P<0.0001$). At the same time, an increase of HexSph concentration was detected in LBD patients compared to PD patients ($P=0.045$) but not compared to control group ($P=0.13$). Also, in patients with MSA we found an increase in LysoSM concentration as compared to patients with LBD ($P=0.033$) and control group ($P=0.017$). Moreover, Lyso509 was increased in MSA patients compared to LBD patients (P=0,037) and controls (P=0,042). We found no significant changes in enzymatic activities between the groups.

Conclusions: Here, we demonstrated increased HexSph, LysoSM and Lyso509 concentrations in blood of patients with MSA. In the LBD group, HexSph level was elevated, but not as pronounced as in patients with MSA, the most aggressive synucleinopathy. At the same time, no differences between PD and controls were revealed. The obtained results allow us to extend our knowledge about the pathogenesis of synucleinopathies.

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Early diagnosis of progressive supranuclear palsy using quantitative MRI analysis of brain structure

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Objective: We examined whetherquantitative MRI analysis of brain structure was useful for early diagnosis of PSP.

Background: Quantitative analysis of brain structure by means of MRI in patients with progressive supranuclear palsy (PSP) has showed that distribution of atrophy was useful for distinguishing PSP from Parkinson's disease (PD). However, there has not been reported on the quantitative brain MRI analysis in an early stage of PSP patients.