

European Project on Mendelian Forms of Parkinson's Disease (MEFOPA)

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Research Areas



Biomarker Research

Diagnostic

At a Glance

- Status: **Completed Consortium**
- Year Launched: **2010**
- Initiating Organization: **European Commission Seventh Framework Programme (FP7)**
- Initiator Type: **Government**

Abstract

The European Project on Mendelian Forms of Parkinson's Disease (MEFOPA) is a consortium that advances the basic and clinical research on rare Mendelian forms of Parkinson's disease (PD) by identifying and validating relevant disease-related molecular pathways, drug targets, and biomarkers for disease susceptibility and progression.

Mission

MEFOPA focuses on the molecular pathways underlying inherited forms of PD with autosomal-dominant and autosomal-recessive inheritance. By integrating cellular and animal models, with the latest analytical technologies, MEFOPA aims to provide targets for novel, disease-modifying treatment strategies. In a third subproject, a European registry and biobank for patients with rare Mendelian forms of PD will also be established. Body fluids will be collected and systematically analyzed by unbiased proteomic techniques as well as by focused analysis of candidate proteins, and ex vivo cellular models will be generated, in order to allow validation of disease-related alterations detected in the models analyzed in subprojects 1 and 2.

Through this integrated, translational approach combining basic and clinical research groups, MEFOPA aims to achieve measurable progress in defining the relevant targets and readouts for disease-modifying therapies and will set the stage for rationally designed drug trials in carefully selected groups of patients and even presymptomatic mutation carriers.

The mutations in the dominant genes are thought to cause PD by a gain-of-function mechanism. SNCA- as well as most cases of LRRK2-related PD are pathologically characterized by aggregates of α -synuclein ("Lewy-pathology"). It is therefore appropriate to assume that the pathogenic mechanisms of SNCA- and LRRK2-related PD interconnect. Research on the pathogenesis of those Mendelian forms of PD is coordinated in subproject 1.

Autosomal-recessive mutations in the genes for parkin, PINK-1, and DJ1 are believed to exert their pathogenic effect because of loss of some essential protective function. There is converging evidence suggesting that dysfunction of all recessive PD genes contribute to a state of increased cellular stress because of mitochondrial dysfunction and increased burden of radical oxygen species (ROS) and that at least two of the recessive PD genes, parkin and PINK1, operate within one single pathway. The molecular underpinnings of recessive Mendelian forms of PD will therefore also be investigated together, in subproject 2.

The findings in the basic science work packages of the first two subprojects will directly feed into subproject 3, where a European registry and biobank for patients with rare Mendelian forms of PD will be established in order to advance investigations of biomarkers.

Financing

MEFOPA is funded through the European Union Seventh Framework Programme under Project No. 241791. The European Commission contribution is €6 million.

Homepage <http://www.mefopa.eu/>

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Updated: **04/15/2016**