"Mitochondria, Peroxisomes and Lysosomes - the ""menage a trois"" of cellular metabolism"

Fact Sheet

Project Information

**MITOPEXLYSONETWORK**
Grant agreement ID: 337327

Closed project

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<th>Start date</th>
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<td>1 February 2014</td>
<td>31 January 2019</td>
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**Funded under**
Specific programme: "Ideas" implementing the Seventh Framework Programme of the European Community for research, technological development and demonstration activities (2007 to 2013)

**Total cost**
€ 1 345 200,00

**EU contribution**
€ 1 345 200,00

**Coordinated by**
UNIVERSITAETSMEDIZIN GOETTINGEN - GEORG-AUGUST-UNIVERSITAET GOETTINGEN - STIFTUNG ÖFFENTLICHEN RECHTS
Germany

**Objective**

The metabolic roles of mitochondria, peroxisomes and lysosomes are well established. Numerous genetic defects affecting the function of these organelles
result in a wide spectrum of metabolic diseases. The involvement of these organelles in signalling pathways is receiving increasing attention. Furthermore, interactions between them and other cellular components have been elucidated. Evidence is now emerging that dysfunction in mitochondria, peroxisomes or lysosomes causes secondary perturbations in the other two organelles. The fundamental hypothesis presiding to this proposal is that mitochondria, peroxisomes and lysosomes form an interdependent network (MytoPexLyso), which is likely to have fundamental roles in cell biology, metabolism and metabolic diseases.

To test this hypothesis and elucidate the role of the MitoPexLyso network in physiology and disease, we will employ state-of-the-art imaging and systems biology approaches. First, we will uncover how dysfunction of each MitoPexLyso organelle affects the network. We will test if mitochondrial dysfunction can trigger lysosome biogenesis, and also systematically address how perturbations in one organelle affect the other two. Second, we will identify signalling pathways sensing perturbations on the MytoPexLyso network, and elucidate their pathologic significance, both in cell lines and in animal models of metabolic diseases. Third, we will test a novel strategy to cure mitochondrial diseases: enhanced removal of damaged mitochondria through increased lysosomal autophagic capacity. We will generate a novel mouse model with higher lysosomal capacity in the skeletal muscle, and use a mouse model of mitochondrial myopathy, to test this premise in vivo.

This proposal addresses key questions in cell biology and metabolism, and will lay the foundation for a new field of “organelle networks” which will profoundly impact our understanding of metabolism and metabolic diseases and drive future research endeavours.

**Fields of science**

- natural sciences
- biological sciences
- cell biology
- medical and health sciences
- basic medicine
- physiology

**Programme(s)**

**FP7-IDEAS-ERC - Specific programme: "Ideas" implementing the Seventh Framework Programme of the European Community for research, technological development and demonstration activities (2007 to 2013)**

**Topic(s)**

**ERC-SG-LS4 - ERC Starting Grant - Physiology, Pathophysiology and Endocrinology**
Call for proposal

ERC-2013-StG
See other projects for this call

Funding Scheme

ERC-SG - ERC Starting Grant

Coordinator

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Activity type
Higher or Secondary Education Establishments

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Principal investigator
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Links
Contact the organisation_website

EU contribution
No data

Beneficiaries (1)
UNIVERSITAETS MEDIZIN GOETTINGEN - GEORG-AUGUST-UNIVERSITAET GOETTINGEN - STIFTUNG OEFFENTLICHEN RECHTS

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Other funding
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