TRPML1 in intracellular Fe2+ and Ca2+ homeostasis/signaling

https://neurodegenerationresearch.eu/survey/trpml1-in-intracellular-fe2-and-ca2-homeostasissignaling/

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Country

Canada

Title of project or programme

TRPML1 in intracellular Fe2+ and Ca2+ homeostasis/signaling

Source of funding information

CIHR

Total sum awarded (Euro)

€ 387,774

Start date of award

01/04/2012

Total duration of award in years

5

Keywords
Research Abstract

The lysosome is viewed as a garbage dump and recycling center inside of the cell. Dysfunction of the lysosome causes accumulation of abnormal materials in the cell, eventually leading to cell death which is manifested by what is known as neurodegeneration. One toxic factor is lipofuscin, a non-degradable yellow-brown pigment granule in the lysosome. Because it is often accumulated with age or seen in aging diseases such as Alzheimer’s Disease and Parkinson’s Disease, lipofuscin is also called ‘aging pigment’. Lipofuscin production and accumulation is tightly regulated by the level of lysosomal calcium and iron. By using a unique technique that I developed, we recently showed that lysosomal protein TRPML1 regulates lysosomal iron and calcium levels, and that TRPML1 deficient cells display lipofuscin accumulation. Therefore, I

propose to study the role of TRPML1 in lysosomal lipofuscin accumulation. I hypothesize that an increase in iron-dependent lipofuscin production and a decrease in calcium-dependent lipofuscin exclusion could be the reasons for the accumulation of lipofuscin in TRPML1 deficient cells and ageing cells. These studies will not only help us understand the pathogenesis of Mucolipidosis type IV disease which is caused by deficient TRPML1 but also have broad implications for the understanding and clinical management of neurodegenerative diseases as a whole.

Further information available at:

Types:
Investments < €500k

Member States:
Canada

Diseases:
N/A

Years:
2016

Database Categories:
N/A

Database Tags:
N/A