Determining the role of lysosomal Ca2+ signalling in the pathogenesis of Alzheimer's disease

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Contact information of lead PI Country

United Kingdom

Title of project or programme

Determining the role of lysosomal Ca2+ signalling in the pathogenesis of Alzheimer's disease

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Alzheimer's Research UK

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€ 229,163

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01/10/2015

Total duration of award in years

Keywords Research Abstract

The main aim of this grant is to determine how changes in the recycling compartment of the cell, called the lysosome, are responsible for abnormal protein buildup and cell death in Alzheimer's disease. Like the stomach, lysosomes are acidic and use the acid to help their enzymes break down and recycle proteins. Lysosomes are essential for normal cell function and any problems with lysosomal function often causes severe neurological diseases, usually in children. Our collaborator has found that in familial Alzheimer's disease (FAD) the lysosomes are much less

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acidic, which means proteins do not get digested correctly and this may conribute to Alzheimer's pathology. My group are experts on lysosomal function and we are interested in how changes in lysosomal acidity can affect other key lysosomal functions. We have previously shown that lysosomes are full of calcium, which the cell uses for communication and as a trigger for delivering recycled material to other parts of the cell. Our data indicates that FAD lysosomes had much lower calcium that damatically affects recycling within the cell. We believe that these changes in lysosomal acidity and calcium contribute to AD pathophysiology and represent major new therapeutic intervention points for this disease.

Further information available at:

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