Mitochondrial functions of the Parkinson's disease-associated proteins HtrA2 and Fbxo7

Guest lecture by:

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Understanding of the pathogenesis of Parkinson's disease, a common neurodegenerative disorder, has been significantly furthered by the identification of genetic mutations which cause rare familial forms of the disease. By investigating the physiological functions of the products of these genes, a number of pathogenic mechanisms have emerged and mitochondrial dysfunction in particular has been repeatedly implicated. Two of the genes associated with Parkinson's disease encode the mitochondrial protease HtrA2 and the F-box protein Fbxo7. We have investigated the functions of both proteins at the mitochondria, using predominantly live cell imaging techniques to investigate mitochondrial physiology in HtrA2 knockout mouse neurons, and protein biochemistry and immunocytochemistry to investigate mitochondrial clearance in Fbxo7 deficient cells. This work has revealed a novel effect of HtrA2 on the ATP synthase and an unexpected role for Fbxo7 in mitochondrial maintenance.