# Parkinson's UK Edinburgh Branch Research Interest Group

PARKINSON'S<sup>UK</sup> CHANGE ATTITUDES. FIND A CURE. JOIN US.

#### Newsletter August 2015 Issue No. 12

### View from the Chair

Professor Patrik Brundin gave the fourth Edinburgh Parkinson's Lecture on 22<sup>nd</sup> April 2015 at the Royal College of Physicians of Edinburgh. Once again we attracted a capacity audience, who were treated to a masterly overview of recent research in a lecture with the intriguing title: "*The battle against Parkinson's: the end of the beginning*". You can download the slides and audio files from our web site. A DVD of the lecture is available to borrow – just email ken@edinburghparkinsons.org

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## WPC Scientific Update 2015

Following the success of last year's Scientific Update, WPC has scheduled another set of webcasts for 6<sup>th</sup>-8<sup>th</sup> October 2015. We will again hold a meeting of ERIG to screen extracts and discuss the implications on **Wednesday 21<sup>st</sup> October 2015**, at **Bellevue Chapel** from **2 pm** to **4 pm**.

### **Forthcoming Research Talk**

Dr Maria Doitsidou, Chancellor's Fellow in the Centre for Integrative Physiology at the University of Edinburgh, will give a talk on her research into the degeneration of dopaminergic neurons on **Saturday**, **7**<sup>th</sup> **November 2015** at the Scottish Centre for Regenerative Medicine (SCRM), courtesy of Dr Tilo Kunath. The meeting will start at 10.30 and finish at 12.30 after which there will be a sandwich lunch for those wishing it. Please advise Ken Bowler by email to ken@edinburghparkinsons.org if you wish to attend.

	α-synuclein strains cause distinct faults
Research News:	Misfolded protein aggregates represent a continuum with overlapping features in neurodegenerative diseases, but differences in protein components and affected brain regions. The molecular hallmark of synucleinopathies such as Parkinson's disease, dementia with Lewy bodies and multiple system atrophy are megadalton $\alpha$ -synuclein-rich deposits suggestive of one molecular event causing distinct disease phenotypes.
distinct α-SYN strains display differential seeding capacities, inducing strain- specific pathology and neurotoxic phenotypes.	A recent paper published in <i>Nature</i> on 18 June 2015 assesses the properties of structurally well-defined $\alpha$ -SYN assemblies (oligomers, ribbons and fibrils) after injection in rat brain and claims to prove that $\alpha$ -SYN strains amplify in vivo. Fibrils seem to be the major toxic strain, resulting in progressive motor impairment and cell death, whereas ribbons cause a distinct histopathological phenotype displaying Parkinson's disease and multiple system atrophy traits. Additionally,they show that $\alpha$ -SYN assemblies cross the blood-brain barrier and distribute to the central nervous system after intravenous injection. Their results suggest that distinct $\alpha$ -SYN strains display differential seeding capacities, inducing strain-specific pathology and neurotoxic phenotypes. <b>Journal Reference</b> : $\alpha$ -Synuclein strains cause distinct synucleinopathies after local and systemic administration, Peelaerts W, Bousset L, Van der
	Perren A, Moskalyuk A, Pulizzi R, Giugliano M, Van den Haute C, Melki R, Baekelandt V. <i>Nature</i> . 2015 Jun 18; <b>522(7556):340-4</b> . doi: <b>10.1038/nature14547</b> . Epub 2015 Jun 10.
	Monoclonal antibodies may provide treatment
	Science Daily reports that a team led by Fernando Goni, PhD, an adjunct associate professor of Neurology, and Thomas Wisniewski MD, director of the Center for Cognitive Neurology at NYU Langone, showed that a novel class of monoclonal antibodies successfully targeted proteins that change shape and misfold, becoming toxic and triggering the hallmark beta-amyloid plaques and abnormal tau proteins that are known to accumulate in Alzheimer's and other neurodegenerative conditions. The monoclonal antibodies were also successful at targeting the proteins linked to Parkinson's development. The new research suggests that monoclonal antibodies designed to specifically target these misfolding proteins in soluble, aggregated states, may be ideally suited to treating neurodegenerative diseases.
•	ich web site is at <u>www.edinburghparkinsons.org</u> and the Research Interest .edinburghparkinsons.org/research-interest-group/
	be directed to the Editor and Chair of the Research Interest Group, <u>Ken Bowler</u> inburghparkinsons.org
	's UK is the operating name of the Parkinson's Disease Society of the United A charity registered in England and Wales (258197) and in Scotland (SC037554).