Alzheimer's Drug Discovery and Development
Wednesday, 25 June 2014
Cineworld: The O2, London, SE10 0DX, UK
www.regonline.co.uk/AlzDrug2014

There are currently no treatments that will stop or reverse the progress of Alzheimer’s disease. With an aging population and increasing number of people with Alzheimer’s, the need to develop ways to halt and treat the disease have become paramount. This event will discuss current research into Alzheimer’s drug discovery and development including analysis of current clinical trials.

This event is part of The 2014 Alzheimer’s Disease Congress -www.AlzheimersDiseaseCongress2014.com and has CPD accreditation.

The deadline for abstract submissions for oral presentation is March 10th 2014. Abstracts for poster presentation only can be submitted up to two weeks before the event. You can download the instructions for authors at www.euroscicon.com/AbstractsForOralAndPosterPresentation.pdf

Talk times include 5 – 10 minutes for questions

9:00 – 9:45 Registration
9:45 – 10:00 Introduction by the Chair
10:00 – 10:30 Talk title to be confirmed
  Dr Hugo Geerts, In Silico Biosciences, United States

10:30 – 11:00 New era in AD drug design: intracellular and exosomal targets
  Dr Botond Penke, Professor, University of Szeged, Department of Medical Chemistry, Hungary
AD is a multifactorial disease, caused by a combination of different risk factors. The newest genetic (Jonsson T. 2012) and integrative genomic studies (Rhinn H. 2013) very recently provided solid proof of the amyloid hypothesis. Conventional AD drug development failed for many reasons (the therapeutic targets, e.g. extracellular amyloid, have been incorrect; drugs have been used too late in the disease process; proof of concept problems, etc.). Amyloid pathology correlates with progression at earlier stages of the disease. In the new era of AD drug design we use new targets: intracellular Abeta formation, early stages of Abeta aggregation and direct or exosomal cell-to-cell transmission of prionoids (Abeta and pTau).

11:00 – 11:30 Speakers’ photo then mid-morning break and poster exhibition and trade show
  Please try to visit all the exhibition stands during your day at this event. Not only do our sponsors enable Euroscicon to keep the registration fees competitive, but they are also here specifically to talk to you.

11:30 – 12:00 The role of herpes simplex virus type 1 in Alzheimer’s disease
  Professor Ruth Frances Itzhaki, University of Manchester, Manchester, UK

12:00 – 12:30 Oral Presentations

12:30 – 13:30 Lunch, poster exhibition and trade show
  Please try to visit all the exhibition stands during your day at this event. Not only do our sponsors enable Euroscicon to keep the registration fees competitive, but they are also here specifically to talk to you.
13:30 – 14:30  **Discussion session**  
This discussion session is an informal question and answer session. This is an ideal opportunity to get advice and opinion from experts in this area. This session is not for questions about specific talks, which can be asked after the speakers session, but for discussing either general topics or specific issues. There are three ways you can ask questions:

1. Before the session you can submit your question to Euroscicon staff at the registration desk,
2. Before and during the session you can submit a question or comments, by email, which will be provided on the day of the event
3. During the session you can put your hand up and join in

14:30 – 15:00  **Modulators of γ-secretase activity can facilitate the toxic side-effects and pathogenesis of Alzheimer’s disease**  
*Dr Željko M. Svedružić*, Assistant Professor, Faculty of Medicine, and Department of Biotechnolgy, Univeristy of Rijeka, Croatia  
We describe modulation of Aβ production in cells, by studying how different drug-candidates affect changes in γ-secretase activity caused by gradual increase in Aβ metabolism. Aβ secretion in presence of γ-secretase modulators shows biphasic activation-inhibition dose-response curves. These synergistic activation-inhibition effects can drastically reduce γ-secretase’s capacity to process its physiological substrates, and thus facilitate the toxic side-effects and the pathogenic events. The presented mechanism can explain why moderate inhibition of γ-secretase did not lead to effective therapies, and how quantitative analysis of the catalytic capacity of γ-secretase can be used to prepare novel drug-candidates and early diagnostic strategies.

15:00 – 15:30  **Afternoon Tea, last poster session and trade show**

15:30 – 16:00  **Discovery of multitarget lead candidates to tackle the multifactorial nature of Alzheimer’s disease**  
*Professor Andrea Cavalli*, University of Bologna and Italian Institute of Technology, Italy

16:00 – 16:30  **How to Prevent Dementia and Alzheimer’s**  
*Dr Allen J. Orehek*, Innovator/Physician, Dementia Prevention Center, USA
Dr Orehek’s unique talent is the prevention of dementia. New scientific concepts will open the door to your personal dementia prevention. The brain does not age, however suffers damage over time. The DPC will inform you on how best to identify your baseline or your current degree of damage. Identification of the causes of damage (micron strokes), allows for the mitigation of additional damage. In addition to explaining these new concepts in a medical and scientific manner, Dr Orehek will also provide a mathematical explanation that can be measured, tested, evaluated and judged. Enjoy!

16:30 – 17:00  **Activities of daily living: a new approach to discovering Alzheimer therapies**  
*Dr Robert Deacon*, UK
Activities of daily living (ADL) become impaired in Alzheimer’s (AD). Hitherto, the search for new AD therapies has relied heavily on animal models of learning and memory. At Oxford we have developed simple tests for quantifying ADL of mice. Not only are these tests cheap and sensitive, they provide a novel approach to the search for AD therapies. In terms of mouse welfare, they involve virtually no stress, as deprivation of food/water and aversive motivation is unnecessary, minimising the possibly confounding effects of stress. The aetiological and face validity of these ADL models is enhanced by their dependence on the hippocampus, one of the first brain areas to be affected in AD.

17:00  **Chairman’s Summing Up and Close of Meeting**

**Registration Website:** www.regonline.co.uk/AlzDrug2014
Keywords: Alzheimer’s disease, Amyloid beta, MPC-7869, γ-secretase, AL-108, PBT2, Immunotherapy, Passive immunotherapy, AN-1792, Gamma secretase, LY451039, Tarenflurbil, PBT2, Simvastatin, Allopregnanolone, Angiotensin, Cannabinoids, Methylthioninium chloride, Immunotherapy, neuroinflammation, neurodegeneration, γ-secretase, amyloid, enzyme-mechanisam, FAD mutations, Burrowing, hippocampus, nesting, hoarding, marble, dementia, brain ageing, alzheimer’s, intracellular Abeta, prionoid, exosome, cell-to-cell spreading

About the Speakers


Željko M. Svedružić is currently Assistant professor at Department of Biotechnology and at Faculty of Medicine, University of Rijeka, Croatia. Dr. Svedružić got his training in γ-secretase and the related drug-development efforts, working as a senior scientist on a collaborative project between world leading expert Professor Bart de Strooper and Drug Hunting Team of Eli Lilly Company. Dr Svedružić earned his Ph.D. in enzymology from Oklahoma State University, and did his postdoctoral research at University of California at Santa Barbara, Duke University, and Washington State University.

Allen J. Orehek, MD is an innovator and physician whose unique talent is the prevention of dementia and Alzheimer’s. Board certified in Internal Medicine and Pediatrics, Orehek’s fresh new concepts are designed for the motivated individual. It is a method that has resulted in stopping progression of dementia for many and providing reversal for some. Works include: ’The Micron Stoke Hypothesis of Alzheimer’s Disease and Dementia’ (Medical Hypotheses 78 (2012) 562-570). Prevention is Difficult - But Possible on amazon.com.

Robert Deacon obtained his first degree in pharmacology at the University of Wales, where he also studied psychopharmacology for his doctorate. After learning stereotaxic surgery at Bradford University, he spent 8 years in the pharmaceutical industry (Roussel Laboratories) before starting research at Oxford University in 1991. He has worked extensively on the hippocampus and its function in mice and rats, and about 10 years ago formulated the novel idea that the hippocampus was vital for mediating species-typical behaviours, equivalent to rodent ADL, and proposed that these ADL tests could be used to provide an alternative approach in the hunt for new AD therapies.

Frequently asked questions about our events

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- Can I have the speakers slides?
  We cannot give out the slides from our speaker’s presentations as they are deleted immediately after each event. If you require a particular set of slides please approach the speaker. We will however have a meeting report and you will be emailed when this report is published.

- Can I have a notepad?
  Notepads and pens are provided in the delegate bags and at the registration desk.
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