CP04: Addiction

Speaker: David Nutt, UK

Abstract

My talk will focus on the brain mechanisms of addiction with a particular emphasis on the use of brain imaging [both PET and MRI]. I will discuss the latest research in relation to the neurotransmitter mechanisms of addiction and show how we can develop imaging techniques to explore the modes of actions of existing and novel treatments for addiction.

References

Nutt DJ, Lingford-Hughes A, Erritzoe D, Stokes P (2015) Dopamine and addiction: 40 years of highs and lows Nature Reviews Neuroscience 16: 305–312 doi:10.1038/nrn3939

Nutt, D. J. (2013). The role of the opioid system in alcohol dependence. J Psychopharmacol. doi:10.1177/0269881113504017

Lingford-Hughes AR, S Welch S, L Peters L, Nutt DJ (2012) BAP updated guidelines: evidence-based guidelines for the pharmacological management of substance abuse, harmful use, addiction and comorbidity: recommendations from BAP. Journal of Psychopharmacology 26: 1-54 DOI: 10.1177/0269881112444324 http://www.bap.org.uk/pdfs/BAP-addictionEBG_2012.pdf

Nutt DJ, Lingford-Hughes A, Chick J (2012) Through a glass darkly: can we improve clarity about mechanism and aims of medications in drug and alcohol treatments? Journal of Psychopharmacology 26(2) 199–204

Nutt DJ Lingford-Hughes A (2008) Addiction: the clinical interface. Brit J Pharmacol 154: 397–405

Speaker: Toshikazu Saito, Japan

Abstract

The concept of addiction will be discussed in the context of alcohol-related disabilities. In this lecture, the focus of addiction will be alcohol dependence.

S24: Novel Therapies for Psychiatric Disorders: From Translation to Implementation

Chair: Michael Berk, Australia
Co-Chair: Tijen Uktan, Turkey

Speaker 1: Peter Kalivas, USA

Title: Glutamate Transport: A new bench to bedside mechanism for treating drug abuse

Abstract

Glutamate transmission in cortical synapses into the basal ganglia, in particular into the nucleus accumbens, are markedly altered by addictive drugs. A primary alteration seen after withdrawal from all addictive drugs in self-administration animal models of addiction is a reduction in the elimination of glutamate that is released from these synapses. Specifically, there is a reduction in the glial glutamate transporter EAAT2.

As a result of reduced EAAT2 the fidelity of cortico-accumbens transmission is corrupted such that when an animal trained to associate a cue with drug delivery is shown that cue in the absence of drug, the cue is highly motivating to seek the drug. This level of motivation is associated with a number of transient changes in the cortico-accumbens synapses that collectively indicate that the synapses are transiently potentiated (t-SP). Importantly, the same self-administration protocol for a natural reward such as sucrose pellets does not alter EAAT2, nor is cue-induced sucrose seeking associated with t-SP. After a brief description of the cortico-accumbens neuropathology that appears to contribute to relapse in animal models of addiction, we will discuss the success we and other have had in inhibiting relapse in animal models by pharmacologically restoring EAAT2. In the preclinical literature, restoration of EAAT2 can be accomplished by repeated administration of a number of compounds, including N-acetylcysteine (NAC) and ceftriaxone. However, because NAC is orally active and has a long record of clinical use for acetaminophen overdose and as a mucolytic agent in cystic fibrosis, we and others have examined NAC in clinical trials for treating addiction and other neuropsychiatric diseases that are characterized in part by symptoms of intrusive thinking. Here, I will highlight recent trials with marijuana and cocaine addicts, as well as patients co-morbid for post-traumatic stress disorder (PTSD) and substance use disorder. In these studies, NAC was at least partly effective at reducing craving, relapse and/or criteria for a...