Relapse prevention in addiction – from bench to bedside

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From genes to treatment: The effect of polymorphisms in neurotransmitter systems on addictive behaviour, neural response and relapse

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Introduction The development and maintenance of an alcohol addiction is a complex interaction between genetic and environmental factors. Genetic effects seem to contribute substantially to the risk of developing an addiction, but also to its course and patients' responses to different treatments. Recent studies identified associations between polymorphisms in the genes of glutamate and μ-opioid receptors and addiction risk. Those receptors are of special interest, because they are targets of therapeutic agents, such as acamprosate and topiramate.

Objectives and aims Several studies were conducted, in order to further determine the effects of genetic polymorphisms in glutamate and opioid receptor genes on addictive behavior, neural response to alcohol cues and relapse risk.

Methods Genetic effects were investigated in samples of alcohol-dependent patients using functional imaging techniques, neuropsychological tests and follow-up investigation after standard clinical treatment. Data on clinical parameters, neuronal response to alcohol cues, functional neuronal connectivity and relapse risk were collected and analyzed.

Results Results demonstrate effects of genetic polymorphisms in glutamate and opioid receptors on neuronal response to alcohol cues in frontal and mesolimbic brain areas, subjective craving and time to first relapse. Current findings will be discussed in the light of existing evidence on the contribution of genetic effects to treatment outcome and patient stratification.

Conclusions The investigation of genetic risk factors and mechanisms by which they affect addiction related phenotypes seems to be a promising tool to identify molecular treatment targets and predictors for successful treatment strategies.

Disclosure of interest The authors have not supplied their declaration of competing interest.

http://dx.doi.org/10.1016/j.eurpsy.2016.01.898

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Role of inhibitory processes in relapse prevention treatment

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Alcohol dependence is a chronic disorder with frequent relapses during recovery. Most studies have pointed out that craving is the main process involved in relapse, but recently other factors have been implicated in it, such as attentional bias and impulsivity. Some authors consider that different stages could be involved in the relapse process, and each may be governed by different mechanisms: Attentional bias; motivational response to alcohol cues and inhibitory control.

Motivationally salient cues attract and hold selective attention, and this "attentional bias, (AB)" is related to individual differences in appetitive and aversive motivation. In a recent review, attentional bias has been shown to be significantly present in alcohol-dependent and is associated with craving and risk to a relapse in alcohol consumption.

In alcohol-dependent subjects, alcohol-related cues reach a very high motivational valence (Motivational response, MR), which, in effect, increases craving for alcohol and activates behavioral strategies towards alcohol intake. One method used to assess motivational valence of alcohol is the craving self-assessment. In addition, in recent years, the affective modulation of the startle reflex has been used as an objective measure of craving. It has been shown that subjects with a low baseline startle response when viewing alcohol-associated pictures are at major risk of relapse compared to those with increased reactions.

Once alcohol craving has appeared, the subject will either drink or not, depending on his ability to resist his behavior towards alcohol consumption (impulsivity or inhibitory control, IC). Moreover, subjects that exhibit greater impulsivity are those more likely to relapse.

Our group has recently conducted a study on a sample of 172 alcohol-dependent patients seen in outpatient therapeutic program during 12 weeks. All of them were assessed with the following measures: Attentional bias was assessed using the dot task, motivational response was evaluated using the affective modulation of the startle reflex paradigm, inhibitory control was assessed by the stop-signal reaction time task. Alcohol relapse variables were: relapse, days to the first relapse and days of accumulated abstinence.

One of the most relevant results was that processes related to inhibitory control (Stop-signal reaction time and attentional bias) were the most relevant measures to explain variables related to relapse in alcohol consumption during the treatment period.

Our results support the use of assessment strategies, therapeutic and pharmacological inhibtoria aimed at improving the ability of serious alcohol-dependent patients.

Disclosure of interest The author has not supplied his declaration of competing interest.

Acknowledgements This project has been funded with the EA 1123 ERAB grant application.

http://dx.doi.org/10.1016/j.eurpsy.2016.01.899

Self-related processes and underlying brain networks: Relevance for major psychiatric disorders

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Self-related networks and negative symptoms in psychotic disorders

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Objective Two factors of negative symptoms in schizophrenia have been consistently described based on factor analysis, "expressive deficits" and "social amotivation". We aimed to investigate the putatively differential involvement of self-related networks, as measured with BOLD fMRI during a self-evaluation task, in two dimensions of negative symptoms in schizophrenia (reduced expression and social amotivation).

Method Forty-five patients with a diagnosis of schizophrenia participated in an fMRI study in which they performed a

self-evaluation task. The task comprised a self-reflection, close other-reflection, and a semantic (baseline) condition. We compared correlates of Expressive versus Social amotivation factors (summed items from the PANSS interview) for the contrasts self-baseline and self-other. Significance threshold was set at $P < 0.05$ family-wise error (FEW) corrected.

Results Social amotivation correlated significantly with self-evaluation vs. baseline in right and left ACC, and in the sulcus of frontal lateral lobe between inferior frontal triangularis and middle frontal gyrus. This was also significant, but less pronounced, in the direct comparison of social amotivation vs. expressive deficits scores (for the self-baseline contrast). No activation differences survived critical thresholds for the self-other contrast.

Conclusion Differential neural correlates for the two dimensions of negative symptoms support the validity of this distinction based on factor analyses. Intact functioning of brain circuitry for self-referential processing may be of relevance to actively seek social interaction.

Disclosure of interest The author has not supplied his declaration of competing interest.

http://dx.doi.org/10.1016/j.eurpsy.2016.01.900

S85

The social self in schizophrenia: A neural network perspective on integrative external and internal information processing

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Social impairment is recognized as a basic aspects of schizophrenia. Although the nature of aberrant self-other relationship in schizophrenia is still poorly understood, it has been suggested that some social impairments could have their roots in self-disturbances typical of schizophrenia. For instance, experiencing otherness could become problematic with anomalous self-recognition. Furthermore, deficits in the processing of self-relatedness of social stimuli disconnect the self from its social environment. On the one hand, this could lead to problems in self-other distinction caused by misattributions of ownership of experience and agency in social interaction. On the other hand, this could result in feelings of isolation and reduced intersubjectivity due to interrupted self-referential processing of social stimuli, likely also mediated by memory and emotion. Brain networks involved in self-referential processing, sense of ownership, and agency also have been implied in social cognition. Whereas cortical midline structures are associated with self-referential processing of external stimuli including social information, sensorimotor and affective networks involved in bodily and interoceptive self-processing are also involved in the ability to share others' experiences. Schizophrenia has been linked with a reduced integrity of these networks underlying various aspects of self and social impairments, though rather separately. Recent neuroimaging findings will be highlighted explaining how self-disturbances can pervade the social domain in schizophrenia. In particular, disruptions of the social self in schizophrenia will be addressed from a neuronal network and connectiomics perspective providing a unifying framework.

Disclosure of interest The author has not supplied his declaration of competing interest.

http://dx.doi.org/10.1016/j.eurpsy.2016.01.901

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Psychopathology of the self and the altered cortical midline structures in psychiatric disorders – a marriage?

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The self is central in our mental life and disturbances of the self-figure most prominently in psychopathological symptoms. The cortical midline structures (CMS) have been associated with self-related processing and its changes in schizophrenia, depression and other psychiatric disorders. However, the exact neuronal mechanisms underlying self-related processing in CMS and its changes in psychiatric disorders remain unclear. Especially the neural overlap between high resting state activity levels and self-related processing in CMS is rather puzzling. I present recent data on the rest-self overlap in healthy subjects showing that resting state activity in CMS can predict self-relatedness. The implications for psychological symptoms as in depression and schizophrenia are pointed out.

Disclosure of interest The author has not supplied his declaration of competing interest.

http://dx.doi.org/10.1016/j.eurpsy.2016.01.902

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Brain networks sub-serving self-referential processing in depression

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Introduction Persistent pondering over negative self-related thoughts is a central feature of depressive psychopathology.

Objectives In the present study, we sought to investigate the neural correlates of abnormal negative self-referential processing (SRP) in patients with major depressive disorder (MDD) and its impact on subsequent cognitive control-related neuronal activation.

Aims We hypothesized aberrant activation dynamics during the period of negative and neutral SRP in the rostral anterior cingulate cortex (rACC) and in the amygdala in patients with MDD. We assumed abnormal activation in the fronto-cingulate network during Stroop task execution.

Methods Nineteen depressed patients and 20 healthy controls participated in the study. Using an event-related fMRI design, negative, positive and neutral self-referential statements were displayed for 6.5s and followed by incongruent or congruent Stroop conditions.

Results In contrast to controls, patients did not exhibit valence-dependent rACC activation differences during SRP. A novel finding was the significant activation of the amygdala and the reward-processing network during presentation of neutral self-referential stimuli relative to baseline and to affective stimuli in patients. The fMRI analysis of the Stroop task revealed a reduced BOLD activation in the right frontoparietal network of patients in the incongruent condition after negative SRP only.

Conclusions Thus, the inflexible activation in the rACC may correspond to the inability of depressed patients to shift their attention away from negative self-related stimuli. The accompanying negative affect and task-irrelevant emotional processing may compete for neuronal resources with cognitive control processes and lead thereby to deficient cognitive performance associated with decreased frontoparietal activation.

Disclosure of interest The authors have not supplied their declaration of competing interest.

http://dx.doi.org/10.1016/j.eurpsy.2016.01.903