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EW0178

Psychiatric symptomatology as the initial presentation of brain cancer

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Glioblastoma multiforme is the most common primary adult brain tumor. Clinically, non-specific psychiatric symptoms may arise as their first and only manifestation, prior to any neurological deficits. The most form of psychiatric presentation of neurological diseases are depressive complaints, although these may also be accompanied by behavioral and/or cognitive, anxious and psychotic symptoms. By explaining this case report we aim to emphasize the importance of considering the diagnosis of an organic brain disease, even when only primary psychiatric symptoms are evident. The bibliographic research was made using PubMed and Scielo, and analysis of the electronic patient process. Man of 68 years with a history of hypertension, nephrectomy, splenectomy and left brachial plegia after a car accident. He had been previously seen by a psychiatrist for a 6-month history of depressive symptoms, which had been successfully treated. He later developed new behavioral changes such as heteroaggressiveness, social maladjustment and disfasia, for which he was sent to the emergency room. Brain-CT scan displayed a left front temporal expansive injury. Admitted to the Neurology Department for further diagnostic investigation. Subsequent MRI, detected massive infiltrative lesion with significant mass effect and cystic/necrotic area. The anatomopathology disclosed a glioblastoma grade IV. This case reinforces the importance of carrying a imagiologic workup in cases like this, especially on patients with atypical presentation of psychiatric symptoms.

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EW0179

Differential effects of mGluR5 receptor blockade on behavior, schizophrenia-relevant gene expression and neuronal activation patterns from development to aging mice

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Introduction The glutamate system is implicated both in mood disorders and schizophrenia. Mice lacking metabotropic mGlu5 receptors (mGluR5 KO) display schizophrenia-like abnormalities. Additionally, mGluR5 antagonists represent promising alternative anxiolytics/antidepressants. However, the underlying age-specific molecular/cellular mechanisms are only partially understood.

Objectives We aimed at identifying molecular alterations associated with a genetically induced mGluR5 deletion, which results in a schizophrenia-like phenotype. Additionally, we investigated age-specific effects of mGluR5 antagonists on emotional behaviour and c-fos activation.

Methods For analysis of mRNA and protein levels we performed Real-time RT-PCR and Western blot investigations of brains from mGluR5 KO and wild-type mice. Additionally we used classical behavioral tests for determining anxiety- and depression-like changes triggered by the mGluR5 antagonist 2-Methyl-6-(phenylethynyl)pyridine (MPEP). Finally, we used profiling of c-Fos expression, as marker of neuronal activity, induced by MPEP from postnatal day 16 (P16) to adulthood (P90).

Results We found reduced expression levels of reelin, GAD65, GAD67, parvalbumin, as well as NMDA and AMPA receptor subunits in mGluR5 KO mice, especially in the prefrontal cortex (PFC). We measured age-specific alterations in emotional behaviour of mGluR5 KO mice, with marked increase of anxiety during aging. There was a remarkably conserved activation of the paraventricular nucleus of the hypothalamus, implicated in stress regulation, by MPEP at all investigated ages, whereas the extended amygdala was specifically activated in adulthood only.

Conclusions Our animal data provide new insights into the potential role of mGluR5 in neurochemical and behavioural changes associated with schizophrenia and mood disorders during the life-span.

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EW0180

Influence of personal meaning organization and 5-HTTLPR genotype on cortisol stress reactivity in healthy women

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Introduction Reactivity to acute psychosocial stress in the framework of a physiological multidimensional pattern affects several individual-level systems that include genetic factors and features related to personality development. The 5-HTTLPR genotype has been implicated in the modulation of susceptibility to environmental stimuli.

Objectives In the present study, 91 healthy young women were investigated (i) for their reactivity to a standardized psychosocial laboratory stressor (TSST), as measured by changes in salivary cortisol; (ii) in terms of 5-HTTLPR genotype and (iii) in terms of their personality profile according to the post-rationalist personal meaning organizations (PMOs), which are considered as adaptive modes of response to environmental stressors.

Methods Participants were divided into three 5-HTTLPR genotype groups (s/s; s/l, and l/s). The quantitative and qualitative variables that may affect circulating cortisol were compared among the three groups. A multiple linear quantile regression analysis was then performed to evaluate the effect of the personality profile, as Outward/Inward PMO, and 5-HTTLPR genotype on the median level of cortisol, considered as dependent variable.

Results Comparison of the variables that may affect circulating cortisol no significant differences. Salivary cortisol changed significantly in the course of the TSST. Reactivity to stress was affected by personality profile and the 5-HTTLPR genotype and also by body mass index and age.

Conclusions The present data suggest that the psychosocial stress response is a multidimensional physiological event that is affected by a variety of factors as diverse as 5-HTTLPR genotype, personality profile, BMI, and age.

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EW0181

Skin conductance response to emotional stimuli and injury location in patients with single right hemisphere damage

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Introduction Right hemisphere damage (RHD) has been related to alterations in emotion processing. However, results regarding physiological responses are limited and inconsistent. More research regarding specific brain areas involved in emotional physiological responses is needed.

Objectives To examine the skin conductance response (SCR) to emotion eliciting images in patients with single RHD. To explore the relationship between SCR and brain injury location in patients with single RHD.

Aims To examine the relationship between SCR and cortical and subcortical damage in RH regarding emotional processing.

Method Forty-one individuals with RHD due to stroke were assessed (mean age 68.5, SD 12.2, 51.1 males). The amplitude of event-related SCR was registered through a biofeedback system while observing 54 photographs from the international affective picture system (IAPS). Emotional images were classified using two different approaches: emotional valence (pleasant, unpleasant, neutral) and social vs. non-social content. Brain damage location, determined through medical records, included cortical (frontal, parietal, temporal and occipital lobes) as well as sub-cortical (caudate nucleus, thalamus, lenticular nucleus, insular cortex, basal ganglia and limbic system) structures.

Results Amplitude of SCR to emotional images was significantly lower in individuals with occipital cortex injury compared to those with damage in other brain locations ($P < 0.05$). These results were consistent through all stimuli categories but non-social pictures, which presented the same pattern though, did not reach statistical significance.

Conclusions Results show a relationship between occipital areas in HD and SCR to emotional eliciting stimuli, suggesting occipital right lobe involvement in physiological emotional processing.

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EW0182

The use of polygenic risk scores to inform aetiology of mood and psychotic disorders

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Introduction Polygenic risk scores (PRS) incorporate many small genetic markers that are associated with conditions. This technique

was first used to investigate mental illnesses in 2009. Since then, it has been widely used.

Objectives We wanted to explore how PRS have been used to the study the aetiology of psychosis, schizophrenia, bipolar disorder and depression.

Aims We aimed to conduct a systematic review, identifying studies that have examined associations between PRS for bipolar disorder, schizophrenia/psychosis and depression and psychopathology-related outcome measures.

Methods We searched EMBASE, Medline and PsychInfo from 06/08/2009 to 14/03/2016. We hand-searched the reference lists of related papers.

Results After removing duplicates, the search yielded 1043 publications. When irrelevant articles were excluded, 33 articles remained. We found 24 studies using schizophrenia PRS, three using bipolar PRS and nine using depression PRS. Many studies successfully used PRS to predict case/control status. Some studies showed associations between PRS and diagnostic sub-categories. A range of clinical phenotypes and symptoms has been explored. For example, specific PRS are associated with cognitive performance in schizophrenia, psychotic symptoms in bipolar disorder, and frequency of episodes of depression. PRS have also demonstrated genetic overlap between mental illnesses. It was difficult to assess the quality of some studies as not all reported sufficient methodological detail.

Conclusions PRS have enabled us to explore the polygenic architecture of mental illness and demonstrate a genetic basis for some observed features. However, they have yet to give insights into the biology, which underpin mental illnesses.

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EW0183

Identification of biological pathways to Alzheimer's disease using polygenic scores

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Introduction Single nucleotide polymorphisms (SNPs) contribute small increases in risk for late-onset Alzheimer's disease (LOAD). LOAD SNPs cluster around genes with similar biological functions (pathways). Polygenic risk scores (PRS) aggregate the effect of SNPs genome-wide. However, this approach has not been widely used for SNPs within specific pathways.

Objectives We investigated whether pathway-specific PRS were significant predictors of LOAD case/control status.

Methods We mapped SNPs to genes within 8 pathways implicated in LOAD. For our polygenic analysis, the discovery sample comprised 13,831 LOAD cases and 29,877 controls. LOAD risk alleles for SNPs in our 8 pathways were identified at a P -value threshold of 0.5. Pathway-specific PRS were calculated in a target sample of 3332 cases and 9832 controls. The genetic data were pruned with $R^2 > 0.2$ while retaining the SNPs most significantly associated with AD. We tested whether pathway-specific PRS were associated with LOAD using logistic regression, adjusting for age, sex, country, and principal components. We report the proportion of variance in liability explained by each pathway.

Results The most strongly associated pathways were the immune response (NSNPs = 9304, $= 5.63 \times 10^{-19}$, $R^2 = 0.04$) and hemostasis (NSNPs = 7832, $P = 5.47 \times 10^{-7}$, $R^2 = 0.015$). Regulation of endocytosis, hematopoietic cell lineage, cholesterol transport, clathrin and