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Introduction: Paranoia querulans is a type of persistent delusional disorder of the persecutory subtype, recognized under ICD-10 and DSM-IV. Being a classically described entity, evidence is lacking from its conceptualization as a nosological entity to diagnosis and treatment. Furthermore, controversy still exists regarding its interplay between the judicial and mental health systems.
Objectives: To summarize current evidence and knowledge regarding Paranoia querulans on its conceptualization, ethiopathological explanations, therapeutical management and interface between psychiatry and the law.
Methods: A systematic review was undertaken between June and October 2020 in the PubMed, Web of Science and Scopus databases according to PRISMA directive. Key-terms: ((querul* OR vexatious) AND (paranoia OR delusio* OR neuros* OR behavi* OR complai*) OR litig*) AND psychiatry. No language or time restrictions were established.
Results: A total of 1648 studies were initially identified (PubMed: 679; WOS: 945; Scopus: 24; other: 0); after duplicates were removed, n=1381 studies remained. After screening title and abstract, 56 studies were included. Their main content was categorized into: 1. Conceptualization (n=26): Neurosis (n=5), psychosis (n=9), behavioral disorder (n=5); no psychiatric diagnosis (n=7). 2. Descriptive psychopathology (n=8) 3. Etiopathogenesis (n=9): Social or personality basis (n=3), culture (n=4), trauma (n=1), cognitive decline (n=1) 4. Management (n=1) 5. Psychiatry and Law: same object, different objectives (n=12)
Conclusions: There is controversy regarding the nosological entity of querulousness, from psychosis to neurosis or behavioral disorders. Some authors consider this behavior to not be a psychiatric diagnosis. Furthermore, most papers dealt with a social or nurture-based origin. There is a dearth of information regarding treatment.

Conflict of interest: JPE has received CME-related fees from Lundbeck.
Keywords: Paranoia querulans; Vexatious litigant; Psychiatry and Law; Delusional disorder

Genetics & molecular neurobiology

EPP0700

Systematic review of economic evaluation studies in psychiatric pharmacogenomics

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Introduction: Nowadays, many relevant gene-drug associations have been discovered, but pharmacogenomics (PGx)-guided treatment needs to be cost-effective as well as clinically beneficial to be incorporated into standard health-care.
Objectives: To address current challenges, this systematic review provides an update regarding previously published studies, which assessed the cost-effectiveness of pharmacogenomics testing for the prescription of antidepressants and antipsychotics.
Methods: Our initial screening revealed 1159 articles, which was subsequently reduced to 32 articles, deducted by analysis of their abstract. Full-text analysis performed by all authors resulted in 18 papers that were further included in the analysis.
Results: Of the 18 studies evaluations, 16 studies (88.89%) drew conclusions in favor of PGx testing, of which 9 (50%) were cost-effective and 7 (38.9%) were less costly based on cost analysis. In brief, we found sufficient evidence on the cost-effectiveness of PGx in psychiatric disease care. More precisely, supportive evidence exists for CYP2D6 and CYP2C19 gene-drug associations and for combinatorial PGx panels, but evidence is limited for many other drug–gene combinations. Amongst the limitations of the field are the unclear explanation of perspective and cost inputs in many economic studies, as well as the underreporting of study design elements, which can influence significantly the economic evaluations.
Conclusions: Overall, this systematic review highlights the need for additional research on economic evaluations of PGx implementation with an emphasis on psychiatric pharmacogenomics.

Keywords: Pharmacogenomics; Cost-effectiveness analysis; cost analysis; Systematic review

EPP0705

The role of GSK-3 in mood disorders: Preliminary data from an experimental study

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Introduction: The identification of potential biomarkers is crucial to improve the management and treatment of mood disorders. Glycogen synthase kinase-3 (GSK-3) is a multifunctional enzyme with an important role in the etiology of mood disorders. Recent findings suggested GSK-3 as a putative biomarker in mood disorders.
Objectives: The aims of the study are: - to evaluate GSK3 as potential biomarker for differential diagnosis (MDD and BD); - to analyze the regulation of GSK3 by psychopharmacological treatments.
Methods: Patients included fulfill the following criteria: (a) principal diagnosis of MDD or BD (DSM-5); (b) age ≥ 18 years; (c) drug-free for at least 4 weeks before the inclusion. For each patient included a healthy control is enrolled, matched by gender and age. All included subjects at the study entry point (t0) are assessed through: - semistructured clinical interview and clinical rating scales (Hamilton Depression Rating Scale, Hamilton Anxiety Rating Scale; Young Mania Rating Scale, Clinical Global Impression) - blood draw, to measure GSK-3 levels Patients with MDD or BD are assessed again after 1 week (T1) and after 2 month (T2) of specific pharmacological treatment.
Results: So far, we enrolled 16 patients and 16 healthy controls. The enrollment is still ongoing.
Conclusions: We expect to find GSK-3 levels differently expressed between healthy controls, patients with DDM and patients with BD. This finding would be crucial as it could contribute to the improvement of differential diagnosis. Moreover, we expect to observe a change in GSK-3 levels after psychopharmacological treatments.

Keywords: Mood disorder; Biomarkers; GSK-3; Differential Diagnosis

EPP0706

Mitochondrial ATP production is impaired in neural stem/progenitor cells derived from olfactory neuroepithelium of patients with schizophrenia

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Introduction: Neural stem/progenitor cells derived from olfactory neuroepithelium (hereafter OE-NS/PCs) are emerging as a viable proxy and a valuable tool for translational studies on severe mental illnesses (SMI). In this respect, the use of OE-NS/PCs as a surrogate cellular model of schizophrenia (SZ) has enabled insights into cell signaling and cell cycle dynamics in this disease.
Objectives: We explored whether mitochondrial dysfunction, which has been already associated with SZ, may have a role in the altered proliferation pattern previously observed in OE-NS/PCs of SZ patients.
Methods: OE-NS/PCs were collected from 20 patients and 20 healthy controls (Hcs) by nasal brushing, cultured in proper medium and expanded. Fresh OE-NS/PCs at passage 3 of both groups underwent BrdU proliferation assays or were frozen for later use. Mitochondrial ATP production was measured in both fresh and thawed OE-NS/PCs by using the ATPlite Luminescence Assay kit.
Results: Fresh OE-NS/PCs of patients grew at a higher rate than those of HCs (M-W U=0; p<0.001), whereas the proliferation of thawed OE-NS/PCs of both groups exhibited an opposed pattern (at passage 6, p=0.002). Mitochondrial ATP production was significantly lower in OE-NS/PCs of patients than in those of HCs (M-W U=0; p=0.02), regardless of freeze-thaw conditions (M-W U=6; p=0.77).
Conclusions: Mitochondrial ATP production is negatively affected in OE-NS/PCs of SZ patients as compared to those of HCs. This evidence does not differ in fresh OE-NS/PCs and OE-NS/PCs undergoing freeze-thaw cycles, which instead perturb the proliferation pattern of SZ OE-NS/PCs.

Keywords: cellular models; mitochondria; schizophrénia; translational psychiatry

EPP0707

Drug-induced metabolic syndrome hasn't associations with 5-HT receptor genes polymorphisms in patients with schizophrenia

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Introduction: Metabolic disturbances are common in patients maintained on neuroleptics. These abnormalities significantly increase the physical comorbidity and mortality rates due to cardiovascular disease. We hypothesized that 5-HT receptor genes polymorphisms have associations with drug-induced metabolic syndrome development in schizophrenic patients.
Objectives: To investigate the role of polymorphic variants of serotonin receptors genes in the development of antipsychotic-induced metabolic syndrome.
Methods: 467 patients with schizophrenia receiving long-term antipsychotic therapy were investigated. The mean age was 40.0±11.6 years. The standard phenol-chloroform method for DNA isolation was used. Genotyping was carried out on eight SNP's of genes HTR1A, HTR2A, HTR3A and HTR2C with the MassARRAY® Analyzer 4 (Agena Bioscience™) using the set SEQUENOM Consumables iPLEX Gold 96 on the base The Core Facility "Medical Genomics", Tomsk NRMC.
Results: The prevalence of metabolic syndrome was 26.1%. In the study sample, there were significantly more women with metabolic syndrome (56.6%) than men (43.4%) (p=0.002). The majority of patients with metabolic disturbances were aged >40 years (62.3%), versus 40.9% in the group without metabolic disorders (p<0.001). The duration of the disease was statistically significantly higher in the group of patients with metabolic syndrome (p=0.003). We did not find statistically significant associations of polymorphic variants of the studied genes with the development of the drug-induced metabolic syndrome.
Conclusions: Our results do not demonstrate any significant association between allelic variants of serotonin receptor genes and metabolic syndrome in patients with schizophrenia. Conflict of interest. The authors declare no conflict of interest. Supported by Grant of RSF 19-75-10012.

Keywords: Serotonin receptors; Metabolic syndrome; schizophrénia; Genes

EPP0708

Investigation of the role of polymorphic variants FTO gene in schizophrenia patients with metabolic syndrome

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