Introduction: Olanzapine (OL) represents one of the main chooses for the treatment of psychosis. However, OL increase the risk of metabolic syndrome (MS). Previous literature proposes the "Leptin resistance" as a possible hypothalamic pathogenesis of OL induced MS because of the occurrence of weight gain with increased Leptin blood level.
Objectives: The aim of our study is to investigate in a murine model of full-MS phenotype induced by Olanzapine the hypothalamic gene expression of the pathways involved in Leptin receptor signalling to clarify if a Leptin resistance occurs.
Methods: For the experiment C57BL/6Jfemale mice are used.The OL group (n=15) received pure Olanzapine compounded into chow (54 mg/kg of diet). The vehicle group (n=15)is fed with the same chow without OL. Weight gain is measured every 5 days. After 4 weeks of treatment, mice are sacrificed by rapid cervical dislocation. Blood is collected for Glucose, Insulin and Leptin evaluation. Hypothalamus is dissected and RNA-seq is performed.
Results: The OL group shows a significantweight gain compared to Control (p= 0.02). Likewise blood glucose, Insulin and Leptin levels appear increased (p= 0,0089, p= 0,01, p= 0,0012). From the analysis of RNA-seq hypothalamic differentially expressed genes the anorexigenic POMC pathway downstream to the Leptin Receptor shows a 4-fold increased compared to control.
Conclusions: In our study the "Leptin resistance" involvement in OL induced MS is not confirmed. In fact, although there is an increase in blood Leptin, the expression of Leptin receptor downstream pathways shows a significant increase. This could suggest a possible "POMC resistance" mechanism for OL induced MS.

Disclosure: No significant relationships.
Keywords: Translational medicine; Metabolic syndrome; Hypothalamic RNAseq analysis; Psychopharmacology

O229

Predicting the risk of drug-drug interactions in psychiatric hospitals

J. Wolff[1]*, G. Hefner[2], C. Normann[3], K. Kaier[4], H. Binder[4], K. Domschke[3], M. Marschollek[1] and A. Klimke[5]

[1]Peter L. Reichertz Institute For Medical Informatics, Medical School Hannover, Hannover, Germany; [2]Forensic Psychiatry, Vitos Clinic, Eltville., Germany; [3]Department Of Psychiatry And Psychotherapy, Medical Center - University of Freiburg, Freiburg, Germany; [4]Institute Of Medical Biometry And Statistics, Medical Center - University of Freiburg, Freiburg, Germany and [5]Department Of Psychiatry And Psychotherapy, Vitos Hochtaunus, Friedrichsdorf, Germany

*Corresponding author.
doi: 10.1192/j.eurpsy.2021.409

Introduction: The most common medical decision is the prescription of medicines. More than 130 different drugs with proven efficacy are currently available for the treatment of patients with mental disorders.
Objectives: The aim was to use routine data available at a patient's admission to the hospital to predict polypharmacy and drug-drug interactions (DDI).
Methods: The study used data obtained from a large clinical pharmacovigilance study sponsored by the Innovations Funds of the German Federal Joint Committee. It included all inpatient episodes admitted to eight psychiatric hospitals in Hesse, Germany, over two years. We used gradient boosting to predict respective outcomes. We tested the performance of our final models in unseen patients from another calendar year and separated the study sites used for training from the study sites used for performance testing.
Results: A total of 53,909 episodes were included in the study. The models' performance, as measured by the area under the ROC, was "excellent" (0.83) and "acceptable" (0.72) compared to common benchmarks for the prediction of polypharmacy and DDI, respectively. Both models were substantially better than a naive prediction based solely on basic diagnostic grouping.
Conclusions: This study has shown that polypharmacy and DDI at a psychiatric hospital can be predicted from routine data at patient admission. These predictions could support an efficient management of benefits and risks of hospital prescriptions, for instance by including pharmaceutical supervision early after admission for patients at risk before pharmacological treatment is established

Disclosure: This work was supported by the Innovations Funds of the German Federal Joint Committee (grant number: 01VSF16009). The funding body played no role in the design of the study and collection, analysis, and interpretation of data and in writing the manuscrip
Keywords: Drug Prescriptions; Medication Therapy Management; machine learning; psychiatry

O230

Safety of esketamine nasal spray: Analysis of post-marketing reports submitted to the FDA adverse event reporting system in the first year on the market

C. Gastaldon[1,2]*, E. Raschi[3], J. Kane[4] and C. Barbui[1]

[1]University Of Verona, WHO Collaborating Centre for Research and Training in Mental Health and Service Evaluation, Verona, Italy; [2]Neuroscience, Psychological And Psychiatric Science, Science Of Bio Movement, University of Verona, Verona, Italy; [3]Department Of Medical And Surgical Sciences, University of Bologna, Bologna, Italy and [4]Department Of Psychiatry, The Zucker Hillside Hospital, Northwell Health, Glen Oaks, NY, United States of America

*Corresponding author.
doi: 10.1192/j.eurpsy.2021.410

Introduction: The approval of the esketamine nasal spray for treatment-resistant depression in March 2019 by the Food and Drug Administration (FDA), and few months later by the European Medicine Agency, triggered a vivid debate and many concerns, mainly because of the lack of convincing evidence on its efficacy and safety, based on the development programs, approval trials and few post-marketing trials.
Objectives: We aimed to detect and characterize safety signals for esketamine, by analyzing relevant adverse events (AEs) reports in the FDA Adverse Event Reporting System (FAERS) database (March 2019-March 2020).
Methods: We performed disproportionality analysis through the case/non-case approach: reporting odds ratios (ROR) and information components (IC) with 95% confidence intervals (95%CI) were estimated for esketamine-related AEs with at least four counts. We compared serious and non-serious AEs using non-parametrical tests.
Results: The FAERS database registered 962 reports of esketamine-related AEs in one year. Signals (i.e., statistically significant disproportionality) were detected for several AEs, such as dissociation, sedation, feeling drunk, suicidal ideation and completed suicide. Signals for suicidal ideation, but not suicide attempt and completed

suicide, remained significant when comparing esketamine to venlafaxine. The comparison of patients with serious vs. non-serious esketamine AEs revealed that females, patients receiving antidepressant polypharmacy, co-medication with antipsychotics, mood stabilizers, benzodiazepines or somatic medications were more likely to suffer from serious AEs.
Conclusions: This real-world pharmacovigilance analysis detected signals of serious unexpected esketamine-related AEs, thus reinforcing current worries regarding esketamine safety/acceptability. Further real-world studies are urgently needed to unravel the safety profile of esketamine.

Disclosure: No significant relationships.
Keywords: treatment-resistant depression; pharmacovigilance; esketamine; Suicidal risk

O231

Can atypical antipsychotic drugs cause hepatotoxicity?

R. André[1]*, C. Sereijo[2] and M. Abreu[2]

[1]Psychiatry, Centro Hospital Lisboa Norte, Lisboa, Portugal and [2]Psiquiatria, Centro Hospitalar Universitario Lisboa Norte, Lisboa, Portugal

*Corresponding author.
doi: 10.1192/j.eurpsy.2021.411

Introduction: Neuropsychiatric drugs account for 16% of drugs that can lead to hepatotoxicity and psychiatric patients can have multiple comorbidities that can increase the incidence of liver disorders such as alcoholism, drug abuse and polymedication. The continuous use of atypical antipsychotic drugs (AAD) has raised questions over their tolerability over endocrine, metabolic and cardiovascular systems. They are also associated with mild elevation of aminotransferases and occasionally cause idiosyncratic liver injury with varying phenotypes. Hepatotoxicity is defined based on biological parameters such as elevation of alkaline phosphatase enzyme, SGPT, SGOT and GGT or clinical abnormalities (jaundice and hepatitis).
Objectives: This work reviewed the current available evidence on the hepatic damage produced by AAD.
Methods: Non-systematic review of the literature with selection of scientific articles published in the past 10 years; by searching Pubmed and Medscape databases using the combination of MeSH descriptors. The following MeSH terms were used: atypical antipsychotic drugs; hepatotoxicity; hepatic; Olanzapine; Clozapine; Risperidone; Aripiprazol; Paliperidone.
Results: Atypical Antipsychotic Drugs are generally well tolerated and hepatic alterations are in general very low or rare. The cases published were observed with Clozapine, Olanzapine and Risperidone. Atypical Antipsychotic drugs have a better profile than Chlorpromazine.
Conclusions: In conclusion, the hepatic injury generally occurs within the first weeks of treatment and is usually reversible with drug withdrawal. Hepatic check-ups may be relevant, especially in the beginning of treatment.

Disclosure: No significant relationships.
Keywords: Antipsychotics; hepatic damage; atypical antipsychotics; hepatotoxicity

O232

Psychopharmacological treatment in dissociative identity disorder (DID)

R. Pinilla[1]*, C. Rodríguez Sabaté[2], B. Ordóñez Méndez[3], A. Sotillos[4] and A. Hernández Mata[4]

[1]Psiquiatría, Hospital de Getafe, Madrid, Spain; [2]Psychology, Hospital Universitario de Getafe, Getafe, Spain; [3]Psychiatry, Hospital Universitario de Getafe, Getafe, Spain and [4]Psiquiatría, Hospital Universitario de Getafe, Getafe, Spain

*Corresponding author.
doi: 10.1192/j.eurpsy.2021.412

Introduction: Patients with dissociative identity disorder (DID) present two or more identities. Although it is a widely questioned diagnosis, it is currently found in the main DSM-5 and ICD-10 diagnostic manuals. So far there is no standard psychopharmacological treatment for people with this pathology.
Objectives: Describe the pharmacological treatment associated with the clinical evolution of a patient with DID.
Methods: Follow-up was carried out in a mental health center for a year, undergoing psychopharmacological and psychotherapeutic treatment. The information is taken from the medical history.
Results: The patient presents with anxious and depressive symptoms. She was referred from primary care with 50mg sertraline without response. Dose was increased to 100mg without response. New management started with desvenlafaxine 100mg, associated with lorazepam, partially reducing the symptoms. Later, the patient presented self-referentiality, sounding of thought, began to describe frequent memory losses and a rebound in anxiety-depression symptoms, increasing the dose of desvenlafaxine to 200mg and introducing haloperidol to 1.5mg. Three months later, she presented showing another identity, active, aggressive, pythiatic, without evident anxious symptoms that she previously presented in a marked way. Desvenlafaxine was adjusted to 100mg and haloperidol to 0.5mg every 12 hours. The patient evolved favorably, decreasing anxiety, depressive symptoms and memory loss, in addition to disappearing psychotic symptoms. This treatment was sustained, keeping the patient psychopathological and functional stability and allowing a psychotherapeutic approach.
Conclusions: Treatment with desvenlafaxine and haloperidol was favorable to maintain clinical stability and allow other therapeutic approaches.High dose of antidepressant could favor the expression of another identity of the patient.

Disclosure: No significant relationships.
Keyword: antidepresive antipsicotic disociative memory-loss

Psychosurgery & Stimulation Methods (ECT, TMS, VNS, DBS)

O233

The effectiveness of involuntary electroconvulsive therapy (ECT): A population-based study

E. Salagre[1]*, C. Rohde[2], K. Ishtiak-Ahmed[2], C. Gasse[2] and S. Østergaard[3]