results from their usage such as euphoria and relaxation, however these were counter balanced by those who experienced some serious negative emotional and physical side effects such as anxiety, paranoia, palpitations and convulsions. SC appear to often emulate that of their natural counterpart, yet there is an unpredictability to them which can end with serious consequences. Online forum content gives us a strong base understanding of users experiences of SC. Further research is required to elucidate a more nuanced understanding.

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#### EV75

### The "Endless Trip": Psychopathology and psychopharmacology in the Hallucinogen Persisting Perception **Disorder** (HPPD)

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Introduction Hallucinogen Persisting Perception Disorder (HPPD) is a syndrome characterized by prolonged or reoccurring perceptual symptoms, reminiscent of acute hallucinogen effects. HPPD was associated with a broader range of LSD (lysergic acid diethylamide)-like substances, including cannabis, MDMA (methylenedioxymethamphetamine), psilocybin, mescaline and other psychostimulants. Symptomatology mainly comprises visual disorders (i.e., geometric pseudo-hallucinations, halos, flashes of colours/lights, motion-perception deficits, afterimages, micropsy, more acute awareness of floaters, etc.), even though depressive symptoms and thought disorders may be comorbidly present.

Although HPPD was firstly described in 1954, it was Objective definitely established as a syndrome in 2000 with the revised forth version of the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR). However, neuronal substrate, risk factors, aetiology and pathogenesis of HPPD remains still unknown and under investigation. Furthermore, there are still open questions about its pharmacological targets.

A critical review on psychopathological bases, etiological Aims hypothesis and psychopharmacological approaches towards HPPD was here provided.

Methods A systematic literature search on PubMed/Medline, GoogleScholar and Scopus databases without time restrictions, by using a specific set of keywords was here carried out. In addition, a case report was here described.

Results and conclusions Pharmacological and clinical issues are here considered and practical psychopharmacological recommendations and clinical guidelines here suggested.

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

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#### **EV76**

# Psychosis and polydrug abuse in a patient with Dandy-Walker variant

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Background and purpose Dandy Walker "syndrome" (DWS) was firstly defined by Dandy and Blackfan, and then described by Hart et al. [1] as a series of neurodevelopmental anomalies in the posterior fossa, including Dandy-Walker (DW) malformation, DW variant (cerebellar hypoplasia/aplasia of the cerebellar vermis and cystic dilatation of the fourth ventricle), mega-cisterna magna and posterior fossa arachnoid cyst. Mental symptoms have been associated with DWS in previous reports, but the spectrum of mental symptomatology widely varies between clinical cases, ranging from psychotic/schizophrenia-like to mood/cognitive symptoms [2].

Methods Here we describe a case of psychosis and polydrug abuse in a 27-year-old man with DW variant a 4-year history of polydrug abuse, sporadic alcohol abuse, epilepsy and psychotic symptoms including delusions of reference/persecution, suspiciousness, associated with obsessive thoughts, mood lability and persistent anxiety.

Results He was recovered for a 28-day program of detoxification from drug addiction/stabilization of psychiatric symptoms. Family history of Bipolar Disorder, gambling disorder (father) and depression (mother). The mental status examination at baseline revealed slowness of thought, psychomotor retardation, aboulia/anhedonia/apathy/hypomimic facies/asthenia/social withdrawal/deflected mood/poor thought content/blunted affect/selfneglect/poor insight, cognitive impairment and oppositive and partially collaborative attitude and behaviour. Borderline intelligence activity was found on WAIS-R (IQ = 79). At the baseline, he was taking carbamazepine 400 mg BID (baseline serum level: 6.720 µg/ml), gabapentin (400 mg BID), paroxetine (20 mg/d), olanzapine (10 mg/d) and methadone (70 ml/d), with a poor response/control both on psychotic and seeking drug symptomatology.

References not available.

Conclusions Further DWS clinical cases should be evaluated in order to better investigate the role of this variant to addictive and psychotic symptoms.

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### EV77

# Improved drug-use patterns at six months post discharge from inpatient substance use disorder treatment; results from compulsory and voluntary admitted patients

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