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Background: Individuals with cocaine addiction have been shown to have alterations in food intake and eating behaviour, with increased consumption of dietary fat (and to a lesser extent, carbohydrate) and reduced control over their food intake [1]. Cocaine addiction is thought to desensitise individuals to non-drug rewards such as foods and it may be that this leads to a preference for highly rewarding foods such as those high in fat. On the other hand, models of food addiction suggest that there is a cross-sensitisation to rewards and that cocaine addiction could possibly lead to food-related addictive behaviours [2]. Berridge's incentive-sensitisation theory of addiction states that drug addiction involves an aberrant amplification in psychological 'wanting' of a stimulus, without necessarily an increase in the pleasure derived from the stimulus ('liking') [3].
Objective: We sought to investigate whether cocaine-addicted individuals (N = 78) differ from healthy controls (N = 56) in their levels of wanting and valuing foods. We hypothesised that the cocaine-addicted individuals would show decreased wanting and valuing of foods compared with controls.
Methods: Participants were presented with food pictures in a randomised order. For each picture they were first asked their motivation to eat the food on a 9-point scale and then to indicate how much they would be willing to pay for them, regardless of their current funds or appetite. Appetite was measured using the Simplified Nutritional Appetite Questionnaire (SNAQ) [4], and the importance of different motivations for food choice was assessed using the Food Choice Questionnaire [5]. Data were analysed using analysis of co-variance (ANCOVA) models, with the appetite score and food expenditures included as covariates.
Results: As hypothesised, cocaine-addicted individuals report significantly reduced appetite compared with their healthy peers (all $p<0.001$). They also reported having less money at their disposal and spending less money on food compared with the control group. Interestingly, disposable income was related to the amount of money spent on food in the control group ($r = .41, p = 0.002$) but not in the cocaine group ($r = .08, p = 0.478$). Compared with controls, cocaine users' food choices appeared to be influenced more strongly by mood and familiarity, and less by calorie content (all $p<0.05$). Although cocaine-addicted individuals reported being less willing to eat the presented foods ($F1,128 = 19.9, p<0.001$), they reported being willing to pay as much for the food as control volunteers ($F1,128 = 0.22, p = 0.639$). When presented with pictures of their stimulant drug of choice, they rated their willingness to use and pay for the drug extremely highly, significantly higher than their rating of food items (all $p<0.001$).
Conclusion: Our results suggest an imbalance between monetary value attribution (normal in cocaine users) and the motivation to use money (reduced for food stimuli and increased for cocaine). Cocaine-addicted individuals seem to make more habitual food choices (based upon familiarity) and more emotional food choices (based upon mood), suggesting that aspects of choice behaviour seen in cocaine addiction extend to choosing food. However eating in cocaine addiction is an under-researched topic that requires further investigation.

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P.717 Differences in PANSS score progression at first-episode psychosis in children and adolescents with cannabis use; A longitudinal prospective study

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Introduction: In terms of large-scale epidemiology, worldwide, cannabis is the most commonly used illicit psychoactive substance, and third overall, coming after alcohol and tobacco [1].

Cannabis is frequently used among individuals with first episode psychosis and is associated with poor clinical outcomes [2]. However, the association between cannabis use and the risk of psychosis still remains controversial and only few studies have investigated within whether changes in cannabis use are associated with changes in symptom levels [3,4]. Shedding light on this under-researched matter could have clinical implications for this patient group.

Objectives: The aim of this study is to evaluate the impact that cannabis has in clinical evolution on individuals with established first-episode psychosis in children and adolescents sample of patients.

Methods: A total of n = 30 patients with first-episode psychosis who were hospitalized in an acute term stay psychiatric Unit (Hospital Sant Joan de Déu, Barcelona) were registered between October 2014 and Mars 2018. Included patients were followed during 1 year with assessments at baseline (n = 30), 3 months, 6 months and 12 months.

Psychotic symptoms were measured using the Positive and Negative Syndrome Scale (PANSS) positive, negative, and general symptoms scores. Cannabis use was registered by self-report and confirmed by urine drug test. Database information was completed with electronic medical records. Comparative analysis was performed with IBM SPSS Statistics (Chicago INC) using Levene's Test for equality of variances, T-test for equality of means and simple linear regression for PANSS score tendency estimation.

Results: From 31 children and adolescents patients sample, 33% (10) had cannabis use and 67% (20) do not. PANSS score tendency was respectively: $y = -4.8x+41.8$ (general symptoms in users), $y = -8.4x+48.1$ (general symptoms in non-users), $y = -0.56x+15.8$ (positive symptoms in users) $y = 0.28x+15.7$ (positive symptoms in non-users), $y = -2.9x+20.8$ (negative symptoms in users), $y = -3.97x+21.0$ (negative symptoms in non-users). Mean difference between cannabis users and non-users at PANSS score were respectively; at the baseline 3.50 (negative symptoms), 0.5 $p = 0.04$(positive symptoms), 3.10(general symptoms); at 3 months: -4.60(negative symptoms), -2.65(positive symptoms), -8.3(general symptoms); at the 6 months 3.33 (negative symptoms), 3.67 $p = 0.05$(positive symptoms), 5.33(general symptoms); at the year -3.00 (negative symptoms), 5.00 (positive symptoms), 7.50 (general symptoms).

Discussion: According to our results the tendency to decrease PANSS symptoms is more accentuated in those patients who do not use cannabis compared to patients who use cannabis. In terms of symptoms, in both groups, general symptoms are more likely to decrease compared to negative symptoms and these compared to positive symptoms. This difference is statistically significant in positive symptoms at the third month, and positive symptoms and the sixth month. No other statistically differences were found. Therefore, it should be borne in mind to include cannabis use assessment in first-episode psychosis in order to considerate this clinical evolution implication. Psychoeducation and prevention of adolescent cannabis use should be emphasised in adolescents using cannabis. Further studies are warranted to determinate association between psychosis symptoms evolution and cannabis use between adolescents.

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P.718 The influence of GHB-use and GHB-induced coma on affect and the affective network

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Introduction: GHB is a popular drug of abuse which despite its relative low prevalence of use, it is currently the fourth most common drug of abuse presented at emergency rooms [1]. The observed increase in emergency rooms attendances is mainly associated with the presence of GHB-induced comas and paralleled by equally growing numbers of GHB users who seek treatment for their GHB addiction [1–3]. Moreover, GHB has been seen to increase anxiety and decrease emotional discrimination capacity in rodents [4]. Animal studies have also shown that regular use of GHB is associated with oxidative stress incident in highly sensitive brain areas such as the hippocampus or amygdala [5]. However, little is known about the effects of GHB-use and GHB-induced comas on affect regulation in humans.

Aims: Here we tested the following hypothesis: (a) GHB users with multiple GHB-induced comas report more negative affect and show altered amygdala and hippocampus activity and functional connectivity when compared with the GHB users without GHB-induced comas and polydrug users who never used GHB; (b) GHB users without GHB-induced comas report more negative affect and show altered amygdala and hippocampus activity and functional connectivity when compared with the polydrug users who never used GHB.

Method: We recruited 27 GHB users with $\geq$4 GHB-induced comas (GHB-Coma), 27 GHB users who never had a GHB-induced coma (GHB-NoComa) and 27 polydrug users who never used GHB (No-GHB). Participants completed self-report questionnaires to assess negative affect, and performed an emotional face matching task during func-