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Data in brief





Data Article

Data regarding the effect of cannabis consumption on liver function in the prospective PAFIP cohort of first episode psychosis



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ABSTRACT

The presented article describes data from secondary analyses, related to the research article entitled "Cannabis consumption and Non-Alcoholic Fatty Liver Disease. A three years longitudinal study in first episode non-affective psychosis patients" [1]. We present detailed data regarding the socio-demographic and baseline clinical characteristics of a sample of 390 drug-naïve patients with a first episode of non-affective psychosis, and the differences between cannabis users and non-users in those characteristics. Tables also show the results from cross-sectional and longitudinal statistical analyses exploring the relation between cannabis

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Antipsychotic treatment Mental health consumption and liver function, after excluding those patients with hazardous alcohol drinking.

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Specifications Table

Psychiatry; Hepatology Subject area More specific subject area First episode psychosis; NAFLD Type of data Tables, text file How data was acquired Prospective cohort study, including clinical evaluation. Data format Raw and Analyzed Experimental factors Effect of cannabis consumption on the incidence of liver steatosis (NAFLD). Experimental features Patients diagnosed with a first episode of non-affective psychosis, and being drug-naïves. were included in a prospective cohort (PAFIP). Patients were treated with antipsychotic medication. Clinical, cognitive and physical examinations were carried out prospectively during the 3-years follow-up period. Data source location Autonomous region of Cantabria, Spain. Data available at the following public data repository: Data accessibility Repository name: Mendeley Data Data identification: Crespo Facorro, Benedicto; Vázquez-Bourgon, Javier (2019), "Cannabis use and liver steatosis", Mendeley Data, v1. Direct URL to data: https://doi.org/10.17632/hwn48wt7j6.1 Related research article Vázquez-Bourgon J, Ortiz-García de la Foz V, Suarez-Pereira I, Iruzubieta P, Arias-Loste MT, Setién-Suero E, Ayesa-Arriola R, Gómez-Revuelta M, Crespo J, Crespo Facorro B. Cannabis consumption and Non-Alcoholic Fatty Liver Disease. A three years longitudinal study in first episode non-affective psychosis patients. Progress in Neuro-Psychopharmacology & Biological Psychiatry. 95 (2019) 109677. https://doi.org/10.1016/j.pnpbp.2019.109677.

Value of the Data

- Cannabis consumption is associated with key clinical and sociodemographic characteristics of the psychosis, such as age of
 onset, DUP or symptoms severity.
- After ruling out a probable confounding effect of alcohol drinking, cannabis was associated with a smaller risk of NAFLD in the first 3 years after psychosis breakout.
- · Cannabis effect on liver tissue might be through the modulation of weight gain.

1. Data

We present in this article data derived from secondary analyses of a previous study on the relation between cannabis consumption and NAFLD in a Spanish cohort of drug-naïve patients with a first episode of non-affective psychosis [1]. Raw data has been made accessible through the public data repository "Mendeley Data" at https://doi.org/10.17632/hwn48wt7j6.1.

Table 1 describes some of the main clinical and socio-demographic characteristics of the global study sample and of each cannabis groups (consumers and no consumers).

Table 2 describes the baseline and 3-years liver function tests differences between groups (cannabis users vs non users), after having excluded those patients with a moderate-severe alcohol consumption.

Table 3 shows the longitudinal differences in liver function tests between groups (cannabis users, discontinuers, non-users), after having excluded those patients with a moderate-severe alcohol consumption.

Table 1Baseline sociodemographic and clinical characteristics.

	Cannabis users	No cannabis users	Total	Stats ^a		
	Mean (SD)	Mean (SD)	Mean (SD)	df	F	P
Age at admission, years	25.2 (6.0)	33.7 (9.9)	30.4 (9.5)	1; 389	90.59	< 0.001
DUP, months	7.3 (10.3)	16.3 (36.7)	12.8 (29.7)	1; 386	8.56	0.004
DUI, months	18.1 (21.49)	26.3 (45.25)	23.98 (37.86)	1; 376	4.22	0.041
SANS-SAPS at inclusion	21.4 (7.6)	19.6 (7.6)	20.3 (7.7)	1; 388	5.24	0.023
Initial antipsychotic doses ^b	215.3 (86.4)	206.0 (82.8)	209.6 (84.2)	1; 389	1.13	0.289
	% (N)	% (N)	% (N)	N	X^2	P
Sex, males	80.0 (120)	40.4 (97)	55.6 (217)	390	58.60	< 0.001
Education level, secondary or lower	58.7 (88)	37.5 (90)	45.6 (178)	390	16.67	< 0.001
Family socioeconomic status, Not/Low qualified	52.0 (78)	49.8 (119)	50.6 (197)	389	0.18	0.375
Unmarried	88.0 (132)	65.0 (156)	73.8 (288)	390	25.28	< 0.001
Living with family	72.7 (109)	74.2 (178)	73.6 (287)	390	0.11	0.416
Student						
Unemployed	45.3 (68)	39.2 (94)	41.5 (162)	390	1.45	0.136
Diagnosis, schizophrenia	54.5 (81)	50.8 (121)	52.2 (202)	387	4.70	0.453
Hospitalization at inclusion	72.0 (108)	65.3 (156)	67.9 (264)	389	1.91	0.101
Drug consumption						
Tobacco smoking, yes	88.7 (133)	37.5 (90)	57.2 (223)	390	98.70	< 0.001
Alcohol consumption, yes	85.2 (127)	29.3 (70)	50.8 (197)	388	114.94	< 0.001
Hazardous alcohol consumption, yes ^c	4.9 (19)	2.8 (11)	7.7 (30)	388	0.152	0.435
Concomitant treatments						
Anticholinergics, baseline	4.7 (7)	3.3 (8)	3.9 (15)	389	0.462	0.336
Hypnotics, baseline	33.6 (50)	28.9 (69)	30.7 (119)	388	0.95	0.195
Benzodiazepines, baseline	66.4 (99)	58.2 (139)	61.3 (238)	388	2.66	0.064
Antidepressants, baseline	1.3 (2)	1.7 (4)	1.5 (6)	388	0.07	0.578
Mood stabilizers, baseline	0 (0)	0.4(1)	0.3 (1)	387	0.63	0.615

^a Statistical analyses: Un-adjusted analysis of variance (ANOVA) for continuous variables and chi-square test for categorical variables. Abbreviations: DUP: Duration of untreated psychosis. DUI: Duration of untreated illness. SANS: Scale for the Assessment of Negative Symptoms. SAPS: Scale for the Assessment of Positive Symptoms.

And Table 4, presents the clinical impact of cannabis use over the 3 years period, again after having excluded those patients with a moderate-severe alcohol consumption.

2. Experimental design, materials and methods

2.1. Population description

To obtain the present data, we included adult patients presenting a first episode of non-affective psychosis between 2001 and 2015 (full description of inclusion criteria in Pelayo et al., 2008), for whom we had information of cannabis use (yes/no) both at baseline and at 3-years follow-up [2]. Patients were evaluated at baseline and periodically thereafter until year 3. Anthropometric measures and fasting blood samples for lipid, glycemic and liver determinations, were collected. Main NAFLD and liver fibrosis scores (FLI, FIB-4 and NAFLD fibrosis scores) were calculated accordingly to previous literature [3—5]. Cannabis and other drugs were recorded from patients self-reports.

Table 1 shows the main clinical and socio-demographic characteristics of the study sample, and of each consuming groups (cannabis users and no users), at study entry. It also contains the results from the statistical analyses comparing these two groups regarding their clinical and sociodemographic characteristics. Patients reporting cannabis consumption were significantly younger than the nonconsumers. They also presented a shorter duration of untreated psychosis, and more severe psychotic symptomatology at study entry. More patients among the cannabis group reported smoking tobacco and drinking alcohol than in the no-cannabis group.

^b Equivalent doses of antipsychotic medication following Gardner et al., 2010 criteria.

^c Alcohol consumption thresholds for the diagnosis of NAFLD: 140 and 210 g of alcohol per week in women and men, respectively (Leoni et al., 2018).

Table 2Baseline and 3-years liver function tests in first episode psychosis, excluding patients with severe alcohol consumption.

	Baseline				3-years						
	Cannabis users Mean (SE)	No cannabis users	Stats ^a			Cannabis users	No cannabis users	Stats ^a			
		Mean (SE)	Mean (SE)	df	F	P	Mean (SE)	Mean (SE)	df	F	P
FLI algorithm factors											
BMI (kg/m ²)	21.9 (0.4)	24.0 (0.3)	1; 342	12.585	< 0.001	24.1 (0.9)	27.3 (0.3)	1; 334	10.049	0.002	
Waist circumference (cm)	81.6 (1.7)	84.3 (1.1)	1; 180	1.346	0.247	78.7 (3.3)	91.2 (0.9)	1; 207	12.912	< 0.001	
Triglycerides	83.5 (4.5)	79.3 (2.9)	1; 282	0.512	0.475	85.3 (11.9)	110.9 (3.5)	1; 333	4.158	0.042	
Liver laboratory tests											
AST	23.5 (2.1)	27.8 (1.3)	1; 313	2.375	0.124	24.3 (1.9)	24.4 (0.6)	1; 334	0.001	0.973	
ALT	21.8 (2.9)	28.9 (1.9)	1; 335	3.205	0.074	26.3 (3.9)	29.9 (1.2)	1; 335	0.753	0.386	
GGT	29.2 (4.9)	15.7 (3.2)	1; 316	4.276	0.039	26.1 (9.4)	27.0 (2.8)	1; 334	0.008	0.930	
AP	83.3 (9.6)	92.0 (7.2)	1; 123	0.417	0.520	66.2 (5.5)	65.6 (1.9)	1; 125	0.010	0.920	
Bilirubin	0.71 (0.09)	0.80 (0.07)	1; 103	0.513	0.476	0.68 (0.09)	0.59 (0.03)	1; 122	0.863	0.355	
Albumin	4.54 (0.04)	4.54 (0.03)	1; 305	0.006	0.939	4.54 (0.05)	4.53 (0.01)	1; 322	0.039	0.844	
Other laboratory tests											
Platelets	250.4 (7.9)	249.6 (5.1)	1; 267	0.005	0.941	252.5 (12.5)	243.7 (3.9)	1; 321	0.437	0.509	
Leptin	6.6 (1.1)	9.5 (0.7)	1; 264	3.680	0.056	10.0 (2.2)	14.8 (0.6)	1; 324	4.274	0.040	
hsCRP	0.17 (0.09)	0.16 (0.06)	1; 154	0.009	0.926	0.14 (0.12)	0.29 (0.03)	1; 201	1.495	0.223	
Hepatic disease indexes											
FLI	15.8 (3.5)	18.8 (2.2)	1; 153	0.432	0.512	7.4 (7.9)	38.6 (2.1)	1; 202	14.169	< 0.001	
FIB-4 score	0.69 (0.05)	0.68 (0.03)	1; 250	0.031	0.876	0.73 (0.05)	0.69 (0.01)	1; 318	0.738	0.391	
NAFDL score	-3.54(0.14)	-3.41 (0.09)	1; 242	0.508	0.477	-3.35 (0.19)	-3.05 (0.06)	1; 311	2.277	0.132	

Abbreviations: FLI, fatty liver Index; BMI, body mass index; GGT, Gamma-glutamyltransferase; AST, aspartate aminotransferase; ALT, alanine aminotransferase; AP, alkaline phosphatase; hsCRP, high sensitivity C-reactive protein; FIB-4, fibrosis 4 score; NAFLD, non-alcoholic fatty liver disease fibrosis score.

^a ANCOVA model: parameter was used as the dependent variable, cannabis use was the fixed factor and age, sex, and tobacco and alcohol consumption use were used as covariates.

 Table 3

 Longitudinal differences in liver function tests, after 3 years of antipsychotic treatment, excluding patients with severe alcohol consumption.

	Cannabis users	Discontinuers	Non-users	Statistics ^a		
	Mean diff (SE)	Mean diff (SE)	Mean diff (SE)	df	F	P
FLI algorithm factors						
BMI (kg/m ²)	3.0 (0.7)	4.5 (0.4)	3.6 (0.2)	2; 331	2.791	0.063
Waist circumference (cm)	2.4 (2.8)	7.3 (1.8)	7.2 (1.0)	2; 171	1.336	0.266
Triglycerides	7.8 (12.2)	27.0 (7.5)	35.7 (4.4)	2; 272	2.118	0.122
Other liver laboratory tests						
AST	0.89 (4.03)	-0.91 (2.56)	-3.34 (1.45)	2; 303	0.522	0.594
ALT	6.5 (5.7)	5.7 (3.5)	1.7 (2.1)	2; 325	0.499	0.608
GGT	7.3 (6.6)	7.5 (4.2)	7.2 (2.5)	2; 306	0.002	0.998
AP	-5.4 (16.3)	-18.2 (10.3)	-30.4(7.2)(6.5)	2; 121	0.902	0.409
Bilirubin	-0.26(0.11)	-0.03(0.08)	-0.22 (0.05)	2; 98	2.556	0.083
Albumin	0.001 (0.09)	-0.020(0.05)	-0.028(0.03)	2; 259	0.040	0.961
Other laboratory tests						
Platelets	-10.3 (11.9)	1.2 (7.1)	-13.4 (4.2)	2; 254	1.473	0.231
Leptin	4.6 (2.7)	7.1 (1.6)	5.4 (0.9)	2; 247	0.563	0.570
hsCRP	-0.005(0.22)	0.060 (0.12)	0.102 (0.07)	2; 145	0.115	0.892
Hepatic disease indexes						
FLI	-3.7 (6.6)	19.6 (4.2)	19.8 (2.3)	2; 144	5.826	0.004
FIB-4 score	0.022 (0.09)	-0.035 (0.05)	0.047 (0.03)	2; 237	0.803	0.449
NAFDL score	0.21 (0.23)	0.38 (0.15)	0.58 (0.08)	2; 224	1.274	0.282

Abbreviations: FLI, fatty liver Index; BMI, body mass index; GGT, Gamma-glutamyltransferase; AST, aspartate aminotransferase; ALT, alanine aminotransferase; AP, alkaline phosphatase; hsCRP, high sensitivity C-reactive protein; FIB-4, fibrosis 4 score; NAFLD, non-alcoholic fatty liver disease fibrosis score.

^a ANCOVA model: mean differences after 3 years of treatment were used as dependent variables, evolution of cannabis use was the fixed factor and age, sex, and tobacco and alcohol use trajectories use were applied as covariates.

 Table 4

 Comparison of proportion of subjects with pathological liver functions tests, at baseline and at 3-years in each cannabis consumption group, excluding patients with severe alcohol consumption.

	3 year follow-up	Baseline	% difference	N	p ^a
	% (n)	% (n)			
AST, > 35 UI/L					
Continuer	6.9 (2)	13.8 (4)	-6.9	29	0.687
Discontinuers	9.7 (7)	15.3 (11)	-5.6	72	0.424
Non-users	7.8 (16)	14.6 (30)	-6.8	206	0.044
Total	8.1 (25)	14.7 (45)	-6.6	307	0.015
ALT, > 40 UI/L					
Continuer	16.7 (5)	10.0 (3)	6.7	30	0.625
Discontinuers	22.0 (18)	8.5 (7)	13.5	82	0.019
Non-users	16.6 (36)	12.4 (27)	4.2	217	0.253
Total	17.9 (59)	11.2 (37)	6.7	329	0.013
GGT, > 32 UI/L	` '	` ,			
Continuer	10.0 (3)	0	10.0	30	_
Discontinuers	23.0 (17)	6.8 (5)	16.2	74	0.002
Non-users	19.9 (41)	10.7 (22)	9.2	206	0.001
Total	19.7 (61)	8.7 (27)	11.0	310	< 0.001
Leptin, > 10 ng/ml	` '	` '			
Continuer	10.0 (2)	0	10.0	20	_
Discontinuers	42.6 (26)	13.1 (8)	29.5	61	< 0.001
Non-users	64.5 (109)	36.1 (61)	28.4	169	< 0.001
Total	54.8 (137)	27.6 (69)	27.2	250	< 0.001
hsCRP, > 0.3 ng/dL	, ,	, ,			
Continuer	0	11.1 (1)	-11.1	9	_
Discontinuers	20.5 (8)	7.7 (3)	12.8	39	0.125
Non-users	24.2 (24)	7.1 (7)	17.3	99	0.001
Total	21.8 (32)	7.5 (11)	14.3	147	< 0.001
FLI, \geq 60	, ,	• •			
Continuer	0	0	0	12	_
Discontinuers	28.1 (9)	6.2 (2)	21.9	32	0.022
Non-users	25.0 (25)	9.0 (9)	16.0	100	< 0.001
Total	23.6 (34)	7.6 (11)	16.0	144	< 0.001

Abbreviations: FLI, fatty liver Index; GGT, Gamma-glutamyltransferase; AST, aspartate aminotransferase; ALT, alanine aminotransferase; hsCRP, high sensitivity C-reactive protein.

^a McNemar test for repeated measures.

2.2. Secondary analyses excluding patients with moderate-severe alcohol consumption

Due to the well-known deleterious effect of alcohol on liver, and despite being one of the study's exclusion criteria presenting an alcohol dependence, we considered appropriate carrying secondary analyses after exclusion of those patients with alcohol consumption qualifying for moderate-severe drinking. For this, moderate-severe alcohol use was defined using the accepted alcohol consumption thresholds for the diagnosis of NAFLD: 140 and 210 g of alcohol per week in women and men, respectively [6]. Tables 2-4 contains the results from the statistical analyses, both cross-sectional and longitudinal, after excluding these patients (n=40).

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Conflict of Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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