(measured by Clinical Global Impression objective and subjective form) were assessed.

Results One hundred and four patients suffering from schizophrenia ($n=67$), schizoaffective disorder ($n=30$), polymorphic psychotic disorder ($n=3$), schizotypal disorder ($n=2$) and delusional disorder ($n=2$) were included in the study. The results showed that there was a high positive correlation between negative coping and self-stigma, and the negative correlation between positive strategies and the overall score of self-stigma. Stepwise regression analysis showed that negative coping (especially resignation), subjective severity SubjCGI and positive coping strategies (especially positive self-instruction) explains 52.8% of the overall score variance of self-stigma (Tables 1–3).

Conclusions This study revealed that there is a connection between self-stigma and coping strategies in patients suffering from schizophrenia spectrum disorders.

Table 1 Description of the sample, demographic and clinic at data.

VARIABLE	MEAN AND STANDARD DEVIATION
Age	42.19 ± 10.09
Gender (M: F)	41:63
Age of the disease onset	26.06 ± 8.95
Lifetime duration of treatment Minimum Maximum	15.67 ± 9.57 1 45
Number of hospitalizations	4.17 ± 4.03
Psychiatric heredity	
Same disorder	15 (14.4 %)
Other disorder	39 (37.5 %)
Without	48 (46.2 %)
Education:	
elementary	10 (9.6 %)
vocational training	26 (25.0 %)
secondary school	51 (49.0 %)
university	16 (15.5 %)
Marital Status:	
single	61 (58.7 %)
married	24 (23.1 %)
divorced	16 (15.4 %)
widowed	1 (2.8 %)
Employment Yes/No	33/71
Retirement	88
Full invalidity	61
Partial invalidity	20
Old-age	7
From parent family	66
From incomplete family	31
Brother/sister Yes/No	91/13
Birth order	
First-born	44
Second-born	37
Third-born	10
Using psychiatric medication Yes/No	102/2
Regular use	94
Regularly, more than prescribed amount	2
Irregularly use	7
ObjCGI severity	4.12 ± 0.95
SubjCGI severity	2.76 ± 1.39

Table 2 Description of using coping strategies and self-stigma in outpatients.

COPING STRATEGIES	T-score mean	Self-stigma ISMI	Mean and sd
Underestimation	47.77 ± 12.87	**Alienation**	13.40 ± 3.86
Guilt denial	54.35 ± 12.2	**Stereotype agreement**	14.06 ± 3.37
Diversion	50.88 ± 9.88	**Perceived discrimination**	11.17 ± 3.25
Compensatory satisfaction	55.57 ± 10.2	**Social withdrawal**	13.11 ± 3.69
Situation control	44.95 ± 11.08	**Stigma resistance**	12.67 ± 2.36
Reaction control	47.76 ± 10.8	**Overall score**	64.30 ± 13.49
Positive self-instruction	41.37 ± 11.95		
Need for social support	50.98 ± 11.02		
Active avoidance	55.76 ± 8.9		
Escape tendency	**61.82 ± 9.42**		
Perseveration	49.9 ± 12.5		
Resignation	**60.44 ± 10.95**		
Self-accusation	53.29 ± 12.61		
Using negative coping	**59.04 ± 11.24**		
Using positive coping	49.5 ± 11.8		

Abbreviations: Average use of coping 40-60 T-score, more than 60 overusing, less than 40 reduced using

Table 3 Correlations between self-stigma and coping strategies.

Coping / Subscore	Whole score	Alienation	Stereotype agreement	Perceived discrimination	Social withdrawal	Stigma resistance
Underestimation	-0.424***	-0.397***	-0.300**	-0.282**	-0.459***	-0.219*
Guilt denial	-0.256**	-0.149	-0.317**	-0.152	-0.226*	-0.261**
Diversion	-0.365***	-0.310**	-0.336**	-0.254*	-0.276**	-0.363***
Compensatory satisfaction	-0.223*	-0.089	-0.233*	-0.132	-0.165	-0.294**
Situation control	-0.219*	-0.202*	-0.218*	-0.103	-0.133	-0.263**
Reaction control	-0.377***	-0.337***	-0.385***	-0.313**	-0.300**	-0.265**
Positive self-instruction	-0.555***	-0.464***	-0.521***	-0.322**	-0.447***	-0.468***
Need for social support	0.121	0.192	0.047	0.154	0.097	0.070
Active avoidance	-0.019	0.047	-0.138	-0.059	0.033	-0.039
Escape tendency	0.434***	0.428***	0.271**	0.236*	0.375***	0.303**
Perseveration	0.436***	0.504***	0.281*	0.345***	0.456***	0.148
Resignation	0.637***	0.631***	0.485***	0.388***	0.570***	0.403***
Self-accusation	0.454***	0.494***	0.381***	0.266***	0.417***	0.194*
Negative coping	0.598***	0.632***	0.412***	0.386***	0.570***	0.280**
Positive coping	-0.491***	-0.399***	-0.464***	-0.315***	-0.406***	-0.431***

Abbreviations: Pearson´s correlation, * p<0.05; ** p<0.01; *** p<0.001

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FC73

Lifetime antipsychotic use and brain structures in schizophrenia and other psychoses – 43-year study of the Northern Finland Birth Cohort 1966

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Introduction The effects of long-term antipsychotic medication use on structural brain changes in psychoses are still unknown. Severity and duration of illness are key confounders when evaluating antipsychotic effects on brain morphology.
Objectives Understanding the role of antipsychotic medication on brain morphology in psychoses.
Aims To analyze whether cumulative lifetime or current antipsychotic medication dose relates to brain morphology in schizophrenia and other psychoses at age of 43 years.
Methods Forty-four schizophrenia cases and 35 with other psychoses from the Northern Finland Birth Cohort 1966 were scanned on a 1.5T GE Signa scanner and brain structures were extracted using volBrain automated volumetry system (http://volbrain.upv.es). Data of antipsychotic medication were collected from medical records and interviews. We used linear regression model to analyze the effect of antipsychotic medication on brain volumes and used intracranial volume and onset age as covariates. We also performed additional analyses adding psychotic symptoms (PANSS Total score) as a covariate.
Results Higher lifetime and current dose associated to left lateral ventricle increase ($b = 0.33$, $P = 0.033$; $b = 0.307$, $P = 0.042$, respectively) and right and left accumbens decrease ($b = -0.405$, $P = 0.013$, $b = -0.404$, $P = 0.010$; $b = -0.302$, $P = 0.027$, $b = -0.282$, $P = 0.036$, respectively) in schizophrenia but not in other psychoses. When PANSS was added to the model, the findings remained regarding right and left accumbens, but not regarding left lateral ventricle.
Conclusions It seems that antipsychotic medication affects the brain in schizophrenia, but not in the heterogeneous group of other psychoses. In schizophrenia, brain changes associated to antipsychotic medication cannot be explained by illness duration or symptom severity.
Disclosure of interest The authors have not supplied their declaration of competing interest.

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FC74

Association between drug-induced hyperprolactinemia related adverse events on women schizophrenia patients with DRD2 Taq1 polymorphism

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Objectives To observe the association between adverse effects of long-term use of antipsychotic drugs in female schizophrenic patients and dopamine D2 receptor (DRD2), cytochrome P450 (CYP) 2D6, *estrogen receptor-α* gene (ESR1).
Method The subjects were 89 female schizophrenic patients (age range from 18 to 40) who had been taking the same medication for more than 3 months. The adverse effects with regard to hyperprolactinemia were studied through the blood collection at one point of the subjects. Furthermore, the effect of DRD2, CYP2D6, ESR1 on serum prolactin level and amenorrhea was analyzed.
Results There was a lower concentration of E2 in patients with amenorrhea. In addition, an inverse correlation was found between prolactin level and E2 level. Hyperprolactinemia (HPRL) was commonly found in patients who had been using risperidone, amisulpride and paliperidone; in contrast, HPRL was found less in those who had been taking aripiprazole, olanzapine, ziprasidone, clozapine and quetiapine. Moreover, female schizophrenic patients who had DRD2 Taq1 A1 allele had twice the chance of developing amenorrhea than those who did not have A1 allele. Female schizophrenic patients who had Taq1 A1 allele also had 48% higher concentration level of prolactin than those who did not have A1 allele. There was no association found between prolactin and CYP2D6 or ESR1.
Conclusion Female schizophrenic patients who had DRD2 Taq1 A1 allele showed high prolactin level and high-frequency of HPRL. Therefore, reducing the use of prolactin-elevating antipsychotics for female schizophrenic patients with DRD2 Taq1 A1 allele would be one method minimizing the adverse effects of drug-induced hyperprolactinemia.
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FC75

Affectivity during social behaviour in a schizophrenic-like rat

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Introduction Rats are social animals that produce high-frequency whistles said to reflect their underlying affective state. Injecting rats with a glutamate agonist (domoic acid) at a sensitive period of brain development, models aspects of schizophrenia. This is known as the neonatal DOM model.
Aims We investigated whether DOM rats display altered social behaviour – as seen in patients with schizophrenia – using their high-frequency whistles as a proxy for the emotional valence of social situations.
Methods We used 19 male Sprague Dawley rats, injected with either a low-dose of domoic acid or saline at postnatal days 8 to 14. The social behaviour of the rats was investigated at four levels:
– anticipation of social interaction;
– dyadic encounter;
– three-chamber test;
– tickling.
Tests were carried out at postnatal days 34 to 40 and 50 to 56. Rat whistles were recorded on all days of testing.
Results In progress.
Conclusions The interest in rat whistles as a supplement to traditional behavioural tests has increased. New software allows for detailed qualitative analysis of the whistle subtypes and thus new complexity to their interpretation. This study can help unravel information encoded in the whistles and shed light on the social behaviour of the DOM rat thus investigating it is applicability as a model of schizophrenia.