ual subject seed-to-voxel connectivity maps, to the corresponding seeds of the default mode network.

Results Fig. 1.

Conclusions Our results show a significant increase in connectivity between LDLPC and anterior prefrontal cortex, dorsolateral prefrontal cortex and somatosensory association areas, especially between patients and controls. It is noteworthy to mention that we found a significant decrease in connectivity between LDLPC and supramarginal gyrus, superior temporal gyrus and somatosensory association areas between unaffected relatives and controls.

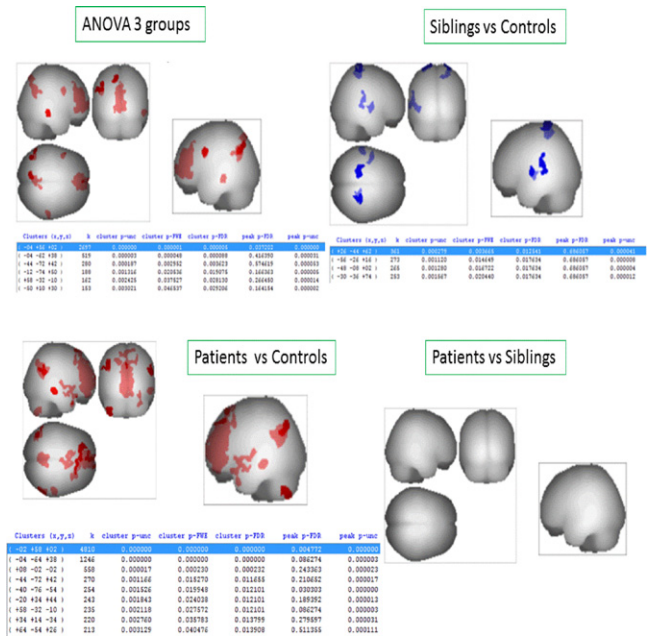

Fig. 1

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

Acknowledgments L. Galindo is funded by the fellowship Río Hortega Spanish government ISCIII (CM14/00111).

http://dx.doi.org/10.1016/j.eurpsy.2016.01.074

FC71

An interventional, multi-center, randomized, double-blind, placebo-controlled, active reference, flexible dose study of brexpiprazole in adults with acute schizophrenia

S.R. Marder[1], M. Hakala[2,*], M. Gislum[3], A. Skuban[4], E. Weiller[5], C. Weiss[6]

[1] *Desert Pacific Mental Illness Research, Education and Clinical Center, Semel Institute for Neuroscience at UCLA, Department of Psychiatry and Biobehavioral Sciences, Los Angeles, USA*

[2] *Lundbeck A/S, ICR Psychiatry, DK, Valby, Denmark*

[3] *H. Lundbeck A/S, Department of Biostatistics, Valby, Denmark*

[4] *Otsuka Pharmaceutical Commercialization and Development Inc., Global Clinical Development, Princeton, USA*

[5] *H. Lundbeck A/S, Medical Affairs, Valby, Denmark*

[6] *Otsuka Pharmaceutical Commercialization and Development Inc., Global Medical Affairs, Princeton, USA*

* *Corresponding author.*

Introduction Brexpiprazole is a serotonin-dopamine activity modulator that is a partial agonist at 5-HT_{1A} and dopamine D_2 receptors at similar potency, and an antagonist at 5-HT_{2A} and noradrenaline alpha$_{1B/2C}$ receptors.

Objectives Evaluating the efficacy, safety, and tolerability of flexible doses of brexpiprazole compared with placebo in patients with acute schizophrenia.

Aim Primary endpoint was change from baseline to week 6 in PANSS total score and key secondary endpoint was change from baseline to week 6 in CGI-S score.

Methods Phase 3, multi-center, randomized, double-blind, placebo-controlled, active reference, trial (NCT01810380). Hospitalized patients were randomized to brexpiprazole (2 to 4 mg/day), placebo, or quetiapine extended release (400 to 800 mg/day) for 6 weeks. Quetiapine was included as an active reference. Changes from baseline were analyzed using an MMRM approach.

Results Mean change in PANSS total score was −20.0 and −15.9 in the brexpiprazole ($n = 150$) and placebo ($n = 159$) groups, respectively ($P = 0.056$). Sensitivity analyses suggested treatment effect (e.g., ANCOVA, LOCF: $P = 0.025$; ANCOVA, OC: $P = 0.026$). Mean change in PANSS total score (−24.0) with quetiapine ($n = 150$) was significantly greater than that with placebo ($P < 0.001$), demonstrating sensitivity of the assay. Brexpiprazole separated from placebo on the mean change in CGI-S score (−1.2 vs. −0.9, $P = 0.014$). The proportion of patients reporting TEAEs were similar between the brexpiprazole and placebo treatment groups (54% versus 54.7%).

Conclusion Treatment with brexpiprazole showed a clinically meaningful improvement in patients with acute schizophrenia. While the difference between brexpiprazole and placebo only approached statistical significance, sensitivity analyses and secondary endpoints supported a treatment effect of brexpiprazole.

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

http://dx.doi.org/10.1016/j.eurpsy.2016.01.075

FC72

Are self-stigma and coping strategies interrelated in outpatients with schizophrenia spectrum disorders using the psychiatric medication? Cross-sectional study

M. Holubova[1,2,*], J. Prasko[1], R. Hruby[3], K. Latalova[1], M. Slepecky[4], M. Marackova[1], D. Kamaradova[1], T. Gubova[2]

[1] *Faculty of Medicine and Dentistry, Palacky University Olomouc, University Hospital Olomouc, Department of Psychiatry, Olomouc, Czech Republic*

[2] *Regional Hospital Liberec, Department of Psychiatry, Liberec, Czech Republic*

[3] *Psychiatric Outpatient Department, Psychiatric Outpatient Department, Martin, Slovakia*

[4] *Faculty of Social Science and Health Care, Constantine the Philosopher University in Nitra, Department of Psychology Sciences, Nitra, Slovakia*

* *Corresponding author.*

Introduction Self-stigma is the maladaptive psychosocial phenomenon that can affect the patient's self-image, may lead to dysphoria, social isolation, reduced adherence and quality of life. Maladaptive coping strategies may adversely disturb the overall functioning of psychiatric patients.

Objectives Thinking about coping strategies and self-stigma in practice may play a significant role in understanding patients with schizophrenia spectrum disorders, especially for mental health professionals. Focus on coping strategies could be a useful concept in supportive and educational therapy to help patients in using more adaptive coping strategies and decrease their self-stigma.

Aims The aim of this study was to determine the relation between coping strategies and the self-stigma among outpatients with schizophrenia and related disorders.

Methods Stress Coping Style Questionnaire (SVF-78), Internalized Stigma of Mental Illness (ISMI) and severity of the disorder

(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

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

http://dx.doi.org/10.1016/j.eurpsy.2016.01.076

FC73

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

S. Huhtaniska[1,2,3,*], I. Korkala[1,2], T. Heikka[1], J. Tohka[4], J. Manjon[5], P. Coupe[6], J. Remes[7], J. Moilanen[3,8], V. Kiviniemi[7], L. Björnholm[1], M. Isohanni[1,8], J. Veijola[1,3,8], G. Murray[9,10], E. Jääskeläinen[1,2,3,8], J. Miettunen[1,2,3,8]

[1] *University of Oulu, Institute of Clinical Medicine, Research Unit for Clinical Neurosciences, Oulu, Finland*

[2] *University of Oulu, Center for Life Course Epidemiology and Systems Medicine, Oulu, Finland*

[3] *Oulu University Hospital and University of Oulu, Medical Research Center Oulu, Oulu, Finland*

[4] *Universidad Carlos III de Madrid, Department of Bioengineering and Aerospace Engineering, Madrid, Spain*