suggests that HE is an autoimmune disorder instead of thyroid disease.

Disclosure of interest The author has not supplied his/her declaration of competing interest.

http://dx.doi.org/10.1016/j.eurpsy.2017.01.1547

EV1218

Brain metabolic abnormalities in schizophrenia patients

M. Amorim[1,*], A. Moreira[1], A. Marques[2], T. Summavielle[3]
[1] *School of Allied Health Technologies of the Polytechnic Institute of Porto, Clinical Analysis and Public Health, Vila Nova de Gaia, Portugal*
[2] *School of Allied Health Technologies of the Polytechnic Institute of Porto, LabRP, Vila Nova de Gaia, Portugal*
[3] *I3S consortium, University of Porto, Addiction Biology Group, Porto, Portugal*
[*] *Corresponding author.*

Introduction Main schizophrenia symptoms result from abnormalities in brain function, such as hypofrontality and structural deficits on the prefrontal-thalamic-cerebellar circuit, as shown in brain imaging studies in first-episode SCZ patients. Whether metabolic alterations may be underlying these events is being studied thoroughly.

Objectives/aims To assess brain metabolic disturbances in first episode and/or drug-naïve SCZ patients.

Methods We conducted a literature review through Pubmed search for MeSH: schizophrenia, metabolism, glucose, insulin, brain. Controlled studies on first episode and/or drug-naïve SCZ patients were included.

Results Lower metabolic activity in the frontal regions of the brain is associated to an increase in norepinephrine transmission and decrease in dopaminergic transmission with reduced dopamine efflux in the frontal cortex. This seems to lead to cellular changes resulting in resulting lower blood flow and glucose demand. Molecular analysis of postmortem SCZ patients' brains has indicated alterations in glucose metabolism and insulin signalling pathways, showing evidence for prefrontal cortex decreased expression of glucose metabolism, namely glycolytic enzymes such as glyceraldehyde 3-phosphate dehydrogenase, hexokinase, phosphoglycerate mutase, enolase and pyruvate kinase and decreased levels and phosphorylation of the insulin receptor and insulin signalling proteins AKT1 and GSK3β. Significantly elevated glucose concentrations in cerebrospinal fluid were observed in SCZ patients, but with no serum levels differences. A SCZ brain specific increased glucose could be explained by preferential utilization of lactate, predominantly produced by astrocytes, over glucose as an energy substrate.

Conclusions Abnormalities in brain glucose metabolism and insulin signalling seem to appear in early stages of SCZ, suggesting a role in SCZ onset and pathophysiology.

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

http://dx.doi.org/10.1016/j.eurpsy.2017.01.1548

EV1219

Peripheric metabolic abnormalities in schizophrenia patients

M. Amorim[1,*], A. Moreira[1], A. Marques[2], T. Summavielle[3]
[1] *School of Allied Health Technologies of the Polytechnic Institute of Porto, Clinical Analysis and Public Health, Vila Nova de Gaia, Portugal*
[2] *School of Allied Health Technologies of the Polytechnic Institute of Porto, LabRP, Vila Nova de Gaia, Portugal*
[3] *i3S consortium, University of Porto, Addiction Biology Group, Porto, Portugal*
[*] *Corresponding author.*

Introduction Schizophrenia (SCZ) is frequently associated with metabolic symptoms including dyslipidaemia, hyperinsulinemia, type 2 diabetes and obesity. In fact, SCZ patients have been reported to present higher prevalence of these conditions than general population, commonly associated to second generation antipsychotic therapy. Recent studies, however, have demonstrated that peripheral metabolic disturbances can appear at disease onset or drug-naïve patients.

Objectives/aims To assess metabolic disturbances in first episode and/or drug-naïve SCZ patients.

Methods We conducted a literature review through Pubmed search for MeSH: schizophrenia, metabolism, glucose, insulin. Controlled studies on first episode and/or drug-naïve SCZ patients were included.

Results Several studies showed no change in SCZ patients' fasting blood glucose, while others found increased glucose levels and impaired glucose tolerance in SCZ patients compared to healthy controls in several recent studies. Hyperinsulinemia and insulin resistance have also been identified in antipsychotic-naïve SCZ patients and it has been suggested that early onset patients are more likely to present insulin resistance. In addition, there's evidence of increased circulating levels of chromogranin A, pancreatic polypeptide, prolactin, cortisol, progesterone, thus emphasising that multiple components of the hypothalamic-pituitary-adrenal-gonadal axis may be affected in SCZ. These elevations were associated to normal glycaemia suggesting there may be insulin intolerance during early stages of SCZ, requiring an increased secretion from pancreatic Bcells to maintain normal glucose levels.

Conclusions Recent studies of first onset and/or drug-free schizophrenia patients have shown impaired fasting glucose tolerance, hyperinsulinemia and insulin intolerance, suggesting that metabolic abnormalities may play a role in SCZ onset and pathophysiology.

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

http://dx.doi.org/10.1016/j.eurpsy.2017.01.1549

EV1220

Systemic review: High dose olanzapine treatment for treatment resistant schizophrenia

Z. Azvee[1,*], J. Lally[2]
[1] *Beaumont Hospital, Liaison Psychiatry, Dublin, Ireland*
[2] *Royal College of Surgeons Ireland, Psychiatry, Dublin, Ireland*
[*] *Corresponding author.*

Objectives Schizophrenia is a major mental illness with a progressive course. Thirty percent of cases of patients with schizophrenia do not respond to adequate trials of at least 2 different groups of antipsychotics,are currently classified as having treatment resistant schizophrenia (TRS). Clozapine remains the gold standard, treatment of choice for TRS. However, clozapine does not come without its own challenges. Its risk profile, particularly agranulocytosis, reported in 1% of cases, has led to the necessity of weekly blood counts within the first 18 weeks of treatment and subsequently every month with slow dose titration. Clinically, sedation, weight gain and hypersalivation may further hamper the compliance of patients. Non-compliance has been reported to cause rebound psychosis. Recent studies have raised questions as to which antipsychotic is most efficacious for TRS. Thus, we conducted a systematic review of high dose olanzapine treatment for people with TRS.

Method A systematic review of prospective studies found through search of PubMed, Scopus and hand-searched key papers which included randomized controlled trials and open-label studies which looked at high dose of olanzapine treatment response for TRS.

Results The study is currently ongoing and preliminary results will be presented at the conference in April 2017.
Conclusions The gravity of burden TRS brings to patients extends itself to their families, carers and clinicians. Further evidence on which antipsychotic is more efficacious for patients with TRS would have huge implications in terms of health benefits for the patients, better informed clinical decisions and also health economics in general.
Disclosure of interest The authors have not supplied their declaration of competing interest.

http://dx.doi.org/10.1016/j.eurpsy.2017.01.1550

EV1221

Systemic review: High dose olanzapine treatment for treatment resistant schizophrenia

Z. Azvee[1,*], J. Lally[2]
[1] *Beaumont Hospital, Liaison Psychiatry, Dublin, Ireland*
[2] *Royal College of Surgeons Ireland, Dublin, Ireland*
[*] *Corresponding author.*

Objectives Schizophrenia is a major mental illness with a progressive course. Thirty percent of cases of patients with schizophrenia do not respond to adequate trials of at least 2 different groups of antipsychotics are currently classified as having treatment resistant schizophrenia (TRS). Clozapine remains the gold standard, treatment of choice for TRS. However, clozapine does not come without its own challenges. Its risk profile, particularly agranulocytosis, reported in 1% of cases, has led to the necessity of weekly blood counts within the first 18 weeks of treatment and subsequently every month with slow dose titration. Clinically, sedation, weight gain and hypersalivation may further hamper the compliance of patients. Non-compliance has been reported to cause rebound psychosis. Recent studies have raised questions as to which antipsychotic is most efficacious for TRS. Thus, we conducted a systematic review of high dose olanzapine treatment for people with TRS.
Method A systematic review of prospective studies found through search of PubMed, Scopus and hand-searched key papers which included randomized controlled trials and open-label studies which looked at high dose of olanzapine treatment response for TRS.
Results The study is currently ongoing and preliminary results will be presented at the conference in April 2017.
Conclusions The gravity of burden TRS brings to patients extends itself to their families, carers and clinicians. Further evidence on which antipsychotic is more efficacious for patients with TRS would have huge implications in terms of health benefits for the patients, better informed clinical decisions and also health economics in general.
Disclosure of interest The authors have not supplied their declaration of competing interest.

http://dx.doi.org/10.1016/j.eurpsy.2017.01.1551

EV1222

The comprehensive Icf core set for schizophrenia from the perspective of psychiatrists: A content-validity study using the Delphi technique

L. Nuño[1], M. Barrios[2,*], E. Rojo[3], J. Gomez-Benito[2], G. Guilera[2]
[1] *Hospital Clínic de Barcelona, Department of Psychiatry and Clinical Psychology, Clinic Institute of Neurosciences, Barcelona, Spain*
[2] *University of Barcelona, Social Psychology and Quantitative Psychology, Barcelona, Spain*
[3] *Hospital Benito Menni CASM- Sisters Hospitallers, Department of Psychiatry- International University of Catalonia, Barcelona, Spain*
[*] *Corresponding author.*

Introduction Schizophrenia is a chronic mental illness associated with several functional impairments. There has been an increasing interest in the impact of schizophrenia on functioning. The development of the Comprehensive International Classification of Functioning, Disability and Health (ICF) Core Set for schizophrenia, a shortlist of 97 ICF categories that are relevant for describing functioning and disability of people living with schizophrenia, has derived from this interest.
Objectives This study aims to explore the content validity of this core set from the perspective of psychiatrists.
Methods In a 3-round Delphi survey, psychiatrists experienced in schizophrenia treatment were asked about patients' problems, resources and environmental factors they treat in patients with schizophrenia.
Results A total of 352 psychiatrists from 65 countries representing all six World Health Organization regions completed the first round questionnaire. The response rate at the third round was 86%. Answers were linked to 422 ICF categories. Of all these, 109 ICF categories reached consensus ($\geq$ 75% agreement) at the third round. Eighty-seven out of the 97 ICF categories that form the comprehensive ICF core set for schizophrenia were represented in this list. All the comprehensive ICF core set for schizophrenia categories reached consensus except five categories.
Conclusions The content validity of the comprehensive ICF core set for schizophrenia from the perspective of psychiatrists was largely supported. However, further research is needed including other health professionals (e.g., psychologists, nurses and occupational therapists) to further obtain new content validity evidences.
Disclosure of interest The authors have not supplied their declaration of competing interest.

http://dx.doi.org/10.1016/j.eurpsy.2017.01.1552

EV1223

Clinical and genetic predictors of the severity and activity of paranoid schizophrenia

Y. Barylnik[*], E. Kolesnichenko, Y. Abrosimova, S. Pakhomova, D. Samoylova, N. Filippova, E. Bachilo, A. Antonova, S. Sizov, T. Ledvanova, V. Kuryshev
Saratov State Medical University, Department of Psychiatry, Narcology, Psychotherapy and Clinical Psychology, Saratov, Russia
[*] *Corresponding author.*

Clinical symptoms, course and outcomes of paranoid schizophrenia are polymorphic. Reliable predictors of severity and activity of schizophrenic process could provide clinicians important prognostic information for adequate and timely implementation of therapeutic and rehabilitative measures. Overall, 206 patients with paranoid schizophrenia were examined. Clinical predictors were collected from hospital records and interviews. *BDNF* gene Val66Met polymorphism (rs6265 G>A), *DRD2* gene C939T polymorphism (rs6275C>T) and *5-HTR2A* gene T102C polymorphism (rs6313 T>C) were studied as potential markers of prognosis for paranoid schizophrenia. Results of research testify that the *DRD2* gene C939T polymorphism and *5-HTR2A* gene T102C polymorphism cannot be used as predictors of the severity and activity of paranoid schizophrenia. The MetMet genotype of *BDNF* gene Val66Met polymorphism can be used as marker of favorable prognosis for paranoid schizophrenia. Schizoid, epileptoid, psychasthenic and conformal accentuation of personality in the premorbid, early onset of psychosis, paranoid and hallucinatory-paranoid variants of onset predicted more expressed severity of paranoid schizophrenia. These prognostic factors can be taken into account in clinical practice.
Disclosure of interest The authors have not supplied their declaration of competing interest.

http://dx.doi.org/10.1016/j.eurpsy.2017.01.1553