permeability and stress-related psychiatric disorders. Front Cell Neurosci 2015;9.
[3] Mass M, Kubera M, Leunis JC. The gut-brain barrier in major depression: intestinal mucosal dysfunction with an increased translocation of LPS from gram negative enterobacteria (leaky gut) plays a role in the inflammatory pathophysiology of depression. Neuroendocrinol Lett 2008;29(1):117–24.

http://dx.doi.org/10.1016/j.eurpsy.2016.01.839

S24

Can the pathophysiology of autism be explained by the nature of the discovered urine peptides and dietary antigens?

K.L. Reichelt
Oslo University, Blindern, Oslo and lab 1, Sandvika, Norway

Purpose A: 1. To develop the urine analysis for exorphins for routine use in blood and cerebrospinal fluid (CSF).
2. Disorders where patient related validation must be carried out: schizophrenia, depression (uni- and bipolar) and autism.
Method A: HPLC-MS/MS (fragmentation mass spectrometry) technology.
With both a specific HPLC retention time and MS/MS (fragmentation) this method is close to an absolute technique for peptide recognition.
B: ELISA against specific proteins (gliadin, gluten and casein and transglutaminase 6) (Table 1 og 2).
Background A: schizophrenia: increased opioid peptide levels have been found in Schizophrenia using HPLC, immune assay and behavioral tests. [1–6] as part of a general peptide increase in urine. Since peptides are signaling compounds inhibition of peptidases during transport and work up of samples is critical to prevent break down, which is as expected fast at room temperature.
Strongly supporting is view is the data on postpartum psychoses (a very symptom rich psychosis) where also amino acid sequence of human casomorphin found increased, has been done [7–8]. The opioids can explain most of the symptoms of the psychotic schizophrenic state [6]. It is of paramount importance then to measure these peptides in carefully diagnosed patients on and without medication, in urine, blood and spinal fluid.
As can be seen in Table 1, it is important to measure IgA and IgG antibodies against the precursor proteins for the exorphins, which are found increased by several groups, and also have direct effects on the nervous system [9].
B. In depression increase levels of peptides has been found [18,28,29] and also opioid levels measured as opium receptor binding peptides [28]. In schizoaffective psychosis MS/MS exact detection of exorphins have been published [6]. Also in this syndrome it is critical to be able to measure the exorphins in blood and CSF, especially since the peptidases involved in break down of exorphins are decreased in depressions [30,31]. Inflammatory interleukins are also increased in depressions both uni- and bipolar [32] indicative of inflammatory processes probably in the gut. Inflammatory interleukins increase the permeability of epithelial membranes [33].
C. Autism. Considerable work has been done using HPLC with UV detection and co-chromatography [12,34–40]. However, with HPLC–MS/MS we can ensure that we are measuring only the exorphins and not chromatographically similar peaks that hide inside the main peak [41–43]. We therefore need to validate the new method in autism for both urine, blood and CSF (CSF collected only when spinal tap has to be done in any case).
Inhibition of break down in urine, blood and cerebrospinal fluid (CSF)
After extensive testing we have been left with three inhibitors. Citric acid 0.2 M; acetic acid 0.2 M and aprotenine [44,45].
These body fluids will be provided by Prof Dr E. Severance and Prof Dr R. Yolken (Johns Hopkins Univ.) and Prof Dr. Cunningham (Uppsala Univ. Sweden). Lab 1 provides monovettes with citric acid as peptidase inhibitor for urine collection. Blood will be collected in EDTA – aprotenin vacuum test tubes (Vacutainer) as will be CSF.
HPLC and MS/MS detection.
The amount of urine analyzed on the HPLC after work up = 250 nanomles creatinine. To pick out generally active peptides in any one disorder, five and five autistic children or schizophrenic derived and depressive derived urines are mixed, creatinine re-determined and rerun. Peaks that are common to all patients increase or remain the same, while individual peaks of material on the HPLC runs are diluted out.
The complete procedure is published in detail [48]. If we use reporter ions we do not have to match all the peaks as shown in attached figures. On Fig. 1, synthetic bovine β-casomorphine 1-4 (Y-P-F-P) is compared to biologically isolated compound from a batch of five autistic children. On Fig. 2, the faster routine analysis using reporter ions is shown for bovine β-casomorhne 1-4. Top trace is synthetic casomorphin 1-4 and bottom trace is biologically isolated compound. The complete analysis for a series of opioids is published [48].
Program is then in sequence:
– A: further validation of method for urine in the different disorders;
– B: validation of method for blood in the same disorders;
– C: validation of method for CSF (spinal fluid) in schizophrenics and depressive patients.
NB.
To avoid overlooking new compounds a complete HPLC run with UV 215 nm (peptide bonds); 280 nm (aromatic groups) and 325 nm (Indolyl-acryloid) shall be run for urines. If sufficient serum is available and spinal fluid these will also be run on HPLC in addition to MS/MS detection.
Antibody assays will be done at Johns Hopkins using ELISA, Transglutaminase 6 antibodies at Lab 1 also using ELISA assay.
Figures and references not available in the abstract.

Table 1 Antibodies of type IgA and IgG increased in relevant disorders.

Disorder	References
Autism spectrum	Reichelt et al. [10]; Lucarelli et al. [11]; Cade et al. [12]; Vojdani et al. [13]; Kawashti et al. [14]; Trajkowski et al. [15]; Lau et al. [16]; de Magistris et al. [17]
Depression	Sælid et al. [18]; Maes [19]
Bipolar	Severance et al. [20]
Schizophrenia	Dohan et al. [21]; Reichelt and Landmark [22]; Samaro et al. [23]; Dickerson et al. [24]; Severance et al. [25]; Jin et al. [26]; Niebuhret al. [27]

Reference no in parenthesis is found in the reference list. The antibodies are of the IgA and IgG type and not IgE often found in allergic pathology.

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

http://dx.doi.org/10.1016/j.eurpsy.2016.01.840

S25

Gastroenterology issues in schizophrenia: Why the gut matters

E. Severance
Johns Hopkins University School of Medicine, Department of Pediatrics, Baltimore, MD, USA

Numerous risk factors for schizophrenia can be reconciled through a common enteric source. These risk factors include systemic

and localized inflammation, compromised endothelial barriers, IgG sensitivities to food antigens, exposure to viral and parasitic pathogens, and autoimmunity. The gut in a homeostatic state equates with a functional digestive system, cellular barrier stability and properly regulated recognition of self and non-self antigens, as managed by a complex community of resident microbes. Our studies address how environmental and genetic factors relate to GI dysfunction, impact the resident gut microbiota and result in dysregulation of processes in the host central nervous system. We hypothesize that disturbance to GI equilibria activates peripheral immune factors including complement pathway components that function in synaptic pruning. We evaluate these issues with peripheral immune biomarkers and deep sequencing in a number of case-control psychiatric cohorts that include antipsychotic-naïve individuals. Although certain medications and lifestyle factors might affect GI functioning, our findings support a GI pathology inherent to the schizophrenia disease process and a role for the gut-brain axis in complex brain disorders. The identification of those individuals affected by GI-related risk factors will enable appropriate and individualized treatments to be designed and tested for efficacy of both gut and brain-related symptoms.

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

http://dx.doi.org/10.1016/j.eurpsy.2016.01.841

S26

The role of the gut microbiota in mood and behaviour. Whether psychobiotics can become an alternative in therapy in psychiatry?

S. Van Hemert[2], W. Marlicz[3], P. Szachta[1,*], E. Pekelharing[2], G. Ormel[2], I. Łoniewski[4], L. Ostrowska[5], J. Samochowiec[6]
[1] *Vitaimmun Medical Center, Research Department, Szczecin, Poland*
[2] *Winclove Probiotics, Hulstweg 11, 1032 LB, Amsterdam, Netherlands*
[3] *Pomeranian Medical University, Department of Gastroenterology, Szczecin, Poland*
[4] *Sanprobi, Sp. Z o.o. Sp. K., Szczecin, Poland*
[5] *Medical University, Department of Dietetics and Clinical Nutrition, Bialystok, Poland*
[6] *Pomeranian Medical University, Department of Psychiatry, Szczecin, Poland*
* *Corresponding author.*

Introduction Novel research concepts based on therapies aiming to modulate intestinal microbiota are emerging. The evidence is mounting that gut-brain axis plays an important role in the development of mood and depressive disorders [1]. The similarities between blood brain barrier (BBB) and gut vascular barrier (GVB) and their role in chronic diseases have been recently unraveled [2]. Especially convincing data come from animal models, where administration of probiotics and antibiotics in germ and pathogen free mice showed beneficial role in the regulation of behavior, cognition, pain, anxiety and mood.

Aims and results Based on available data as well as on studies looking at the effect of multispecies probiotics (Ecologic® Barrier containing B.bifidumW23, B.lactisW52, L.acidophilusW37, L.brevisW63, L.caseiW56, L.salivariusW24, L.lactisW19, L.lactisW58) on cognitive reactivity to sad mood in healthy volunteers [3] we designed the human trial aiming to compare microbiome alterations and response to therapy in patients with depression and schizophrenia. Moreover, in vitro and in vivo data support the notion that multispecies probiotics are capable of improving gut barrier function [4] and may alleviate disorders affecting mood and depressive-like behavior. We postulate that therapies modulating the microbiome-gut-brain axis warrant further investigations.

Conclusion Multispecies probiotics have the potential to influence the gut-brain axis and alleviate mental disorders. Ongoing clinical study in patients with depression and schizophrenia will help to further unravel the role of gut-brain axis in the treatment of patients with psychiatric disturbances.

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

References

[1] Smith PA. Brain meet gut. Nature 2015;526:312–4.
[2] Spadoni I, Zagato E, Bertocchi A, et al. A gut vascular barrier controls the systemic dissemination of bacteria. Science 2015;350(6262):830–4.
[3] Steenbergen L, Sellaro R, van Hemert S, et al. A randomized controlled trial to test the effect of multispecies probiotics on cognitive reactivity to sad mood. Brain Behav Immun 2015.
[4] Van Hemert S, Ormel G. Influence of the multispecies probiotic Ecologic® barrier on parameters of intestinal barrier function. Food Nutr Sci 2014;5:1739–45.

http://dx.doi.org/10.1016/j.eurpsy.2016.01.842

E-mental Health: Updates on recent achievements and pitfalls

S27

E-Mental Health and models of care: The evidence base and feasibility of picking one vs. another?

D. Hilty
Keck School of Medicine at USC and LAC + USC Medical Center, Los Angeles, USA

The patient-centered care features quality, affordable, and timely care in a variety of settings – technology is a key part of that – particularly among younger generations and child and adolescent patients. The consumer movement related to new technologies is nearly passing clinicians by, as new ways of communicating with others (text, email, Twitter, Facebook) revolutionizes how we experience life and access healthcare. This paper explores a continuum with healthy, innovative behavior on one end (e.g., social media) and pathological Internet use on the other end–and the range of self-help and e-mental healthcare options being used. Specifically, it focuses on how social media adds to, yet may complicate healthcare delivery, such that clinicians may need to adjust our approach to maintain therapeutic relationships, interpersonal/clinical boundaries, and privacy/confidentiality. We suggest planning ahead to discuss expectations about online communication between doctors and patients as part of the informed consent process, offer other do's and don'ts for patients and clinicians, and review applicable guidelines. More research is needed on consumer and patient use of technology related to healthcare, as is an approach to basic and advanced measurement of outcomes.

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

http://dx.doi.org/10.1016/j.eurpsy.2016.01.843

S28

After all, is E-Mental Health capable of making a paradigm shift?

M. Krausz
Institute of Mental Health at UBC, Vancouver, BC, Canada

Only a very small percentage of adolescents and young adults with mental challenges is able to access specialized care. Access is