personalised therapeutics as well as recurrence-preventive strategies.

No conflict of interest

References

[1] Geoffroy, P.A., Bellivier, F., Scott, J., Etain, B., 2014. Seasonality and bipolar disorder: A systematic review, from admission rates to seasonality of symptoms. Journal of Affective Disorders 168, 210–223. doi:10.1016/j.jad.2014.07.002.
[2] Hochman, E., Valevski, A., Onn, R., Weizman, A., Krivoy, A., 2016. Seasonal pattern of manic episode admissions among bipolar I disorder patients is associated with male gender and presence of psychotic features. Journal of Affective Disorders 190, 123–127. doi:10.1016/j.jad.2015.10.002.

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P.355
Delayed increase of perinatally downregulated serum brain-derived neurotrophic factor concentrations may be a risk factor of postpartum depression

T. Mikoteit [1,2], N. Von Felten [1], I. Hoesli [3], S. Tschudin [3], M. Hatzinger [1], A. Eckert [4], S. Brand [2,5,6]

[1] *Psychiatric Services of Solothurn and University of Basel, Clinic for Psychiatry- Psychotherapy and Psychosomatics, Solothurn, Switzerland*
[2] *Psychiatric Clinics of the University of Basel, Center for Affective- Stress and Sleep Disorders, Basel, Switzerland*
[3] *University Hospital Basel, Clinic of Obstetrics and Gynecology, Basel, Switzerland*
[4] *Psychiatric Clinics of the University of Basel, Neurobiology Laboratory for Brain Aging and Mental Health, Basel, Switzerland*
[5] *Kermanshah University of Medical Sciences, Department of Psychiatry, Kermanshah, Iran*
[6] *University of Basel, Department of Sport- Exercise and Health, Basel, Switzerland*

Objectives: Postpartum Depression (PPD) is a depressive episode occurring after childbirth. Prevalence estimates range from 13-19% [1]. As biological backgrounds of PPD are still unclear, it is unknown how to predict the development of PPD by a biomarker. One candidate biomarker could be brain derived-neurotrophic factor (BDNF), a neurotrophic growth factor, which is involved in neuronal cell differentiation, growth and survival. As serum BDNF (sBDNF) correlates with central BDNF levels, sBDNF has been acknowledged as a peripheral indicator of neuroplasticity. Under chronic stress conditions, sBDNF is downregulated, and low sBDNF concentrations have been linked to the development and maintenance of major depression [2,3]. In pregnancy sBDNF levels vary physiologically, with lowest concentrations in the third trimester. Thus, perinatally decreased sBDNF may also be a risk factor for PPD. [4] The aim of this study was to examine, if 1) PPD was associated with decreased sBDNF cross-sectionally, and 2) if prenatal sBDNF could predict PPD twelve weeks after delivery.

Methods: Thirty pregnant women (age: 33.33 $\pm$ 4.44 years, 46% primipara) were enrolled in this prospective longitudinal study. Depression severity and sBDNF were assessed three times: At 32nd week of gestation; 2 days and 12 weeks after delivery. Participants completed the Edinburgh Postnatal Depression Scale (EPDS; cut-off: $\geq$ 10); sBDNF was measured with a Sandwich ELISA. Participants with PPD (EPDS $\geq$ 10) were compared to participants without PPD. At 12th week postpartum, eight (38%) of 21 participants suffered from PPD.
Results: Cross-sectionally, sBDNF levels did not distinguish between the groups "PPD at 12th week postpartum" and "no PPD", neither at 12th week postpartum ($t(19) = 1.049$, $p = .307$) nor at 32nd week of gestation ($t(20) = -1.219$, $p = .212$). sBDNF levels increased significantly between 32nd week of gestation and the 12th week postpartum in participants with no PPD ($t(12) = -4.11$, $p = .001$); in contrast, in participants with PPD, sBDNF levels remained unaltered and low during that time period ($t(7) = -.22$, $p = .834$). A mixed ANOVA revealed a significant interaction between time of measurement and depression status ($F(1,19) = 7.03$, $p = .033$, partial $\eta^2 = .217$). sBDNF was not significantly associated with EPDS scores at any time ($r = .314$, $p = .091$, $N = 30$ for 32nd week of gestation and $r = -.212$, $p = .356$, $N = 22$ for 12 weeks postpartum).
Conclusion: The pattern of results suggests that BDNF may be involved in the development of PPD, though in a complex manner: 1) In contrast to our expectations, at 12th week postpartum sBDNF did not differ between participants with PDD and those without PPD; 2) Antenatal sBDNF levels at 32nd week of gestation were not predictive to PPD status at 12th week postpartum. 3) However, a lack in postnatal increase of sBDNF from downregulated antenatal sBDNF levels up to normal postnatal levels was indicative for development of PPD. To summarise, we assume, that a delayed normalization of perinatally downregulated BDNF expression may be a risk factor for development of PPD. Further longitudinal studies on sBDNF trajectories and depression are needed to confirm this assumption.

No conflict of interest

References

[1] O'Hara, M.W., McCabe, J.E., 2013. Postpartum depression: current status and future directions. Annu. Rev. Clin. Psychol. 9, 379–407.
[2] Sen, S., Duman, R., Sanacora, G., 2008. Serum brain-derived neurotrophic factor, depression, and antidepressant medications: meta-analyses and implications. Biol. Psychiatry 64, 527–532.
[3] Bocchio-Chiavetto, L., Bagnardi, V., Zanardini, R., Molteni, R., Nielsen, M.G., Placentino, A., Giovannini, C., Rillosi, L., Ventriglia, M., Riva, M.A., Gennarelli, M., 2010. Serum and plasma BDNF levels in major depression: a replication study and meta-analyses. World J. Biol. Psychiatry 11, 763–773.
[4] Lommatzsch, M., Hornych, K., Zingler, C., Schuff-Werner, P., Höppner, J., Virchow, J.C., 2006. Maternal serum concentrations of BDNF and depression in the perinatal period. Psychoneuroendocrinology 31, 388–394.

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P.358
Reduced cerebellum volume and ataxia-like motoric phenotype in transgenic mouse, carrier of human CYP2C19 gene

F. Milosavljević [1], M. Vučić [1], M. Manojlović [1], T. Miloševski [1], B. Batinić [1], M. Novalen [2], S. Miksys [2], R. Tyndale F [2], M. Ingelman-Sundberg [3], V. Pešić [1], M. Jukić M [1,3]

[1] *Faculty of Pharmacy- University of Belgrade, Department of Physiology, Belgrade, Serbia*
[2] *University of Toronto, Department of Pharmacology and Toxicology & Department of Psychiatry, Toronto, Canada*
[3] *Karolinska Institutet, Section of Pharmacogenetics- Department of Physiology and Pharmacology, Stockholm, Sweden*

Introduction: *CYP2C19* transgenic mouse (*2C19TG*) is generated by the insertion of 12 copies of the human *CYP2C19* gene into mouse genome. This animal model is a tool to study the neurodevelopmental role of CYP2C19 in vivo, since this enzyme is expressed in the foetal brains of *2C19TG* mice and humans. Previous studies [1,2] showed anxiety and depression-like behaviour in these mice, while the aim of this study was to characterize the motoric function of the *2C19TG* mouse.
Methods: Whole brain dopamine concentration was measured in the brain homogenate of 23 adult mice by the HPLC-MS method. Motoric function in 50 mutant and 43 control mice of both genders was tested by the rotarod and beam walking tests. Beam walking test was repeated after treatment with dopaminergic receptor antagonists, Ecopipam (0.1 mg/kg) and Raclopride (0.25 mg/kg) as a follow-up. The sections of 10 6-month old and 8 15-months old mice were stained with anti-tyrosine hydroxylase antibody and the number of dopaminergic neurons was counted on histological slides under microscope. Next, after transcardial perfusion of 30 *2C19TG* and 30 control mice of both genders with contrast agent (4% Paraformaldehyde, 0.05M Gadoteridole, 0.01M Phosphate buffered saline, pH=7.4), cranium containing the whole brain was scanned overnight by the 9.4T MRI scanner. The volumes of 39 brain regions were quantified according to the mouse brain atlas [3]. Student's t-test and two-way ANOVA were used to evaluate statistical significance of between-group differences.
Results: Adult *2C19TG* mice are hyperdopaminergic, as they exhibit 15% increased dopamine concentration ($p<0.001$). They also show hyperkinetic motoric phenotype with the ataxia-like walking pattern and pathological clasping reflex. In the beam walking test *2C19TG* mice had 20% longer beam crossing time ($p=0.007$) and 60% more paw slips ($p<0.001$) then the controls, and this motoric impairment could not be improved with antidopaminergic drugs. Both younger and older *2C19TG* mice exhibited only a marginal reduction in the number of dopaminergic neurons of both substantia nigra and ventral tegmental area in a subset of coronal sections. This was confirmed by the gadolinium-enhanced neuroimaging that showed no change in substantia nigra volume in *2C19TG* mice. On the other hand, significant differences in volume were identified in 11 regions, including cerebellum (-8.3% $p<0.001$) and striatum (+3.0%, $p<0.001$), which are the regions connected with the motoric function. The volumetric changes were detected in the hippocampus (-1.3%, $p=0.027$), amygdala (+2.8%, $p<0.001$), septum (+3.3%, $p=0.014$) and nucleus accumbens (+3.5, $p=0.004$) of *2C19TG* mice. These brain regions are involved in emotional and motivational functions.
Conclusion: Ataxia-like motoric phenotype in *2C19TG* transgenic mice is probably caused by changes in cerebellum, while hyperdopaminergism is most likely the compensatory adaptation, whereas the changes in the hippocampus, amygdala, septum, and nucleus accumbens may be connected with the mutants' depression-like phenotype and susceptibility to stress. Therefore, *CYP2C19* transgenic mouse in potentially useful model of hyperkinetic disorders, and our findings hint at the possible impact of CYP2C19 enzyme on the development of the several brain regions involved in motor and emotional functioning.

No conflict of interest

References

[1] Persson, A., Sim, S.C., Virding, S., Onishchenko, N., Schulte, G., Ingelman-Sundberg, M., 2014. Decreased hippocampal volume and increased anxiety in a transgenic mouse model expressing the human CYP2C19 gene. Mol Psychiatry 19, 733–741.
[2] Jukić, M.M., Opel, N., Ström, J., Carrillo-Roa, T., Miksys, S., Novalen, et al., 2017. Elevated CYP2C19 expression is associated with depressive symptoms and hippocampal homeostasis impairment. Mol. Psychiatry 22, 1155–1163.
[3] Johnson, G.A., Badea, A., Brandenburg, J., Cofer, G., Fubara, B., Liu, S., Nissanov, J., 2010. Waxholm space: an image-based reference for coordinating mouse brain research. Neuroimage 53 (2), 365–372.

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P.359
Expression profile of genes involved in endolysosomal pathway in CD45+ blood cells as potential marker for differentiation of synucleinophaties

A. Bezrukova [1], D. Bogdanova [1], K. Senkevich [1,2], N. Gomzyakova [3], E. Gracheva [4], I. Miliukhina [1,2,4], N. Zalutskaya [3], S. Pchelina [1,2,4], T. Usenko [1,2]

[1] *Petersburg Nuclear Physics Institute named by B.P. Konstantinov of National Research Centre «Kurchatov Institute», Molecular and Radiation Biophysics Division, Gatchina, Russia*
[2] *Pavlov First Saint Petersburg State Medical University, Scientific Research Center, Saint-Petersburg, Russia*
[3] *V.M. Bekhterevs National Medical Research Center Psychiatry and Neurology, Third Department of Psychiatric Geriatrics, Saint-Petersburg, Russia*
[4] *Institute of Experimental Medicine, Center of the Neurodegenerative disease, Saint-Petersburg, Russia*

Introduction: The synucleinopathies are group of neurodegenerative diseases characterised by the abnormal accumulation of neurotoxic forms of alpha-synuclein in neurons and glial cells [1]. These include Parkinson's disease (PD), de-