Mitochondrial respiration in bipolar affective disorder

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Bipolar affective disorder (BAD) is a serious mental disorder. The predisposition to BAD is determined by genetic, other biological and psychosocial factors. Changes in the activities of compounds of intracellular signaling pathways are studied with the aim of discovering new biological markers of mood disorders or predictors of response to pharmacotherapy, which can be easily examined in blood samples.

The aim of our study is to find out association between changes in energy metabolism and different episodes of BAD. Selected mitochondrial parameters were measured in peripheral blood components. The analyses were examined in patients suffering from BAD in different states of BAD and in healthy controls. Clinical evaluation of the BAD patients was provided by experienced clinicians using following questionnaires: MDQ, MADRS, YMRS, CGI-S and BPRS. Mitochondrial respiration was examined in intact and permeabilized blood platelets using high resolution respirometry. It was evaluated by both respiratory rate and respiratory control ratios. Enzyme activities (citrate synthase, electron transport chain complexes - complex I, II+III, IV) were measured spectrophotometrically. Statistical analyses were performed using the STATISTICA data analysis software system, version 12. Activities of individual complexes were normalized to citrate synthase activity. Statistical significance was evaluated using the ANOVA and post-doc Scheffé test.

Our preliminary results showed increased physiological respiration in intact platelets from manic patients. Complex-I linked respiration was found increased in manic patients and in remission compared to healthy controls. Citrate synthase activity was not changed in BAD patients compared to controls. Decreased complex IV activity was observed in BAD depressive patients in comparison to controls.

Our results support the hypothesis that changes in the rate of oxygen consumption and electron transport chain complexes activities may participate in pathophysiology of BAD. In conclusion, better insight into molecular mechanisms of cellular respiration could lead to better understanding of pathophysiology of BAD. Mitochondrial dysfunctions in different episodes of BAD should be further studied.

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