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Research of molecular mechanisms of pathogenesis of depression: bioinformatical analysis of transcriptomic data

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Motivation and Aim: According to the data of World Health Organisation (WHO), depression is one of the most widespread mental disorders, affectimng more than 300 million people of all ages worldwide. Depression is also a leading cause of disability worldwide. Although there are known, effective treatments for depression, fewer than half of those affected in the world (in many countries, fewer than 10 %) receive such treatments.

At this point, a significant amount of studies on the pathogenesis of depression have been done, and a several hypotheses have been formulated. Such hypotheses include monoaminic, stress induced, neuro-plastic and several others. Despite that, no comprehensive understanding of ethiopathogenesis of depression exists. Because of that search for molecular mechanisms, involved in pathogenesis of depression has to go on.

Methods and Algorithms: One of the problems in the research of depression is the impossibility of obtaining brain tissue from a living patient with depression. Due to that, one of the possible approaches to studying depression is to study pathogenesis of depression on animal models. Since no perfect animal model of depression exists, several different models have to be studied. Simultaneous analysis of various models of depression allows to identify the most important processes, which could be involved in the development of this disorder.

Results: At this moment, we are conducting the bioinformatical analysis of transcriptomic data, derived from various models of depression. We study both models, obtained in our own laboratory – model of acute stress (forced swimming test, Porsolt test), model of stimulation of immune system (lipopolysaccharide injections), and ones available in Gene Expression Omnibus (GEO). Also we are analysing the transcriptomic data, derived post-mortem from the patients with major depressive disorder from GEO.

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