At present, depressive disorders affect a considerable proportion of population, their incidence showing an increasing tendency in the developed world. Depression mostly afflicts people in the age of 25-35. It occurs in females twice as often as in males. Depression involves an intense subjective suffering accompanied with a disruption of normal everyday functioning. Moreover, when compared with the healthy population, there exists a thirty times higher danger of suicide in the people affected with depression. The illness unfortunately tends to become chronic, the probability of recurrence of an episode being 50%. Furthermore, some patients suffering from the depressive disorder do not adequately respond to an antidepressant therapy; we call such patients treatment-resistant.

There certainly exists a close relation between the endocrine and the central nervous system, most endocrine disorders being potentially accompanied with changes of psyche and vice versa. With affective disorders the most frequently reported disruption involves an interference with the function of the hypothalamicpituitary-adrenocortical (HPA) axis, though various studies repeatedly acknowledged also deviations of the hypothalamic-pituitary-thyroid (HPT) axis. The most frequent findings comprise an altered serum thyrotropin response to thyrotropin releasing hormone, a transient serum hyperthyroxinemia, and flattening of the diurnal rhythm of the thyroid stimulating hormon.

Several hypotheses currently discussed in the medical science might elucidate changes in the function of the HPT axis in respect of affective disorders. One of them suggests it might be a form of the central subclinical hypothyroidism related to the system euthyroidism, or, alternatively, changes in the consequence of a lowered level of serotonin in the brain, or an altered activity of the beta-adrenerg receptors in the central nervous system, or the central dysregulation of the HPA and HPT axes.

The connection of the HPT axis with affective disorders is supported with efficacy of thyroid hormones in the affective disorder treatment. At present, the triiodothyronin (T3) and thyroxin (T4) are used in particular in the therapy of resistant forms of the disorders. The underlying knowledge being that the deflection of the HPT axis may reflect an increased vulnerability to a relapse of the disease. Many studies have shown a positive potentiating effect of the T3 with tricyclic antidepressants, however, controlled studies with other types of antidepressants are still missing. Thyroxin medicated in a supraphysiological dose is effective in the therapy of the rapid cycling form of the bipolar affective disorder.

The interconnection of the endocrine and neuronal systems in respect to the pathogenesis of depression has not been explained so far. In our research we have to rely on non-straightforward methods; cell models, in particular those of erythrocytes and thrombocytes, or their membranes, are frequently used.

Another factor playing its role in the emergence of depression is a characteristic of cell membranes in specific areas of the brain: it consequently influences changes in the neuromediator systems. The character of a cell membrane might, hypothetically, decide about the accessibility of the binding site of a receptor. The transport via a cell membrane is influenced by its lipid composition and fluidity.

In the research part of the work we attempted, on a peripheral model represented by erythrocytes (RBC), to study the relation of the depressive illness to the parameters of the uptake of L-triiodothyronin (L-T3).The uptake of L-T3 might reflect an interference with the function of the HPT axis. The underlying assumption was supplemented with another one assuming that the transport of L-T3 via a membrane is under the influence of the composition of the membrane.

The fact is that L-T3 is being transported via erythrocyte membrane by means of the facilitated diffusion with help of the stereospecific saturable mediator system independent of Na+.
We aimed at monitoring changes of kinetic parameters of the uptake, i.e. Vmax, maximal velocity, and Km apparent Michaelis constant, and changes in membrane fluidity.
The object of our research was a sample comprising 24 individuals suffering from a depressive disorder; they underwent measuring before and one month after the treatment with citalopram. The resultant data were compared with a group of 19 healthy controls. The biochemical parameters were correlated with clinical assessment based on the depression scales.
All the patients involved adequately responded to the antidepressant treatment.
With depression, the function of the transmitter for L-T3 is changed, for the kinetic parameters of the uptake of L-T3 into erythrocytes were, before the treatment, significantly higher in the depressive patients compared to the controls. Furthermore, we have found out that the function of the transmitter for L-T3 is equally related to the changes of the lipid bilayer in which the transmitter seems to be immersed. The anisotropy of the fluorescence of 1,6-diphenyl-1,3,5-hexatrien (DPH) in RBC membranes, which reflects their membrane microviscosity (and fluidity), was significantly increased in the group of depressive patients before the treatment in comparison with controls. After a month treatment there was virtually no alteration; no normalization of values towards controls took place.
The results of the research support the hypothesis that in clinically euthyroid patients suffering from depression we can find subclinical markers reflecting interference with the function of the HPT axis in the pathophysiology of the affective disorders; they also confirm the hypothesis that an altered function of the axis results in the change of the uptake of L-T3 into erythrocytes. The kinetic parameters of the uptake of L-T3 into erythrocytes, however, do not correlate with the depressive syptomatology and are affected by the characteristics of the lipid bilayer.
The hypothesis concerning disturbation of the function of the HPT axis was therefore interrelated with the membrane hypothesis of the affective disorders, which says that the vulnerability to depression can appear as a consequence of changes in the composition of biophysical characteristics of the lipid part of cell membranes.
We could therefore consider the L-T3 uptake into RBC to be a membrane peripheral marker reflecting the disturbation of the HPT axis in depressions.