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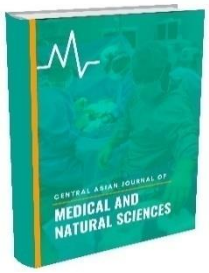

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Dimensional Rhythmological Model of Depression

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Abstract: The systematics of affective disorders is traditionally based on nosological or syndromal basis. Accordingly, depressions are distinguished within such forms of mental diseases as manic-depressive psychosis, schizophrenia, psychogenias, etc., as well as within the framework of the classical clinical and etiological dichotomy. This differentiation is made within the framework of a classic clinical and etiological dichotomy, defining the endogenous or exogenous nature of affective disorders. This distinction ignores a wide range of transitional forms (endoreactive depression, characteristic dysthymia, etc.) and endogenous spectrum disorders (endogenomorphic depression) that, while manifesting within personality, somato- and psychogenic pathology, are similar in a number of clinical parameters to such reference endogenous phases as circular melancholia.

Key words: affective disorders, depression, dimensional rhythmology model.

Introduction. Typological taxonomies of depression, based on the prevalence of one or another component of the hypothymic syndrome (anxious, apathetic, dysphoric, and other types of depression) or the complicity of non-affective registers (hysterical, hypochondriacal, senestopathic depression) in the clinical picture, also (barring wistful depression) fail to account for the association of depression (or the lack thereof) with cognitive deficits.

This is possible because a dimensional approach to the analysis of psychopathology can, on the one hand, rank depression in terms of the involvement of endogenous vital mechanisms and, on the other hand, distinguish between affective states at the opposite endogenous pole and heterogeneous circadian melancholia.

The most relevant dimensional analysis is the rhythmological model, which allows us to rank depression according to the presence of the main components of a vital symptom complex (pathological circadian rhythm, longing, ideas of self-blame, etc.). The doctrine of the rhythms of depression, dating back to the pre-desological period, was given its final form by the Kraepelin synthesis, providing for the separation of manic-depressive psychosis ("manic-depressive illness" in the terminology of F. Goodwin and K. Jamison, on the basis of periodicity (phase) of the onset of affective disorders. Along with the circularity principle, E. Kraepelin, in co-authorship with U. Fleck was the first to put forward the circadian/vital symptom-complex (morning mood deterioration, sleep-

wake cycle disturbance with early insomnia) as one of the clinically significant features of circadian depression. Kreplin's modus operandi for rating affective disorders in terms of their inherent rhythms, despite the introduction of operational criteria, vocabulary changes and other innovations, is retained in contemporary systematics (ICD-10, DSM-IV-TR). The indications given in this case allowed Krasnov to classify the diurnal rhythm with an increase in depression in the morning hours as a major symptom of a depressive episode.

The concept of rhythms as an obligatory component of endogenous depression, thanks to the advances of the past decades in the study of the fundamentals of affective pathology, has been confirmed both by epidemiological evidence of the high frequency of abnormal changes in circadian rhythm in recurrent depressive disorder and by pathophysiological research ("circadian rhythm disturbances are key to understanding the pathophysiology of mood disorders").).

The neurobiological circadian hypothesis views abnormal circadian rhythms as a link between the manifestation of depressive symptoms and a host of factors (genetic susceptibility, environmental stressors, psychotraumatic events, somatic illness, etc.) involved in the pathogenesis of affective illness. This hypothesis is based on the notion of an interaction between rigid circadian rhythms and the sleep-wake cycle, involving two processes: the chronobiological (C-circadian process) and the homeostatic (S-sleep process) in a reciprocal relationship, with a deficiency of C-process a precondition for affective illness. The advanced positions are confirmed in numerous researches executed with use of various fundamental approaches - molecular-genetic, polysomnographic, experimental (daily rhythms of locomotor activity, hormonal secretion and body temperature in rodents under conditions of acute phase shift and disorganization of light/dark cycle).

The concept of the circadian rhythm as a component of the psychopathological structure of depression is now supported in most current clinical studies, both in the nosological tradition and in the neocerebellar trend.

The rationale for this concept is at its most complete in a series of clinical and statistical naturalistic studies by G. Murray [58, 59]. According to the author, the diurnal rhythm of depression is "set" by an endogenous circadian mechanism that provides a mismatch of the "pendulum" of the biological clock, regulated by neurons of the suprachiasmatic nuclei with the participation of the melatoninergic system. The existence of such rhythms is unequivocally interpreted by the author as an informative sign of endogenous (vital) depression.

Conclusions: Thus, the results of both epidemiological and basic and clinical studies allow us to conceptualise the circadian rhythm as an underlying disorder, an obligate component of the structure of depression. However, a very important point must be stressed. The relationships established in the studies discussed above (both early last century and contemporary) are limited to an analysis of circadian melancholy and therefore do not explain a number of phenomena outside of endogenous affective disorders and related not only directly to the psychopathology of circadian rhythm, but also to the problem of depression rhythms in general.

First and foremost, the boundaries of the circadian rhythm require clarification, as it is unclear whether the circadian rhythm operates exclusively within circadian melancholia or extends to affective disorders extending beyond endogenous illness.

The psychopathological characterization of circadian depressions lacking a rhythm is also up for debate. While this is a well-established fact - labelled by some as atypical - the concept of circadian rhythm as an obligatory feature of endogenous depression fails to interpret this fact and hence does not answer the question: what clinical phenomena is rhythm deprivation associated with? Finally, an issue that needs to be analysed from a clinical point of view is the problem of depression rhythms in general. What needs to be established is what happens outside the circadian rhythm. Are circadian and

chronobiological mechanisms in general the only rhythms governing depression, and are there no other pacemakers that provide direction and regulate the severity and frequency of mood disorders?

In seeking to answer these questions, we set out to explore the three questions (in italics) in this study and consequently to select and analyse clinically relevant data for their consideration.

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