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# Individual Metabolic Characteristics of Neuro-Motor Function Disturbances under Experimental Stress and Ways of its Correction

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**ABSTRACT**

All this dictates the need to study the physiological and neurochemical processes underlying adaptive behavior in order to correct the physiological response of the body to an increased level of stress, and develop new approaches to the treatment of its consequences [4].

One of the most important problems in our society is the prevention and treatment of mental disorders. Despite major breakthroughs in neuroscience, there have been no significant changes in the treatment of mental disorders over the past 30 years. This can be partly explained by the lack of compatibility between neurology and clinical practice [5,8]. In recent decades, significant progress has been made in describing the basic neural circuits and cellular-molecular mechanisms of fear conditioning. Converging lines of evidence indicate that the amygdala is necessarily involved in the acquisition, storage, and expression of conditioned fear memory, and long-term potentiation in the lateral nucleus of the amygdala is often proposed as the main synaptic mechanism of associative fear memory [10,26]. Recent research also indicates that the interaction of the prefrontal cortex and the amygdala contributes to the extinction (or inhibition) of conditioned fear. Despite these advances, unresolved issues and results remain that call into question the validity and sufficiency of the current amygdala LTP hypothesis of fear conditioning [3,11]. Drugs with different effects on the catecholaminergic mechanisms of neurotransmission were administered to isolated rats to assess their effect on aggression. L-DOPA in combination with a peripheral decarboxylase inhibitor led to a dose-dependent decrease in the number and average duration of contractions, accompanied by an increase in the latent period of the attack [2,25]. Central levels of dopamine were elevated, while norepinephrine and 5-hydroxytryptamine in the brain were reduced. Almost identical effects on aggression were observed after apomorphine. Pimozide reduced combat, but did not affect the attack delay or the average duration of the battle. All doses of disulfiram virtually eliminated all components of aggressive reactions [1]. A simple system is described to analyze the possibility that enhanced exploratory behavior is indicative of the anxiolytic effect of benzodiazepines in laboratory rodents. The rat was allowed to run freely in a two-chamber arena where two-thirds of the area was lit and one-third was darkened. The two chambers were separated by a black partition fitted with photocells across the opening, and the entire cage rested on an activity

monitor [3,8]. The pathological effects of mitochondrial dysfunction result from both oxidative damage and bioenergetic deficiency and are more pronounced in cells and tissues with high metabolic energy requirements, such as neurons, skeletal muscle, and the rat heart [16].

Metallothionein (MT) is a cysteine-rich, low molecular weight peptide (6-7 kDa) widely distributed in a wide range of eukaryotic species from yeast to mammals. In fact, MT serves as an endogenous detoxifying peptide against toxicity caused by heavy metals, especially cadmium, mercury, and zinc [7]. In addition, recent studies have shown that MT works as an internal scavenger of reactive oxygen species [9]. Zinc exposure is known to cause oxidative stress in living organisms, which can lead to the induction of protective antioxidant defenses, both enzymatic and non-enzymatic. This work aims to provide new data on the existing links between several non-enzymatic components of the antioxidant system that are physiologically related to both metal sequestration and protection against metal-induced oxidative stress, using the blue mussel (Mytilus galloprovincialis) as a model organism [7,21]. However, the role of zinc in preventing oxidative stress caused by diabetic cardiac embryopathy remains unknown. Zinc plays an important role as a cofactor in numerous transcription factors and in a large number of biochemical processes and functions for over 300 different enzymes. It is an essential nutrient required for a variety of biochemical and physiological functions, including growth, development, and puberty in males, etc. Zinc deficiency leads to growth retardation, immune dysfunction and cognitive impairment [9,14]. Some experiments showed that the concentration of zinc in the liver of rats 12 hours after the onset of immobilization stress was significantly higher than that of a rat without stress. Zinc homeostasis is coordinated through regulation by proteins involved in zinc uptake, excretion, and intracellular storage or movement of zinc [13]. Severe stressors, such as associated stress, in animals lead to anorexia and weight loss.

Corticotropin-releasing hormone (CRH) has been shown to be a potent anorectic agent when administered intraventricularly to animals. CRH levels increase in the hypothalamus during immobilization stress. Inhibition of CRH by antibodies or antagonists reverses anorexia associated with restriction stress [11]. Corticotropin-releasing factor (CRF) is involved in the regulation of adaptive responses to stress. In addition to activating the hypothalamic-pituitary-adrenal axis and the autonomic nervous system, CRF also modulates behavioral responses to a wide range of stressful stimuli. Administration of CRF to experimental animals leads to behavioral changes resembling the effects of stress. Inhibition of CRF neurotransmission can normalize a number of stress-induced behavioral changes. Increased activity of the brain's CRF system has been suggested to play a role in stress-related psychopathology, including affective disorders and substance abuse [6,15]. The effects of dopamine and norepinephrine function manipulation, alone or in combination, are considered in relation to two categories of aggression: predatory and affective. Affective aggression is subdivided into shock-induced defensive struggle, isolation-induced aggression, and irritable aggression. The neurochemical, physiological, and behavioral effects of stress exposure have been extensively studied for decades. Most studies in adult rats have shown that the effects of stress fade quickly, so such effects were assessed several hours or days after initial exposure to the stressor [18]. Genetic factors and early adversity play an important role in the etiology of affective and anxiety disorders. Previous studies have shown that postpartum maternal separation can cause persistent abnormalities in emotional behavior and neuroendocrine responses to stress in animals [24].

Some people can develop depression in the absence of any noticeable life stress, while most people who are chronically stressed never develop clinical depression. However, many depressed patients often describe stress-inducing life events as contributing factors. Stress itself is commonly used to induce depressive behavior in animals [13,22]. Studies using models of stress-induced depression have generally indicated an increase in plasma corticosterone levels. It has also been shown that depressive behavior did not appear when the physiological response to

stress was reduced (for example, in genetically modified animals or in animals with removed adrenal glands) [18,27]. Similarly, depressive disorder is associated with an increase in corticoliberin and cortisol production, as well as an increase in the size of the pituitary and adrenal glands, indicating an overall increased activity of the hypothalamic-pituitary-adrenal system together with possible glucocorticoid receptor resistance and defective negative feedback. Studies using these stress-induced models of depression generally report an increase in plasma corticosterone levels, and depression-like behavior does not appear when the stress response is reduced (e.g., in adrenalectomized or genetically modified animals) Similarly, major depressive disorder has been associated with increased production of corticotropin-releasing hormone and cortisol, as well as an increase in the size of the pituitary and adrenal glands, indicating an overall increased activity of the hypothalamic-pituitary-adrenal axis along with possible glucocorticoid receptor resistance and a negative feedback defect. connections [18]. The most common model, and one of the most validated, is the chronic mild stress model, in which animals are exposed to stressors several times a day at unpredictable times. This protocol minimizes the impact of acute stress as each intervention (loud noise, cage tilt, wet bedding, etc.) causes only moderate stress. One of the main advantages of the chronic mild stress model is long-term effects, allowing long-term use of antidepressants to be investigated. Although this model has a reputation for being unreliable, a recent survey suggested that this model may be as unreliable as other models of depression [12,28]. The purpose of this work was to compare the behavior of rats after 2-6 hours of daily support and fixation of the hind limbs (white females and white male rats). and had to compare changes in the size of motor neuron cell bodies. A week of reading after a 7-day download. The experiments were performed on adult male and female white rats weighing 180 ± 200 g, divided into three groups. “Control” was divided into groups “Active” and “Passive” according to the Rotaro method (Moray-Holton anti-orthostatic suspension of the hind limbs for 14 days); We divided into groups by tying stones to our feet in the water. "Readaptation 14 days" from 2 hours to 6 hours (after a 14-day experiment, rats were rapidly injected with zinc-curcumin-glycine-preservative for 14 days and 21 days under normal conditions through a gastric tube for two to three weeks). The mean values of total area in rats in the 21-day group and area occupied by the substance in white female and white male rats were reduced. Rats treated with neuromotor stress showed higher endurance in the 21-day group than rats exposed to neuromotor stress. in a 14 day preparation. In "reacclimatized 14 days" rats, white males and white females, the total area and area of the substance remained the same as in "white male" rats after 14 days, and the body sizes of "large" and "medium" motor neurons were shifted towards growth. As a pharmacological correction of the identified structural and behavioral disorders, local preparations with neuroprotective and endothelioprotective properties, the preservative zinc curcumin glycine, were used. These fundamental results open up new perspectives for preclinical and clinical research. Immune cell growth factors have been proposed to prevent neurotrophins and subsequent atrophy

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