Effects of Physical Activity and Other Types of Stress on Catecholamine Metabolism in Various Animal Species

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Summary

The investigation of catecholamine (CA) metabolism in animals subjected to various types of stress (different pain syndromes; cranial trauma; immobilization; cooling) and physical exercise shows considerable similarity among species in the sequence of changes, leading from the activation to the depletion of the sympathoadrenal system. The changes caused by physical exercise tend to be more pronounced in individuals with a genetic predisposition to greater stress responses. Stress adaption, induced by special training or by long-duration exposure to hypoxia, can substantially prevent the changes caused by physical exercise. Trained rats at rest show accelerated CA turnover, and after exercise, adaptive hypometabolic changes. Physical exercise causes both unspecific changes in CA metabolism, similar to those seen after other types of stress, and more specific ones, i.e., suppression of tissue CA synthesis and nonresponsiveness to exogenous L-tyrosine or L-DOPA. Adrenal CA synthesis could be restored in such animals by treatment with glucocorticoids and cyclic-AMP (c-AMP). The depression of CA synthesis after hard physical activity may be a mechanism for protecting the body from the injurious effect of the excessive CA release that would occur under stress.

Introduction

One of the remarkable qualities of the living organism’s reactions to a variety of differing stressful exterior and interior factors is the generation of universal nonspecific responses. But particular stresses
can also be associated with changes, which are specific, and which serve some particular purpose for the body. CA synthesis and release are affected by stresses, both nonspecifically and, for stresses like severe physical activity, in specific ways. This paper reviews the findings of experiments by us and by others on the changes in CA seen after experimental animals are subjected to various stresses, and to the particular stress of physical activity. Some of this work has been reviewed previously (Matlina, 1972; Matlina and Kassil, 1976; Kassil et al., 1978).

**CA Metabolic Changes Under Stress**

We studied the changes in CA content of rats made to undergo physical activity of different duration and intensity (running in a treadmill for 2, 4, and 6 hours, swimming 45 min, 70 min, 2.5 hours and 8 hours with and without an additional work load), or subjected to the stresses of pain (caused by injecting tetanus toxin into the dorsal horn of the spinal cord, or by electric stimulation of the sciatic nerve); cooling (6 hours); immobilization (7 hours); or, in the rat and guinea pig, cerebral trauma (1.5 min—12 hours) (Kassil et al., 1972; Malysheva and Matlina, 1971; Matlina et al., 1974, 1979; Matlina and Zutler, 1973; Pukhova and Matlina, 1972). On the basis of our observations (Matlina, 1972, 1975) and other investigators' results, we proposed the following three main phases of CA metabolic changes under stress (the duration of each phase is determined by the kind and the force of the effect). The first phase (fast activation) is manifested immediately after the beginning of the stress. It is characterized by release of noradrenaline (NA) in the hypothalamus and in other parts of the central nervous system: this in turn causes activation of adrenergic mechanisms of the brain. During this phase, intensified adrenal medullary activity causes secretion of adrenaline (A) into the blood without reducing the A content in the gland itself; the circulating A acts on the heart, considerably intensifying its work. The second phase (steady continuous activation) is associated with a reduction in the levels of A within the adrenal medulla. There is a release of NA within the heart, and an elevation in the concentration of its precursor, tyrosine. The content of A in liver may increase considerably, leading to a large decrease of glycogen and an increase in the glucose supplied to the body. In the third (depletion) phase, the activity of the sympathoadrenal system is reduced, causing decreases in circulating and tissue NA and A. The concentration of A in the adrenal medulla falls sharply, the secretion into blood is