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Review article

Cross stress adaptation: Phenomenon of interactions between homotypic and heterotypic stressors

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ABSTRACT

Individuals have an inherent capacity to cope with stressors in the form of stress adaptation. Apart from stress adaptation there is another well documented phenomenon known as cross stress adaptation. In this, there is a reduction in stress responsiveness to a novel stressor (in which the adapted organism had never encountered previously) in previously exposed organisms with another stressor given in either continuous or intermittent. However, regarding the existence of cross stress adaptation, there are mixed reports revealing that the positive cross stress adaptation exists between altitude and heat stress; swim and inescapable shock stress, hypoxia and cold stress, psychosocial stressor and exercise. However, there are other reports which reveal the non-existence of cross adaptation between forced swim and noise stress and cold and immobilized stress. The exact mechanisms responsible for cross stress adaptation are not defined and need to be investigated.

Contents

1. Introduction

Individuals have an inherent capacity to cope with psychological stressors in the form of stress adaptation [1]. In the latter phenomenon, there is a reduction in the sensitivity of stress responsive elements including the HPA axis during repetitive stress exposure episodes [37,43]. Apart from stress adaptation, there is another well documented physiological and biochemical phenomenon known as 'cross adaptation' in which there is a reduced stress responsiveness to a novel stressor in previously stress adapted organisms [27,31,46]. Due to bidirectional trend of changes, the adaptive changes caused by one stressor may make the organism more fit to resist the adverse effects of another type of stressor [21,30,31]. Cross adapted organisms respond better to novel stressors at different levels [21,30,31,34]. The clinical and preclinical studies suggest the existence of cross stress adaptation in response to both systemic as well as emotional stressors. Studies have shown the positive cross stress adaptation between altitude and heat stress [19,20,22,38,39], swim and inescapable shock stress [50], hypoxia

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and cold stress [34], and psychosocial and exercise stress [24]. However, there are other reports which reveal the non-existence of cross adaptation between forced swim and noise stress [3] and cold and immobilized stress [35]. The present review describes the discrepancies of the existence of cross stress adaptation at preclinical as well in clinical studies and hypothesizes the mechanisms behind the existence and non-existence of cross adaptation.

2. Evidences of cross stress adaptation

2.1. Preclinical evidences

Preclinical research by LeBlanc demonstrated the existence of cross stress adaptation between repeated cold air exposure and acute hypoxia, and reported that the autonomic adaptation caused by repeated cold air exposure reduces the sympathetic response to acute hypoxia (novel stressor) [33]. The USAF School of Aerospace Medicine performed the cross adaptation studies in rats and revealed the existence of positive cross-adaptation. Repeated exposure of altitude stressor led to lower body weight loss in comparison to single exposure of altitude stressor, suggesting the existence of stress adaptation. In the cross stress adaptation protocol, thermally adapted rats (at 5 °C, 25 °C and 35 °C, for 3 weeks) showed the lower body weight loss in response to acute exposure of altitude stressor. Apart from this, acute exposure of altitude stressor did not produce the significant changes in 24 h urinary excretion rates of creatinine, uric acid, magnesium and sodium in heat acclimatized animals, suggesting the existence of cross stress adaptation at the physiological level. In another set of experiment, the altitude and cold stress adapted rats showed cross adaptation and exhibited lower body weight loss in response to heat and food deprivation as compared to laboratory temperature (non-stress) adapted animals. Furthermore, acute exposure to heat and food deprivation stress did not alter 24-h urinary excretion rates of potassium, uric acid, magnesium, and sodium in altitude adapted rats, thereby suggesting the existence of cross adaptation at the physiological level [19,20,22,38,39].

Weiss et al. [50] studied the phenomenon of adaptation and cross adaptation in rats subjected to inescapable shock and cold swim stressor by assessing the avoidance escape response in the shuttle avoidance escape task. The avoidance-escape deficit was significantly less in repeated inescapable shocks and cold swim stress subjected rats, suggesting the existence of stress adaptation at the behavioral level. Furthermore, the behavioral restoration was also documented in response to acute swim stressor in rats previously subjected to chronic tail shock [50]. Similar findings were reported in response to acute tail shock stressor in repeated chronic swim stress subjected rats suggesting the existence of cross adaptation at the behavioral level. Furthermore, rats subjected to acute tail shock showed a reduction in the norepinephrine levels in the hypothalamus and cortex regions in contrast to stress naive rats. However, rats subjected to chronic tail shock or cold swim did not show a reduction in the norepinephrine levels on exposure to acute tail shock stressor demonstrating the adaptation and cross adaptation at the neurochemical level. Other studies also supported the similar neurochemical adaptation in response to repeated stress exposure [10,36,48,51]. The maintenance of norepinephrine levels in subjects receiving repeated stress exposures was attributed to increase in the tyrosine hydroxylase activity (a rate limiting enzyme involved in norepinephrine synthesis) and decrease in the norepinephrine uptake in the cortex. Based on these, it was proposed that the restoration of the norepinephrine levels in the brain is mainly responsible for adaption as well as cross adaptation in response to different stressors [50].

Cox et al. [17] demonstrated the positive prophylactic effects of repeated swim stress (exercise) on tail shock-induced hypertension in borderline hypertensive rats. These rats were exposed to uncontrollable tail shocks along with daily swim stress and results revealed the significantly lesser rise in the systolic and diastolic blood pressures in repeated swim stress and tail shock as compared to corresponding shock only subjected rats. At the neurochemical level, acute tail shock-induced increase in the norepinephrine levels was less marked in repeated swim stress and tail shock subjected rats, suggesting that the presence of a mild stressor (swim) may have a positive effect and reduce the deleterious effect of severe stressor (electric shock) [17]. Armario et al. [3] studied the effects of chronic tail shock stressor on the physiological responses in male rats previously exposed to chronic immobilization stress. At the endocrine level, normalization of the ACTH response was observed in animals repeatedly exposed to immobilization or tail shock stress suggesting the development of stress adaptation. There have been a number of studies demonstrating the existence of adaptation at the ACTH level in response to repeated immobilization and tail shock stressors [3,17]. However, exposure to a novel acute stressor (tail shock) in chronically immobilized rats was followed by an increase in ACTH response suggesting the non-existence of cross adaptation at the endocrine levels. In contrast, the authors documented the cross adaptation at the physiological level. Chronic tail shock was shown to decrease the body weight in stress naive animals, whereas an increase in body weight was observed in animals exposed to chronic tail shock of same intensity in repeatedly immobilized rats suggesting the existence of cross adaptation between these different stressors. The authors hypothesized that the changes in the food metabolism may be responsible for cross stress adaptation as the previously immobilized animals are metabolically adapted to reduce food intake and therefore, the improved metabolic food efficiency in previously immobilized rats may possibly prevent the weight loss in chronic tail shock subjected rats. The non-existence of cross adaptation at the ACTH level and the existence of cross adaptation at the physiological level probably suggest that the central nervous system pathway controlling the pituitary adrenal axis is resistant to cross stress adaptation, and demonstration of the existence of cross adaptation may actually depend upon the type of variable measured [4].

Pol et al. [44] investigated the effects of repeated episodes of immobilization stress on the acute tail shock subjected rats. At the behavioral level, acute immobilization stress-induced behavioral deficits were significantly less marked in repeated immobilization stress-subjected rats in the hole board test. However, no changes in behavioral deficits were observed in response to acute tail shock exposure in stress naive and repeated immobilization stress subjected rats suggesting the non-existence of cross adaptation at the behavioral level in the hole board test. In contrast, the struggling period was significantly increased in the forced swim test in response to both acute immobilization and acute tail shock exposure in repeated immobilization stress subjected rats, indicating the existence of cross adaptation at the behavioral level in the swim test. It suggests that development of cross adaptation at the behavioral level may also depend on the type of behavioral test employed. In contrast, both acute exposures of immobilization or electric tail shock decreased the norepinephrine levels in different stress responsive brain regions of stress naive rats, whereas this decrease in norepinephrine levels in response to acute stressors (immobilization or shock) was not observed in repeatedly stress subjected rats. Repeated stress-induced preservation of the norepinephrine levels signifies the existence of neurochemical cross adaptation [44]. Michio and Tatsuo [40] demonstrated the phenomenon of cross stress adaptation between chronic exercise and immobilization stress in spontaneously hypertensive rats (SHR). After 4 weeks of chronic exercise stress, the increase in norepinephrine response and the rise in systolic B.P. were significantly reduced in response to acute immobilization stress in SHR. The reduction in systolic blood pressure and norepinephrine responses to novel stressor (immobilization stress) suggests the existence of cross adaptation between chronic exercise and immobilization stressor [40] (Table 1).

In an earlier study, chronic exposure of restraint stress was also shown to exert protection against the acute foot shock exposure and anxiogenic β-carboline (benzodiazepine inverse agonist) administration on the 8th day, suggesting the existence of cross stress adaptation