PERSPECTIVE

Melatonin treatment modifies heat shock protein immunolocalization in the cerebral cortex of heat-stressed rats

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Walker P. Melatonin treatment modifies heat shock protein immunolocalization in the cerebral cortex of heat-stressed rats. J Histol Histopathol Res. 2022;6(3):74-75.

ABSTRACT

Clinically, heat stress causes indications of central nervous system dysfunction. Heat shock proteins (HSPs) act as molecular chaperones, preventing cell damage. Melatonin has both curative and preventative effects on heatstroke-induced organ failure. The purpose of this study was to see if melatonin might protect the cerebral cortex from heat stressi-nduced histological and HSP60 immunoexpression changes in adult male albino rats. Forty adult male albino rats were separated into three groups: I (control), II (3 days heat stressed), and III (3 days heat stressed) (3 days heat stressed with melatonin treatment). Blood samples were taken for oxidative stress and antioxidant analyses, and cerebral cortex tissues were processed to produce slices for light microscopy evaluation.

Key Words: *Cerebral Cortex; Rats; Melatonin*

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Melatonin therapy combined with heat stress resulted in more preserved nerve tissue and an elevation in serum antioxidant marker. HSP60 immunoexpression was poor cytoplasmic in the control group, but it grew dramatically and became strong cytoplasmic after three days of heat exposure. HSP60 immunoexpression was moderate nuclear in some neurons and robust cytoplasmic in others after melatonin treatment with heat stress nuclear. Finally, melatonin alleviates heat-induced degenerative alterations in the cerebral cortex of adult male albino rats, most likely by an antioxidant action with the regulation of the HSP60 immunoexpression pattern.

As a result, more emphasis should be placed on melatonin as a preventative medication to minimize the degenerative effects of heat stress on the cerebral cortex.

The temperature has significant negative effects on bodily fitness, and adaptation to global warming and changes in thermal conditions is critical. Rising temperatures in the tropical climatic zone are disproportionately harming developing countries because they lower labour productivity and hence economic production.

Heat stroke is a potentially fatal condition defined clinically by symptoms of central nervous system dysfunction such as delirium, seizures, or coma. The most effective treatment is quick bodily cooling and support of many organ functions. Despite these efforts, many patients are at risk of developing persistent brain problems, thermoregulatory malfunction, or even death. Heat's cytotoxic impact and systemic inflammatory response caused much organ damage, and heat stroke is best avoided than treated. Food consumption and body weight increase were shown to be reduced following two weeks of daily mice exposure to 38.5°C for 60 minutes. Corticosterone and vasopressin levels in the blood, as well as catecholamine and serotonin metabolite levels in the hypothalamus, rose when mice were exposed to temperatures over 37°C for 60 minutes. Previous research revealed that heat stress inhibited autophagy and slowed auto phagosome degradation, resulting in the buildup of damaged mitochondria

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Received: May 9, 2022, Manuscript No. PULHHR-22-5093; Editor assigned: May 11, 2022, PreQC No. PULHHR-22-5093 (PQ); Reviewed: May 22,2022, QC No. PULHHR-22-5093 (Q); Revised: May 23, 2022, Manuscript No. PULHHR-22-5093 (R); Published: May 30, 2022, DOI: 10.37532/pulhhr.22.6(3).74-75

in muscle cells and the creation of an aberrant cytoplasmic environment.

Proteins must be folded in the cell to perform their biological roles, which are facilitated by molecular chaperones. Heat shock proteins (HSPs) are major molecular chaperones whose function is to mediate the proper folding of cell proteins under normal conditions, maintain their native conformations during stress conditions, and aid in the refolding of proteins that have become denatured as a result of stress. Under normal circumstances, HSP levels in the cell are sufficient to facilitate correct folding and match protein production. Under physiological stress, however, newly generated proteins unfold and surpass the ability of cellular chaperones to prevent protein aggregation, resulting in protein homeostasis loss and proteotoxicity.

HSPs are involved in cell death signalling and influence cell fate in neurological damage and disease states. Melatonin is the primary secretion of the pineal gland and has antioxidant and anti-inflammatory properties. Melatonin's therapeutic and preventive benefits on heatstroke-induced multiple organ failure syndromes in rats under general anaesthesia have previously been studied. As a result, the purpose of this study was to see if melatonin might protect the cerebral cortex from heat stress-induced histological and HSP60 Immuno expression changes, as well as serum oxidative state in adult male albino rats. Hyperthermia has been shown to be detrimental to neuronal structure and function. Neurons are especially prone to the harmful effects of misfolded and/or aggregated proteins since they are post-mitotic cells. The rats in the heat stress group were subjected to daily whole-body heating at 41°C for 1 hour. The animal housing recommendations suggested keeping rats at 22-26°C to ensure proper physiology and growth. In the current study, neuropil edoema, occluded blood vessels, and subarachnoid haemorrhage were found after three days of heat. Previous research has shown that hyperthermia beyond 40°C in humans causes a twofold increase in cerebral blood flow velocity, which may enhance vascular engorgement with cerebral edoema. Furthermore, it has been shown that hyperthermia disrupts the cerebral blood flow autoregulation systems, causing disruption of the blood-brain barrier integrity and exposing the brain to edoema. The current study found that heat stress caused degenerative alterations in the cerebral cortex, as well as a substantial rise in the serum oxidative stress indicators MDA and TNF- and a reduction in the antioxidant marker GPx. Similar findings were reported following heat stress in mouse hypothalamic neural tissue and rat cultured neurons in the form of neuronal apoptosis with shrinking fragmented nuclei, caspase-3 activation, and cytochrome c release. These findings are consistent with prior research that found heat stress might promote programmed cell death of nerve cells, resulting in brain damage, as well as worsen tissue damage induced by brain trauma, stroke, or neurotoxic medications. Previous researchers discovered that hyperthermia impaired oxidative phosphorylation by increasing mitochondrial inner membrane permeability, resulting in energy production disruption. Furthermore, rats exposed to hyperthermia above 42°C were shown to have mitochondrial abnormalities in cardiomyocytes. Furthermore, Riezman proposed that the loss of cell viability caused by heat stress might be attributable to the buildup of denatured or aggregated proteins. Melatonin protects against heat-induced degenerative alterations in the cerebral cortex of adult male albino rats, most likely by an antioxidant action with the regulation of HSP60 immunolocalization. As a result, more emphasis should be placed on melatonin and HSPs modulators as a protective medication against heat stress.