Proteomic analysis of hypothalamic injury in heatstroke rats.

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Abstract

Ischemic and oxidative damage to the hypothalamus may be associated with decreased heat tolerance as well as heatstroke formation. The present study explores the hypothalamic proteome mechanisms associated with heatstroke-mediated hypothalamic ischemia, and oxidative damage. Heatstroke rats had hypotension, hypothalamic ischemia, and lethality. In addition, they had hyperthermia and hypothalamic blood-brain-barrier disruption, oxidative stress, activated inflammation, and neuronal apoptosis and degeneration. 2DE combined LC-MS/MS revealed that heatstroke-induced ischemic injury and apoptosis were associated with upregulation of L-lactate dehydrogenase but downregulation of both dihydropyriminase-related protein and 14-3-3 Zeta isoform protein. Heat-induced blood-brain-barrier disruption might be related to upregulation of glial fibrillary acidic protein. Oxidative stress caused by heatstroke might be related to upregulation of cytosolic dehydrogenase-1. Also, heat-induced overproduction of proinflammatory cytokines might be associated with downregulation of stathmin 1. Heat-induced hypothalamic ischemia, apoptosis, injury (or upregulation of L-lactate dehydrogenase), blood-brain-barrier disruption (or upregulation of glial fibrillar acidic protein), oxidative stress (or upregulation of cytosolic dehydrogenase-1), and activated inflammation (or downregulation of stathmin 1) were all significantly reversed by whole body cooling. Our data indicate that cooling therapy improves outcomes of heatstroke by modulating hypothalamic proteome mechanisms.

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KEYWORDS:

Animal proteomics; Body cooling; Heat stroke; Hypothalamus