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Abstract
The aim of this study was to investigate the therapeutic effect of cultural mycelium extracts of Paecilomyces farinosus (CMEOPF) in rodent models of heatstroke. In the first series of experiments, urethane-anesthetized rats were randomly into the following groups and given: (a) vehicle solution or (b) CMEOPF (0.6 mg/kg body weight, i.v.) after onset of heat stroke. They were exposed to an ambient temperature of 43 oC for exactly 70 min to induce heatstroke.When the vehicle-treated rats underwent heatstroke, their survival time values were found to be 21-25 min. Immediately after the onset of heatstroke, resuscitation with CMEOPE (0.6 mg/kg body weight, i.v.) greatly improved survival (220 min). Compared with the normothermic controls, the vehicle-treated heatstroke rats displayed higher levels of core temperature, brain temperature, intracranial pressure, and brain levels of nitric oxide, glutamate, glycerol, lactat/pyruvate ratio, and dihydroxybenzoic acid (DHBA). In contrast the values of mean arterial pressure, cerebral perfusion pressure and brain levels of local blood flow, and partial pressure of oxygen were all significantly lower during heatstroke. The cerebrovascular function, the increased levels of nitric oxide metabolites, and DHBA in the brain, and hyperthermic occurring during heatstroke were significantly suppressed by CMEOPE. Secondly, unanesthetized unrestraint mice were exposed to ambient temperature of 41.2oC for 1 hour to induce heatstroke, and subsequently transferred into room temperature (24 oC). After four days, heat-treated mice had 100% mortality. In contrast, after an hour of heat treatment, the immediate CMEOPF therapy (0.6 mg/kg, i.p.) was associated with 100% survival rate after 4 days. Four hours after heat treatment, levels of cellular ischemia (e.g. glutamate, lactate-to-pyruvate ratio, and nitric oxide) and damage (e.g., glycerol) markers in the hypothalamus were found to be higher than normothermic controls. In addition, the heat-induced increased serum levels of both interleukin 1β and tumor necrosis factor-α were reduced by CMEOPF therapy. Conversely, immediate CMEOPF therapy after heat treatment, significantly reduced the elevation of cellular ischemia (e.g. glutamate, lactate-to-pyruvate ratio, and nitric oxide) and damage (e.g., glycerol) markers and splenic apoptosis, and decreased the serum levels of both excessively formed interleukin 1β and tumor necrosis factor-α, induced by heat stress. CMEOPF therapy could also enhance the low interleukin-10 level. The results indicate that CMEOPF attenuates heatstroke-induced systemic inflammation, multiorgan dysfunction and cerebrovascular dysfunction.

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