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The role of endogenous opioids in the protective effects of local sublethal hyperthermia against the progression of burn injury

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ABSTRACT

The aim of the present study was to evaluate the role of endogenous opioids in local sublethal hyperthermia-induced protection against burn injury.

Second-degree burn wounds were induced on the back of Balb/c mice. Progression of burn injury and expression of heat shock protein (HSP)-70 were evaluated after 24 h.

Both inhibition of HSP synthesis and blocking opioid receptors before applying local sublethal hyperthermia decreased the protective effects of sublethal hyperthermia against the progression of burn injury. Blocking opioid receptors attenuated induction of HSP-70 by sublethal hyperthermia.

1. Introduction

Many studies have shown that if the environmental temperature rises severely, cells grown at normal temperature will die rapidly. This phenomenon is called 'heat shock.' If these cells are exposed to a sublethal temperature before being exposed to a lethal temperature, their resistance against heat shock will be increased due to the induction of the heat shock proteins (HSPs) at the sublethal temperature. This phenomenon is regarded as 'heat shock tolerance' (thermo tolerance; Lindquist, 1986; Ellis et al., 1993).

Sublethal levels of other stresses can also induce stress tolerance when cells are exposed before being exposed to lethal stresses (Ellis et al., 1993). In addition to cells, tissues can also develop stress tolerance if they are exposed to a sublethal stress. Topping et al. (2001) have shown that if the skin is mildly warmed before being exposed to a high temperature, the thermal injury to the skin will be decreased as a result of the induction of HSPs. Also, if the myocardium undergoes a sublethal ischemia before undertaking a lethal ischemia, its resistance to the lethal ischemia will be increased. (Sommerschild and Kirkeboen, 2002).

It has been shown that in addition to HSPs, opioids and opioid receptors also have a significant role in the stress tolerance of cardiac tissue induced by ischemia and hyperthermia (Patel et al., 2002; Sommerschild and Kirkeboen, 2002; Sonneborn et al., 2004). Opioids are peptides that are mainly produced in the nervous system and are produced in lower quantities by cells of the immune system. Their receptors are on both nerve cells and immune cells (Wybran et al., 1979; Stein et al., 1990; Way et al., 2001).

Skin burn can be regarded as a lethal heat shock to the skin. Cutaneous thermal injury initiates a pathophysiological response with a significant inflammatory component that involves several classes of chemical mediators. These mediators interact in a complex manner to cause the pain and secondary tissue damage associated with burn injury (Shakespeare, 2001). Hair follicles are epidermal appendages that have an important role in the healing process after a burn injury (Dyer and Roberts, 1990). Tissue necrosis (including necrosis of hair follicles) progresses after burning, and it is not limited to the time when the burn occurs (Topping et al., 2001; Shahabi et al., 2006). If a treatment can prevent progression of necrosis after the burn injury, it can prevent the rise in the number of necrotic hair follicles after burning. Thus, the count of hair follicles 24 h after the burn injury is used to assess the skin injury (Topping et al., 2001; Shahabi et al., 2006).

We have previously shown that the endogenous opioid response plays an important role in the protective effects of

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