A POSSIBLE MECHANISM FOR NORADRENALINE INVOLVEMENT IN THE EFFECTOR RESPONSES TO COLD EXPOSURE

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It has been long known that the sympathetic nervous system mediator noradrenaline (NA) is involved in the response to cold. However, the mechanisms of NA participation in these physiological processes remain unclear. An attempt is made to summarize the results obtained in our experiments on human subjects and anaesthetized (nembutal, urethane) rats. The data concern the effect of NA on impulse activity of the central and peripheral thermosensors, thermosensitivity in human, changes in the thresholds and the intensity of cold-defense responses to cooling resulting from a specific route of NA administration, iontophoresis to the cooled skin surface, where thermoreceptors are concentrated. Data on changes in the immune response to antigen under the effect of NA in the cold and thermoneutral conditions are also reviewed. Based on the summarized data, a scheme for NA involvement in the formation of the effector (cold-defense and immune) responses to cold exposure is suggested. NA affects both the peripheral and central thermosensors. A decrease in the activity and sensitivity of the high frequency skin cold receptors presumably results in a decrease in cold sensation. An increase in the static and dynamic activities of the low frequency cold receptors and a moderate increase in the sensitivity of the neurons of the medial preoptic area of the hypothalamus in the low temperature range may produce a decrease in the threshold and an intensification of the thermoregulatory responses during cooling, as well as a considerable stimulation of the antigen binding function of the immune response during deep cooling, which is suppressed in the absence of NA. Intensification of the effector thermoregulatory and immune responses may be also related to the direct NA effect on the effector organs and tissues. The regulatory effect of NA on the effector (thermal-defense and immune) responses appears to be dependent on many factors: its relative concentration in the periphery and center, in the brain, and skin, where the bulk of the thermosensors is localized, and blood supplying the effector tissues.

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