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HYPERPLASIA OF BROWN ADIPOSE TISSUE AND GLUCOSE  
TRANSPORTER EXPRESSION INDUCED BY  
CHRONIC ADMINISTRATION OF CATECHOLAMINES IN RATS

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Brown adipose tissue (BAT) is the major site for metabolic heat production during cold exposure and hyperphagia. It has been documented that chronic cold exposure activates the sympathetic nerves entering BAT and produces hyperplasia of this tissue and a marked increase in the amounts of some specific proteins such as mitochondrial uncoupling protein (UCP) and glucose transporter (GLUT4). To clarify the role of noradrenaline in the cold-induced changes of BAT, in this study, I examined effects of chronic administration of noradrenalin and related agonists on BAT in rats.

When noradrenaline was given continuously for 10 days using an osmotic mini pump, the contents of protein and DNA, and cytochrome c oxidase activity were significantly increased in BAT. Furthermore, the amounts of UCP and GLUT4 estimated by respective specific antibodies were also increased in BAT, but not in other tissues such as white adipose tissue, skeletal muscle and heart. These changes in BAT were quite similar to those seen after cold acclimation. The chronic administration of isoproterenol, a  $\beta$ -adrenergic agonist, gave rise to changes similar to those induced by noradrenaline, but phenylephrine, an  $\alpha$ -adrenergic agonist, had no noticeable effect.

In parallel with the increase in GLUT4, in vivo glucose utilization of BAT was much increased in rats treated with noradrenaline and isoproterenol.

All these results indicate that the trophic effect of sympathetic nerves on BAT is mediated by the  $\beta$ -adrenergic action of noradrenaline.