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# Body fat and the activity of the autonomic nervous system

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## Abstract

The cause of most cases of human obesity is unknown. Specific alterations in the activity of the autonomic nervous system may mediate and perhaps cause obesity in animal models. We therefore looked for alterations in autonomic activity in human obesity. Fifty-six healthy men with various percentages of body fat underwent autonomic testing while at rest. Significant correlations were found between the percentage of body fat and the variation in the R-R interval after beta-adrenergic blockade (r = -0.30, P less than 0.03), the heart rate (r = 0.30, P less than 0.03), the plasma norepinephrine concentration (r = -0.30, P less than 0.05), the plasma epinephrine concentration (r = -0.49, P less than 0.001), and the pupillary latency period (r = 0.39, P less than 0.01). Each of these variables reflects the activity of the sympathetic nervous system or parasympathetic nervous system or both. Depressions in sympathetic and parasympathetic activity were significantly but weakly associated with increasing percentages of body fat. These associations indicate that in obese persons, autonomic changes, though not necessarily causal, involve several organ systems. We suggest that autonomic alterations are important in human obesity, as they are in animal obesity. A disordered homeostatic mechanism may promote excessive storage of energy by decreasing sympathetic activity, while defending against weight gain by decreasing parasympathetic activity. The use of autonomic profiles holds promise for classifying human obesity and identifying obese patients at increased risk for various disorders.



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