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Mechanism of Sarcopenia Induced by Inappropriate Sodium Intake

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Summary

In the present study, we have investigated how catabolism is induced by high salt intake which leads to circulatory changes and sarcopenia during the progression of CKD. Specifically, we have investigated the specific roles of high salt intake, renin-angiotensin system and sympathetic nervous system in high salt-induced catabolism and associated changes. Data have shown that high salt intake induces catabolism and circulatory hemodynamic depression in normal rats, i.e., a typical aestivation response. The central metabolic change in this response is the activation of the urea cycle in the liver, which is associated with arginase activation. These responses were suppressed by renal sympathectomy, suggesting a significant involvement of the sympathetic nervous system. On the other hand, in Dahl salt-sensitive rats, dipping changes in blood pressure was absent in proportion to the degree of kidney damage. Furthermore, the catabolism-induced aestivation response observed in normal animals was absent, but this response was restored by renal denervation. In addition, renal denervation markedly improved survival rate. Specific roles of chymase and mineralocorticoid receptor in the development of salt-sensitive hypertension and its associated renal damage were also shown. Specific relationship between catabolism and sarcopenia in the setting of renal injury was also examined in detail. Data have indicated that the CKD model rats may prevent lethal dehydration by producing and accumulating inorganic and organic osmolytes in the body. In other words, the urine concentrating capacity of the kidney is impaired and the body is constantly losing water, but the body retains fluid by triggering extrarenal compensatory body responses. Such a response may be induced by the production of osmotic substances such as urea and osmolytes in the liver, which requires a continuous supply of endogenous energy and nitrogen from skeletal muscle to support this metabolic demand. In conclusion, biological mechanisms to cope with fluid loss may be an important factor in the pathogenesis of sarcopenia associated with renal failure.