Thesis Title	Effects of Potassium Depletion and Repletion on Contractile Properties of Rat Skeletal Muscle
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ABSTRACT

This study was aimed to determine the functional defects of skeletal muscle in chronic hypokalemic rats and evaluate a possible association of severity of hypokalemia with sexes. Chronic K^+-depletion was induced in young adult Sprague-Dawley rats by giving K^+-deficient diet for up to 120 days. Control animals were paired-fed with standard diet in the same amount as that consumed by the K^+-depleted group. During K^+-depletion, animals become anorexia, consume less amount of diet, decrease weight gain and loss weight particularly at the late phase of experiment (8^{th} week). Polydypsia and polyuria which indicated impairment of urinary concentrating ability also occurred at 8^{th} week of K^+-deficient diet. Male animal was prone to develop this defect as the symptoms appeared earlier and more severe than those in female. K^+-deficiency caused a rapid decrease of plasma K^+ concentration. After rapid drop during the early phase of K^+-depletion, plasma K^+ was maintained at its maximal low level of 1.9-2.4 mEq/l. Plasma Na^+ concentration was not significantly altered during K^+-depletion. Similar to plasma K^+ concentration, urine K^+ concentration showed a rapid decrease during the early phase of K^+-depletion. Evidently the decreasing rate of plasma K^+ and urine K^+ in K^+-depleted male was faster than those in female.

The reduction in plasma K^+ concentration was accompanied with a marked decrease of skeletal muscle K^+ content including soleus (SOL), extensor digitorum longus (EDL) and gastrocnemius. At the end of experiment, muscle K^+ content in K^+-depleted rats were approximately 40-50% of their control values. In contrast to the decrease of muscle K^+ content, muscle Na^+ content increased. With marked changes of K^+ and

Na^+ contents, muscle paralysis was not observed. However, four out of ten males died after 70 days whereas the remained test animals were able to survive up to 110 days with poor conditions as general weakness, polyuria and polydypsia before sacrifice. In contrast, all female animals could survive up to the end of experiment at 120 days. Plasma creatinine kinase activity, an indicator of muscle membrane damage was increased approximately 40 and 15% in K^+-depleted male and female, respectively. Apparently there was sex differences in response to K^+-depletion. When K^+-depleted rats of both sexes were repleted K^+ with standard diet for 14 days, all abnormalities including body weight gain, food consumption, water intake, urine output, plasma K^+ concentration were completely returned to their normal values. The results indicated that changes were specific to K^+ loss and all were reversible.

Contractile properties of isolated soleus muscle was determined by incubating with standard Krebs-Ringer's bicarbonated buffer pH 7.4 at 30°c. Soleus obtained from the K^+-depleted male was significantly smaller than that from the restricted-diet control. Its maximum isometric twitch and tetanic tension was markedly reduced. It was reduced to approximately 40% of control. In K^+-depleted female, these contractile tensions were also reduced but no statistical significance. Therefore, there was sex difference in contractile properties in K^+-depletion. The reduction of contractile tensions in both male and female K^+-depleted rats were further enhanced by adding insulin. These abnormalities of skeletal muscle function were suggested to be due to the defect in the muscle itself. Inexcitabilities as well as atrophy of the fibers might account for the impaired function. Administration of insulin might aggravate the inexcitability of the cells. K^+-repletion by giving standard diet to K^+-depleted rats for 2 weeks could completely reverse all contractile defects.

Therefore, there was sex differences in response to K^+-depletion. Male had more susceptibility to K^+-depletion. All abnormalities observed were reversible after K^+-repletion.