Summary  Relative left ventricle weight was significantly increased irrespective of blood pressure in normotensive Wistar-Kyoto rats given 1% NaCl in drinking water for two months. The present result first suggested that chronic excess salt intake might be one of possible factors inducing cardiac hypertrophy.

Introduction  Although cardiac hypertrophy has been traditionally thought to be a compensatory physiological response of the heart, evidence has been accumulated implicating that cardiac hypertrophy may also result from additional factors independent of the increased afterload. Some genetical factors has been suggested from the studies in young spontaneously hypertensive rats (SHR) of pre-hypertensive stage and from our recent study in cultured vascular smooth muscle cells from SHR. Results of treating SHR with hydralazine are also suggestive of some factor responsible for cardiac hypertrophy.

In search of other possible factors related to heart weight, we tested long-term effect of excess sodium intake in the present study, because the possibility of sodium intake to increase in heart weight has been suggested by our comparative study between the Japanese and the New Zealanders in which relative heart weight was greater in the Japanese than in the latter.

Materials and methods  Fourteen female Wistar-Kyoto rats (WKY) at the age of three month were randomly divided into two groups after measuring blood pressure and body weight. One group (eight in number) was given 1% sodium chloride in drinking water and the other tap water as a control. After two months all rats were checked for blood pressure, body weight and hematocrit and sacrificed to measure the weight of the ventricles. Blood pressure was measured by tail pulse-pickup method.

Results  Changes in blood pressure were in the same degree in both groups; increments were from 116±3 mmHg (±SE) to 126±3 in the salt group and from 115±4 to 124±2 in the water group. Body weight increased from 188±5 (g) to 212±5 and from 186±6 to 225±5 in the salt and the water groups, respectively, being relatively smaller in the former although the difference was not significant. Hematocrit was equal between the groups, 46 percent.

Relative weights of both ventricles were slight but not significantly heavier in the salt group, 359±9 mg per 100 gram body weight, than in the water group, 335±11. Relative weights of the left ventricle were 278±6 and 253±6, respectively, and significantly (p<0.05) greater in the salt group.

Discussions  In WKY, blood pressure remains relatively constant throughout the life and the heart weight increases closely with increasing body weight. The present results have therefore implicated chronic excessive salt intake may increase relative weight of the left ventricle, independently of blood pressure.

The mechanism is unclear at the present time but may possibly be the same as one which induced relative increase in kidney weight in the salt-loaded unilaterally nephrectonized WKY in our previous experiment. Since there was no difference in hematocrit between the groups although fluid intake was far greater in the salt group, a hyperdynamic state in order to excrete more fluid from circulation might be responsible for the relative increase in the left ventricle in salt-loaded rats.

The present result, anyhow first suggested that ingestion of chronic excessive salt might be a possible factor for cardiac hypertrophy and that might explain the increased relative heart weight in the Japanese compared to the New Zealanders.