51) Genetical Disposition to Cardiac Hypertrophy in Rats. H. Tanase^o, R. Horie^o, M. Kihara^{*}, Y. Yamori^{*o} and W. Lovenberg^o. NHLBI^o, NIH, Bethesda, MD, USA and Department of Pathology^{*}, Shimane Medical University, Izumo 693, Japan.

INTRODUCTION Concerning the question as to whether or not cardiac hypertrophy in hypertension is caused secondarily only to hypertension, definite data are not available although many observations were made in acute or chronic experiments. In the present study, the development of cardiac hypertrophy and the thickening of the aorta in relation to blood pressure were investigated in 15 different strains of rats.

MATERIALS AND METHODS Strains of rats used were WKY, SHR, SHRSP, OM, RHA, A₂₈₈₀₇, NSD, F₃₄₄, MR, LOU, ODU, CAR, ACI, M₅₂₀ and MNR which were maintained at NIH. Experimental group consisted of 10 male rats each for the following two experiments. One was a chronological accurate measurement of body weight, blood pressure, heart weight and pulse rate in WKY, SHR and SHRSP at the age of 4, 8, 12 and 16 weeks. The other was the recording of the above-mentioned items at the age of 10 weeks in all these strains of rats. As previously confirmed by Y. Yamori and R. Horie, thickening of the left ventricular wall can be detected accurately by a simple uniformed measurement of the weight of the heart itself. Thus, heart weight was measured as an indicator for cardiac hypertrophy and both absolute values and relative values (heart weight / body weight) were analyzed statistically. In the present study, the weight of the aorta which was uniformly extirpated was also measured in all strains of rats.

RESULTS (*Experiment I*) (a) Before the development of hypertension, heart weight in SHR was the heaviest ($p < 0.05$) in comparison with findings in SHRSP and WKY. In contrast, the weight of the aorta showed no significant difference. (b) After the development of hypertension, the SHRSP with the highest blood pressure showed heaviest weights of the heart and the aorta, and weights in SHR with mild hypertension were next. These weights were lightest in normotensive WKY. These differences were all significant ($p < 0.05$).

(*Experiment II*) (a) Relationship between blood pressure and heart weight in 15 strains of rats was assessed as: 1) hypertensive group (e.g. OM) and 2) normotensive group (e.g. M₅₂₀). In the former, the development of hypertrophy and thickening of the aorta was in parallel to the development of hypertension. Aortic thickening is more likely to be influenced by hypertension itself. Some normotensive groups showed also cardiac hypertrophy despite a normal blood pressure and there was no correlation between blood pressure and the development of hypertrophy or thickening. (b) The variance analysis of absolute and relative weights of the heart or aorta in 15 strains showed that the inter-strain differences were statistically significant, and the degree of genetic determination was 50-60%. The analysis on the weight of the aorta is shown in the Table.

CONCLUSION Cardiac hypertrophy in hypertension is not caused only by hypertension and genetical disposition indifferent to hypertension might also be involved. The degree of genetic determination was around 50%.

Aorta Weight	Source of Variation	Degree of Freedom	Mean Square	F Ratio	Degree of Genetic Determination
Absolute	Between Strains	14	93762	26.7*	0.562 ± 0.069
	Within Strain	136	3512		
Relative	Between Strains	14	9327	47.6*	0.670 ± 0.058
	Within Strain	136	196		

* : $p < 0.01$