Correlation Analysis of Blood Pressure and Platelet Aggregation in SHRSP, WKY, F₁ and Backcross generations. Satoshi Mashiko, Takeshi Onda, Megumi Hamano, Isao Tomita and *Takako Tomita. University of Shizuoka School of Pharmaceutical Sciences, *Graduate School of Health Sciences, Shizuoka, 422, Japan.

Hypofunction of platelets observed in SHRSP at developmental ages of hypertension is neither secondary to the circulation of degranulated platelets (Umegaki et al., Blut 1986), nor rectified by a hypotensive treatment (Umegaki et al., Jpn.Heart J.). The mechanism underlying the hypofunctions seems to be defective protein (p47) phosphorylation which is mediated by protein kinase C (Tomita et al., Hypertens.1989). We also previously reported that the magnitude of hypertension and the degree of platelet hypofunction were inversely correlated in WKY, SHRSP and F₁. This study was undertaken to examine further the genetic association of the hypofunction using male WKY, SHRSP, F₁ and backcross generations.

WKY females were mated to SHRSP males to produce F₁ hybrids. Males of the F₁ hybrids were backcrossed to WKY females to produce backcross generation FW, while female F₁ were mated to male SHRSP to produce FS backcross. They were raised and maintained in our laboratory. SHRSP, FS, F₁, FW and WKY at 16-18 weeks of age were used in this experiment. Systolic blood pressure was measured by a tail-pulse pick-up method in unanesthetized rats after warming them approximately 10 minutes in a chamber maintained at 37°C. Washed rat platelets was prepared as described elsewhere (Tomita et al., Stroke 1984). Platelet aggregation was measured by a turbidimetric method using AGGRETEC TE-500 (ERMA, Tokyo, Japan). Platelet (4×10^8 cells/ml) was stimulated for 3 minutes with thrombin (0.14 U/ml) at 37°C in the presence of 1.5mM Ca²⁺.

Blood pressure of the F₁ hybrids is intermediate between SHRSP and WKY, and that of both backcross generations is also intermediate between F₁ hybrids and respective parental strains [blood pressure (mmHg), mean±S.E.(n): WKY: 146±1 (36), FW: 167±2 (22), F₁: 183±1 (38), FS: 220±3 (22), SHRSP: 235±3 (17)]. The distribution of blood pressure was continuous in each generation, and backcross generations were not likely to be 1:1 segregation. Ratios of heart to body weight ($\times 10^3$) were WKY: 3.12±0.02 (34), FW: 3.25±0.05 (22), F₁: 3.33±0.03 (35), FS: 3.64±0.05 (22) and SHRSP: 4.01±0.05 (17). A positive correlation ($r=0.86$, $n=129$) was observed between heart to body weight ratios and blood pressure. Thrombin-induced maximum platelet aggregation (%) was WKY: 66±1 (33), FW: 61±2 (22), F₁: 47±2 (32), FS: 31±2 (22) and SHRSP: 22±1 (17). There was a strong inverse correlation between platelet aggregation and blood pressure ($r=-0.87$, $n=127$). The phenotypic segregation ratio cannot be regarded as 1:1 in each backcross generation. These results suggest the possibility that platelet hypoaggregability is due to the pleiotropic effect of hypertensive genes, or that genes controlling these two characters are closely linked each other.