Differential Effects of Antihypertensive Agents on Proliferation of Vascular Smooth Muscle Cells from Spontaneously Hypertensive Rats. M. Horiguchi, H. Uchida, S. Kuriyama, T. Hashimoto and O. Sakai. Second Department of Internal Medicine, Jikei University School of Medicine, Tokyo 105.

We have previously shown that Ca-antagonists and α-blockers substantially inhibit the cellular proliferation of cultured rat vascular smooth muscle cells (VSMC). This study explored whether these inhibitory effects on cellular proliferation differ between cultured VSMC from spontaneously hypertensive rats (SHR) and Wistar-Kyoto rats (WKY).

Materials and Methods: Cultured VSMC were derived from thoracic arteries of 12-week-old male SHR and WKY. (1) For the cell number determination, VSMC were cultured in DMEM with 10% FCS in the presence and absence of bunazosin (Bun) or nifedipine (Nif). 5-7 days after, the cells were counted with a coulter counter. (2) 1μCi/ml of 3H-thymidine with or without the agents was added to the VSMC, and the incorporation of the tracer was measured with a scintillation counter. (3) The intracellular water volume (ICWV) was obtained from the equilibrium distribution of 3-O-methyl-D-glucose 14C.

Results: (1) The VSMC number was greater in SHR than that in WKY, in 10% FCS. In the presence of either Nif or Bun, the cell number of either WKY or SHR were reduced. (2) The uptake of the 3H-thymidine was reduced substantially by either Nif of Bun, and the magnitude of these inhibitory effects was more pronounced for SHR cells than WKY cells. (3) Neither Nif nor Bun exerted any affect on ICWV of either SHR VSMC or WKY VSMC.

Discussion: It is concluded that SHR VSMC grow much faster than WKY VSMC and that this abnormality is innate to the SHR cells. It is also concluded that both Ca-antagonists and α-blockers exerted a substantial inhibitory effect on cellular proliferation of the cultured VSMC of either SHR or WKY. Furthermore, the greater inhibition of proliferation in the SHR VSMC suggests that Ca mediated-and/or α-receptor mediated processes of cellular proliferation of SHR could differ from that of WKY and that these abnormalities may contribute to the hyperproliferative changes of VSMC in this model.