chloroplasts, DNA-dependent RNA synthesis is required. By itself, this finding would not necessarily imply that extra-nuclear DNA is involved. However, since the chloroplast ultra-violet targets appear to be DNA-like and since an enormous increase in the number of plastids takes place in enucleate fragments of *Acetabularia*[12], it seems most probable that cytoplasmic or chloroplast DNA is involved.

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Note added in proof. Since this report was submitted, there have been several published reports on the occurrence of chloroplast DNA. (Kirk, J. T. O., *Biochem. J.*, **88**, 45P (1963). Chun, Edward H. L., Vaughan, Maurice H., jun., and Rich, Alexander, *J. Mol. Biol.*, **7**, 130 (1963). Sager, Ruth, and Ishida, M. R., *Proc. U.S. Nat. Acad. Sci.*, **50**, 725 (1963).)

D. R. McCalla
Robert K. Allan

Research Unit for Biochemistry,
Biophysics and Molecular Biology,
McMaster University,
Hamilton, Ontario, Canada.

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PHYSIOLOGY

Increase in Sodium Content in the Arterial Walls during Experimental Hypertension

Evidence has been obtained that the addition of sodium to the diet of animals in which hypertension has been induced aggravates the hypertension and the renal lesions. Similar observations were made in man. Moreover, a salt-poor diet or the administration of natridiuretics lower experimental and clinical hypertension. It has already been reported[1,2] that, in rats rendered hypertensive, an accumulation of sodium in the arterial walls is frequently observed.

The results presented here provide evidence that, in rabbits rendered hypertensive by partial ligation of the renal arteries[3], hypertension is accompanied by a metabolic trouble of the arterial wall which is objectivated by a progressive increase in the sodium content in the arterial walls without significant changes of their potassium-level.

The experiments were performed in adult male rabbits, weighing 2,500–3,000 g. The animals were divided into four groups:

Group I: Control group. 10 rabbits. Maximal arterial blood pressure: 75–90 mm mercury.

Group II: 13 rabbits. Hypertension induced by bilateral partial stenosis of the renal arteries. Maximal arterial blood pressure: 130–150 mm mercury. Killed between 20th and 25th day after the operation.

Group III: 7 rabbits. Hypertension induced as above. Maximal arterial blood pressure: 135–150 mm mercury. Killed between the 55th and 65th day after the operation.

Group IV: 5 rabbits. Hypertension induced by partial bilateral stenosis of the renal arteries and additional hypodermic injection of 5 mg of DOCA every 2 days. Maximal arterial blood pressure: 140–150 mm mercury. Killed between the 20th and 25th day after the operation.

The rabbits were maintained on a standard diet containing 2·2 per cent sodium chloride and received tap water *ad libitum*.

The partial ligation of renal arteries was performed according to the method described by Pickering and Prinzmetal[3] and the blood pressure measured in the auricular artery according to the method of Grant and Rothschild[4]. The electrolytes in the mesenteric arteries and in the abdominal aorta were determined according to the method of Koletsky *et al.*[1] by flame photometry and the results are expressed in m.equiv./100 g of defatted and dried tissue.

The results are presented in Table 1.

Table 1. Sodium and Potassium expressed in m.equiv./100 g of Arterial Tissue

	Mesenteric arteries		Aorta	
	Na	K	Na	K
Group I (10 rabbits)	8·4 ± 2·2	2·3 ± 0·2	7 ± 0·5	1·6 ± 0·06
Group II (13 rabbits)	27·8 ± 15·2	1·1 ± 0·00	17·5 ± 2·3	1·7 ± 0·1
Group III (7 rabbits)	52·1 ± 13	1·4 ± 0·1	51·6 ± 31·8	1·4 ± 0·00
Group IV	26·1 ± 3·8	2·3 ± 0·00	15·3 ± 4·1	1·7 ± 0·00

It is obvious from these results that the sodium content of the mesenteric arteries and of the aorta increases significantly in animals rendered hypertensive by partial ligation of the renal arteries, and that this increase is a function of the duration of the disease.

The potassium content in the arterial walls remains practically unchanged. There is no significant difference between the group of animals rendered hypertensive by simple stenosis of the renal arteries, and those receiving additional injection of DOCA.

Three main points should be stressed:

(1) The increase in the parietal sodium is not correlated to the degree of hypertension and increases with the duration of the hypertension. (2) The differences in the content of the small arteries (mesenteric arteries) and the aorta are not particularly significant. (3) Additional administration of DOCA to animals rendered hypertensive by bilateral arterial stenosis does not increase the sodium arterial retention.

Investigations are now in progress to establish whether the phenomenon of the sodium accumulation in the arterial walls in hypertensive rabbits is related to a direct effect of angiotensine or of aldosterone released by angiotensine or to a different process. Our present results suggest, nevertheless, that the accumulation of sodium in the arteries affects the sensitivity of the intraparietal baroreceptors to pressure changes.

Bernard Halpern Philippe Meyer
Paul Milliez Gilbert Lagrue

Chaire de Médecine Expérimentale,
Collège de France,
and
Centre de Recherches sur l'Hypertension Artérielle,
Paris.

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Sodium Exchange in Rat Muscle

Attempts to measure the rate of exchange of labelled sodium in isolated mammalian muscle have been beset with numerous difficulties. The fibre sodium may show a marked increase[1], the fall in radioactivity of the muscle during a washout may not be linear when plotted on semi-logarithmic paper[2], and the thickness of the tissue