D'autre part, il n'est nullement exclu que les augmentations d'activité enzymatique dans ces deux cas répondent à des processus indépendants.[9]

CONCLUSIONS

L'irradiation par rayonnement γ entraîne, dans un délai de 24 hr, un accroissement des activités enzymatiques microsomiales d'hydroxylation du BaP. Cet effet paraît peu sensible à la cystamine. D'autre part, l'irradiation n'inhibe pas le phénomène global d'induction enzymatique de ces enzymes: l'horaire d'irradiation ni dans certaines limites, la dose ne jouent de role marquant. Cependant un effet inhibiteur partiel ne peut être exclu.

Service de Chimie Médicale, Toxicologie & Hygiène,
Universite de Liège,
151 *Bd. de la Constitution,*
Liège, Belgium

P. DELWAIDE
D. RONDIA
C. HEUSGHEM

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Inhibiting effect of angiotensin on potassium accumulation of adrenal cortex

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THE MAIN sites of effects of angiotensin are: smooth muscle cells of the vascular walls, renal tubular cells and the adrenal cortex. Its vasoconstrictor effect is associated with the decrease of potassium content and increase of sodium content in the aorta cellular phase *in vivo*[1] and *in vitro*[2] but there are also data for its opposite effect.[3]

Large doses of angiotensin inhibit tubular reabsorption of sodium *in vivo*,[4] and inhibit uphill sodium movements in tissue slices of kidney as well.[5] From these data angiotensin seems to inhibit active sodium transport at its sites of effect.

The regulation of aldosterone secretion is not quite clarified yet, but it is established that the following stimuli have a direct effect on the adrenal which specifically stimulate aldosterone secretion: angiotensin, increase of potassium or decrease of sodium in the extracellular surroundings.[6] We supposed that angiotensin would inhibit active sodium–potassium transport in the adrenal cortex too; thereby decreasing the extracellular Na/K ratio, which condition is favourable for increasing aldosterone production.

A suitable method for testing active ion transport of tissues is the study of potassium reaccumulation. As we did not find any data in the literature concerning the potassium accumulation of adrenal cortex, we used the method described for liver[7] after some modification.

We studied the effect of angiotensin on the potassium accumulation of adrenal cortex and liver slices, and the effect of ouabain to prove by its inhibiting activity the presence of active ion transport.

The adrenals and livers from hogs were obtained after slaughter and were transported from the slaughter house to the laboratory in ice. The time between the death of the animal and the beginning of experiment was 30–60 min. The adrenal cortex and the liver were cut into 0·35 mm slices with a mechanical tissue chopper. From each adrenal cortex or piece of liver we obtained five samples. Five samples made a block and were treated in random order according to the following: all tubes were incubated for 60 min in a water bath at 0–0·5° in the air, shaking the tubes every 10 min. Tubes of Group 1 were incubated only in cold water bath, the tissue slices were blotted and their wet weight determined. Tissue slices of groups 2–5 were slightly blotted after cold incubation and placed in Warburg flasks containing 2·5 ml buffer solution. The buffer was a modified Krebs' I. bicarbonate buffer[8] containing 119·7 m-equiv. of Na, 18·8 m-equiv. of K and 111·5 m-equiv. of Cl/1. Group 2: control, containing only buffer solution, group 3: 100 μg/flask angiotensin* added, group 4: 500 μg/flask angiotensin added, Group 5: 2×10^{-4} M ouabain added. Groups 2–5 were incubated in Warburg apparatus for 60 min at 37° flushing through 95 per cent oxygen and 5 per cent carbon dioxide. Our earlier experiments have shown fast inactivation of angiotensin during incubation in the presence of adrenal cortex or liver; therefore we added 100 μg angiotensin to Group 3 and 500 μg to Group 4 at 15 min, 30 min and 45 min of incubation in 0·25 ml of buffer solution. We added to Group 1 0·25 ml of buffer solution and to Group 5 0·25 ml of 2×10^{-4} M ouabain containing buffer solution every 15 min.

After incubation the tissue slices were blotted, their wet weight determined, and then put into air oven (110°) for 24 hr. After the dry weight was determined, the tissue slices were digested in concentrated nitric acid and potassium was measured by flame photometry. The potassium content of the slices was expressed as m-equiv./kg of dry weight.

The statistical analysis of the experiment was carried out with the two way analysis of variance. The significance between the treatments was determined by the multiple comparison method of Dunn.[9]

The results show (Table 1) that ouabain and the larger dose of angiotensin inhibit the potassium accumulation of the adrenal cortex ($P < 0{\cdot}01$). The effect of the smaller dose of angiotensin was not significant, but the potassium accumulation of this group was smaller than that of the control and larger than that incubated with the larger dose of angiotensin.

TABLE 1. EFFECT OF ANGIOTENSIN AND OUABAIN ON POTASSIUM ACCUMULATION OF ADRENAL CORTEX AND LIVER

Group	Conditions of incubation	Tissue potassium content m-equiv./kg of dry weight	
		(A) Adrenal cortex $n = 11$	(B) Liver $n = 6$
1	Cold only	111·3 ± 5·9	115·4 ± 3·2
2	Buffer control	156·5 ± 6·5	158·0 ± 12·0
3	+100 μg/flask angiotensin 4x	139·3 ± 7·8	149·4 ± 12·7
4	+500 μg/flask angiotensin 4x	121·3 ± 5·3	136·5 ± 3·6
5	2×10^{-4}M ouabain	102·2 ± 6·3	114·1 ± 7·7

2A vs. 4A $P < 0{\cdot}01$ 2B vs. 4B $P > 0{\cdot}05$
2A vs. 5A $P < 0{\cdot}01$ 2B vs. 5B $P < 0{\cdot}01$

* All values are means ± S.E.

The potassium accumulation of the liver was inhibited by ouabain only ($P < 0{\cdot}01$), the effect of angiotensin was not significant.

* Val^5-angiotensin-II-asp^1-amide, Hypertensin® CIBA.

Our experiments have clearly shown the inhibiting effect of angiotensin on the potassium accumulation of adrenal cortex *in vitro*. As angiotensin has similar effect on aorta,[1] smooth muscle,[2] kidney[5] and uterus,[10] the inhibition of active sodium transport may be the common mechanism of effect of angiotensin on its different sites of action.

If the inhibiting effect of angiotensin on active sodium–potassium transport of adrenal cortex was valid *in vivo*, angiotensin would exert its effect on aldosterone secretion by changing the ionic gradient between cells and surroundings, since the decrease of sodium and increase of potassium concentration in the extracellular space is favourable for aldosterone secretion. Our hypothesis that angiotensin influences aldosterone secretion by the aforementioned mechanism is strengthened by the experiments of Kaplan:[11] the effect of angiotensin on the biosynthesis of aldosterone was potentiated by an amount of potassium which did not by itself augment formation of aldosterone. Our supposition is also supported by Muller's data,[12] which demonstrated that angiotensin and potassium act on aldosterone secretion at the same site of steroid synthesis.

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Department of Pathophysiology,
Institute of Experimental Medicine,
Hungarian Academy of Sciences,
Budapest, Hungary

KATHLEEN SZ. SZALAY

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