It indicates that the detailed mechanism of protein synthesis may be diverse in the various genera of bacteria.

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Conversion of Tritiated-18-hydroxy-corticosterone to Aldosterone by Slices of Human Cortico-adrenal Gland and Adrenal Tumour

PROGESTERONE (pregn-4-ene-3,20-dione), corticosterone (11β,21-dihydroxypregn-4-ene-3,20-dione) and deoxycorticosterone (21-hydroxypregn-4-ene-3,20-dione) have been shown to be precursors of aldosterone[1–5]. Aldosterone (11β,21-dihydroxypregn-4-ene-3,20-dione-18-al) is produced in the glomerulosa zona of the adrenal cortex[6–8]. It could be assumed that the biogenesis of aldosterone was preceded by hydroxylation of corticosterone in C_{18}. Nicolis and Ulick[9] obtained a slight yield of aldosterone (0·2 per cent) by incubation of tritiated-18-hydroxy-corticosterone (11β,18,21-trihydroxypregn-4-ene-3,20-dione) with bull-frog adrenal tissue and beef zona glomerulosa tissue. Stachenko and Giroud[10] considered that the cyclic 18→20 hemiketal form of 18-hydroxy-corticosterone[11] was responsible for this lack of conversion of 18-hydroxy-corticosterone to aldosterone.

This work demonstrates that human cortico-adrenal tissue is capable of converting tritiated-18-hydroxy-corticosterone (kindly supplied by Dr. S. Ulick) to tritiated-aldosterone with a considerable yield. The adrenal cortex and adrenal tumour of a patient with primary hyperaldosteronism[12] were removed. The tumour was separated from the adrenal gland tissue and slices taken from two parts. 0·6-g slices of adrenal tumour and 0·7 g of cortico-adrenal tissue were incubated with 10 ml. of Krebs-phosphate solution (*p*H 7·4)[13], 5 mg of diphosphopyridine nucleotide (DPN) and 50 mg of sodium fumarate. 60,000 c.p.m. of tritiated-18-hydroxy-corticosterone were added to the first incubation and 50,000 to the second. After incubation at 37° C for 3 h, the slices were ground in a Waring blender and the proteins precipitated with 25 ml. of 96 per cent ethanol. The supernatant was evaporated to dryness, the residue dissolved in 25 ml. of 90 per cent ethanol, left at − 10° C for three days and centrifuged. The supernatant was evaporated to dryness, the residue dissolved in 25 ml. of 80 per cent methanol, left for three days at − 10° C and centrifuged. The supernatant was evaporated to dryness, the residue dissolved in 20 ml. of water and extracted three times with 1 vol. of dichloromethane. The dry dichloromethane extract was chromatographed successively in the chloroform/formamide, toluene–ethyl-acetate/methanol–water (9–1/5–5) and iso-octane–*ter*-butyl alcohol–water (10–5–9) systems. After elution of the aldosterone zone in the third chromatogram, the radioactivity was measured in an automatic liquid scintillator (Tri-carb model 314 *EX*, Packard Automatic Instrument Co.). Part of this elution was acetylated and the product obtained had the same mobility as synthetic aldosterone diacetate in the iso-octane–toluene/methanol-water system (1–4/5–5).

Quantitative evaluation of 1,2-tritiated-aldosterone was determined by a method derived from that of Kliman and Peterson[14].

Table 1 shows the percentage of transfer of tritiated-18-hydroxy-corticosterone to aldosterone after incubation.

Table 1. PERCENTAGE OF TRANSFORMATION OF 18-HYDROXY-CORTICOSTERONE TO ALDOSTERONE BY HUMAN CORTICO-ADRENAL AND ADRENAL TUMOUR TISSUES

	^{3}H-18-hydroxy-corticosterone (c.p.m. incubated)	^{3}H-aldosterone produced (c.p.m.)	Yield (%)
Cortico-adrenal tissue	50,000	5,300	10·60
Adrenal tumour tissue	60,000	2,300	3·85

Human cortico-adrenal tissue, as well as adrenal tumour of Conn's syndrome, is capable of converting tritiated-18-hydroxy-corticosterone to tritiated-aldosterone with yield of 10·60 and 4·33 per cent, respectively, for the quantities incubated. Thus, the biogenesis of aldosterone appears to occur in the following sequence: cholesterol → progesterone → deoxy-corticosterone → corticosterone → 18-hydroxy-corticosterone → aldosterone.

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Inhibition of Phenylalanine Incorporation by Polyuridylic Acid

ADDITION of polyuridylic acid (poly U) to cell-free systems prepared from a number of tissues results in enhanced incorporation of phenylalanine into polypeptide material[1–4]. Arnstein *et al.*[2] noted that, when poly U was added to a reticulocyte ribosome system, the incorporation of other amino-acids was not inhibited and they concluded that natural messenger RNA (*m*-RNA) was not displaced from the ribosomes by the poly U. They questioned whether the poly U effect on phenylalanine incorporation had any physiological significance. Weinstein *et al.*[5] later showed that incorporation of some amino-acids was inhibited when poly U was added to reticulocyte ribosomes. The present report concerns an instance when poly U, added to rat liver polysomes prepared in the absence of magnesium, actually inhibited phenylalanine incorporation. It is argued that this anomalous occurrence, paradoxically, strengthens the concept that poly U stimulation of phenylalanine incorporation is a reflexion of physiological events.

Rat liver was homogenized in 0·25 M sucrose in the cold and centrifuged at 15,000*g* for 10 min. Sodium