not clear. Experiments with specific antisera against α_2- and β-lipoproteins seem to indicate that these proteins are absent in normal urine. This is in accord with results of earlier investigators. Ouchterlony tests with a specific antiserum against α_2-macroglobulin indicated the presence of traces of this protein in the urines of some of the individuals.

By the use of the Ouchterlony technique and specific antisera against purified proteins, urine was found to contain proteins giving reactions of identity with the plasma proteins pre-albumin, albumin, ceruloplasmin, transferrin, and γ-globulin. Proteins giving reactions of partial identity with plasma globulin seemed also to be present in urine.

A detailed report of this investigation will be published elsewhere.

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Steroid 11β-ol Dehydrogenase in Human Placenta

In the course of work on steroid metabolism in pregnancy, we have observed that adrenal steroids with 11β-ol structure are transformed into 11-keto compounds in homogenates of the human placenta at term. The action and properties of the corresponding enzyme have been investigated.

The homogenates were prepared as follows: 5 gm. of wet placenta were ground in an all-glass Potter mill and suspended in 25 ml. of 0·13 *M* sodium chloride buffered to *p*H 7·4 with 0·02 *N* phosphate.

The activity of the enzyme was measured by estimating the percentage of 11-keto compound formed under standard conditions: 1 μgm. of steroid per 1 gm. of placenta (5 ml. of the homogenate), incubated for 30 min. at 37° C., while shaking in a stream of oxygen.

Tritium-labelled steroids were used throughout[1]. The specific activities of cortisol-1,2-^{3}H, of corticosterone-1,2-^{3}H, and of androst-4-ene-11β-ol-3,17-dione, were respectively 835 mc./m.mole, 412 mc./m.mole and 827 mc./m.mole. Dichloromethane extracts of the homogenates were chromatographed on filter paper using the Bush system *C* and/or *B*1[2]. The radioactivity on the chromatograms was detected and measured in a windowless flow scanner[3].

The membranes (chorion) are a particularly rich source of the enzyme, which appears strongly bound to connective tissue. In homogenates lysed with 0·2 per cent 'Triton', only 30 per cent of the activity remained in the supernatant after centrifuging for 30 min. at 30,000 r.p.m.

The activity in the supernatant could be precipitated in the cold with neutral ammonium sulphate at half saturation. Precipitation at room temperature and precipitation with non-neutral ammonium sulphate both inactivated the enzyme. The activity also disappeared after freezing the tissues.

Table 1

No.	Steroid	Conditions	% conversion
1.	Cortisol	Supernatant with 0·2% 'Triton'	0
2.	,,	,, 0·2 μ*M* DPN added	11
3.	,,	,, 0·2 μ*M* TPN added	33
4.	,,	As No. 3, 10^{-3} *M* mercuric chloride	0
5.	,,	As No. 3, 100 μgm. of cortisone (reaction product) added	38
6.	,,	As No. 3, *p*H 6·01	32
7.	,,	As No. 3, *p*H 7·01	53
8.	,,	As No. 3, *p*H 8·03	66
9.	,,	As No. 3, *p*H 9·1	65
10.	Cortisone	0·2 μ*M* of TPN and 0·1 m*M* of glucose-6-phosphate added. Percentage conversion calculated for inverse reaction.	7
11.	Corticosterone	0·2 μ*M* of DPN added	19
12.	,,	0·2 μ*M* of TPN added	51
13.	Androst-4-ene-11β-ol-3, 17-dione	0·2 μ*M* of DPN added	17
14.	,,	0·2 μ*M* of TPN added	36

DPN, diphosphopyridine nucleotide; TPN, triphosphopyridine nucleotide.

The activity of all fractions was suppressed by dialysis for 24 hr. against saline. The activity could be restored by the addition of triphosphopyridine nucleotide. Diphosphopyridine nucleotide also restored the activity, but to a lesser degree.

In the presence of a triphosphopyridine nucleotide-reducing enzyme system (glucose-6-phosphate dehydrogenase) the keto compounds are reduced to the 11β-ol form, indicating the reversibility of action of steroid dehydrogenase.

The enzyme activity shows a broad maximum between *p*H 8 and *p*H 9.

The results are summarized in Table 1. Percentage conversion has been calculated as keto form/(keto form + hydroxy form) × 100.

Finally, it should be pointed out that the 11β-ol dehydrogenase of human placenta has a low substrate specificity since it transforms both C 19 steroids and C 21 steroids with or without a 17-hydroxy group in the side-chain.

The physiological role of the enzyme is uncertain. The conversion of 11-hydroxy group to 11-keto group is a well-known pathway of extra-hepatic steroid metabolism in the animal and in man[4–7].

The placenta enzyme may be part of the regulatory mechanism of the fœtal–maternal endocrine relations. Its presence in the placenta and in the membranes explains the origins of cortisone in normal human amniotic fluid[8].

The technical assistance of Mrs. M. R. Lacroix and Miss Y. Sonnet is gratefully acknowledged. The placenta were kindly supplied by Prof. Schockaert and Prof. Renaer from the Department of Obstetrics and Gynaecology.

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