The antibody secreting cells include histiocytic forms. As discussed in detail elsewhere[6], these antibody secreting histiocytes are primitive, less differentiated forms, not typical macrophages. Hemolytic plaques produced by these cells are smaller than 'lymphoid' plaques, but, on the other hand, are not due to the liberation of a passively acquired antibody[6]. No evidence for an active contribution of the new-born recipients of DC to the overall serological response was found. However, in the omental tissue covering the DC, a significant increase in the number of antibody producing cells can be detected. Omentum per se is a potent immunocompetent area[7]. The mechanism by which the antibody response is induced in the omental cells remains a matter of speculation. The concentration of both antibodies and possibly antigenic fragments diffusing from the DC[2] should be higher in this area than in the distant lymphatic organs. Antigen alone, in the presence of or diffusing from the DC, does not induce any significant response. It might well be that the antibodies formed by the competent cell population inside the DC potentiate the omental cells for the triggering effect of antigen. Nucleotides from the decaying cultured cells and factors liberated during the secondary contact with antigen which increase the mitotic activity of lymphoid cells[8] may have some adjuvant effect[9].

Table II. DC cultures of alveolar cells from normal donors (primary response)

Donor cells in DC		Recipient's organs – PFC per 10^6 viable lymphoid cells				
Original number $\times 10^6$	PFC per 10^6 cells	Tissue covering DC	Lymph nodes mediastinal	Lymph nodes mesenteric	Spleen	Thoracic duct lymph
13	40	1.7	–	–	<0.01	–
20	25	0.3	0.1	<0.01	0.02	0.2
25	56	0.5	0.06	<0.01	0.2	0.3
25	62	0.4	<0.06	<0.01	0.1	<0.1

Table III. DC cultures of alveolar cells from pre-immunized donors (secondary response)

Days after donor immunization	Donor cells in DC		Recipient's tissues – PFC per 10^6 lymphoid cells			
	Original number $\times 10^6$	PFC per 10^6 cells	Tissue covering DC	Lymph nodes mesenteric	Spleen	Thoracic duct lymph
8	10	12	0.8	0.4	0.2	0.2
15	11	40	3.7	0.8	0.2	0.2
15	8	5	0.8	0.03	0.01	<0.08
30	8	17	1.0	0.02	0.03	<0.1
30	9	60	1.2	0.1	0.6	0.3
30	10	37	1.1	0.9	0.8	0.2

Zusammenfassung. Alveolarexsudatzellen von Kaninchen wurden zusammen mit Schaferythrozyten in Diffusionskammern gebracht und diese in neugeborene Kaninchen implantiert. Antikörperbildende Zellen wurden in den Diffusionskammern und im Omentum der Rezipienten festgestellt.

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Distribution of C_{19}-Steroid Conjugates in Plasma Protein Fractions

In a previous communication[1], the association between sulphoconjugated C_{19}-steroids and certain plasma proteins under in-vivo conditions has been reported. In order to extend such information to a possible interaction between endogenous C_{19}-steroid glucuronosides and similar plasma proteins, 27.7 ml plasma, obtained from a 46-year-old female subject 10 min after i.v. administration of 0.201 µg 7α-^{3}H-3β-hydroxy-5-androstene-17-one (dehydroepiandrosterone) with 101×10^6 dpm, were submitted to preparative zone electrophoresis. The latter was performed on PVC in Longsworth buffer of pH 8.6 and an ionic strength of 0.1 at a field strength of 4.4 V/cm and led to a satisfactory separation of albumin, α_1-, α_2-, β- and γ-globulins. The corresponding zones were eluted with 0.15 *M* sodium chloride and the eluates reduced to 5–7 ml by ultrafiltration. Following the exhaustive extraction of free steroids with chloroform, total conjugates were extracted with 10 vol. ethanol-acetone (1:1 v/v). For separation of conjugates into steroid sulphoconjugates and glucuronosides ion exchange chromatography of the filtrate on DEAE-Sephadex A-50 and repeated thin-layer chromatography of resulting fractions on silica gel G in chloroform-methanol-ammonia (10:10:0.2 v/v) and chloroform-methanol-ammonia (20:5:0.2 v/v) proved adequate[2]. Aliquots of the different conjugate fractions, as well as of the free fraction were assayed for ^{3}H-activity prior to cleavage of conjugates by solvolysis[3] or incubation with β-glucuronidase (Ketodase, Warner Chilcott, Morris Plains, N.J., USA). Free and liberated C_{19}-steroids were isolated by multiple thin-layer chromatography, converted into 2,4-dinitrophenyl-

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Free and conjugated C_{19}-steroids in plasma protein fractions

Steroid fraction	Cpm in protein fraction				
	Albumin	α_1-globulin	α_2-globulin	β-globulin	γ-globulin
Free	2,144	3,556	2,201	1,336	2,861
Sulphoconjugates	36,520	20,350	4,810	214	82
Dehydroepiandrosterone	26,190	14,650	1,040	–	–
Androsterone	4,850	3,230	410	–	–
Etiocholanolone	2,090	1,230	221	–	–
Androstenedione	870	312	108	–	–
Androstandione / Etiocholandione	631	358	76	–	–
Androstenediol	1,078	834	146	–	–
Androstenetriol	311	148	18	–	–
Glucuronosides	839	2,692	172	29	36
Dehydroepiandrosterone	69	241	–	–	–
Androsterone	352	1,110	–	–	–
Etiocholanolone	109	817	–	–	–

Androstenedione = 4-androstene-3,17-dione. Androstandione = 5α-androstan-3,17-dione. Etiocholandione = 5β-androstan-3,17-dione.
Androstenediol = 5-androstene-3β,17β-diol. Androstenetriol = 5-androstene-3β,16α,17β-triol.

hydrazones[4] or acetates and rechromatographed in suitable solvent systems. ^{3}H-activity of individual compounds was determined in a Packard Tricarb Spectrometer Mod. 3310. Final identification of compounds was achieved by purification to constant specific activity, eventually after reverse isotope dilution with standard material.

As indicated by the Table, free C_{19}-steroids were randomly distributed over the various protein fractions, whereas the sulphoconjugates prevailed in the albumin and α_1-globulin fractions as observed in earlier experiments[1]. On the other hand, almost 70% of ^{3}H-labelled C_{19}-steroid glucuronosides could be isolated from the fraction of α_1-globulins. On the basis of these findings and previous data it is suggested that endogenous lipophile C_{19}-steroid sulphoconjugates are transported preferably by a post-albumin. C_{19}-steroid glucuronosides, however, which in this experiment were formed through metabolism of free dehydroepiandrosterone, appear to be associated rather with an α_1-globulin. The composition of the different conjugate fractions is quite comparable to that detected in other investigations[5]. In contrast to the fraction of sulphoconjugates, where dehydroepiandrosterone represented the predominant C_{19}-steroid, the fraction of glucuronosides contained primarily 3α-hydroxy-5α-androstan-17-one (androsterone) and 3α-hydroxy-5β-androstan-17-one (etiocholanolone) as the major metabolites of the aforementioned substrate.

Zusammenfassung. Nach i.v. Gabe von 7α-^{3}H-Dehydroepiandrosteron wurde menschliches Plasma einer präparativen Zonenelektrophorese unterworfen und in Albumine, α_1-, α_2-, β- und γ-Globuline zerlegt. Aus den einzelnen Proteinfraktionen trennte man freie Steroide, Steroid-Sulfokonjugate und -Glukuronoside ab und untersuchte letztere Fraktionen auf ihren Gehalt an markierten C_{19}-Steroiden. Es zeigte sich, dass die Steroid-Sulfokonjugate vornehmlich in der Proteinfraktion der Albumine, die Steroid-Glukuronoside dagegen bevorzugt in der Proteinfraktion der α_1-Globuline auftraten.

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Interactions Between α-MSH and Sex Steroids on the Preputial Glands of Female Rats

The preputial glands, which can be regarded as giant sebaceous glands, are structures of epidermic origin whose precise biological significance has still not been clarified. Various studies have been published showing that their development and their secretory function are dependent upon certain sex steroids[1,2] and polypeptides such as STH, LTH, and ACTH[3,4]. Since melanocyte-stimulating hormone (MSH) exerts effects which are essentially of an epidermotropic character, it was felt that it would be interesting to determine to what extent and under what conditions this hormone might also be capable of stimulating growth of the preputial glands.

Method. Sprague-Dawley rats of female sex, weighing 180 g, were given treatment with synthetic α-MSH and sex steroids by the s.c. route for 2 weeks; the treatment

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