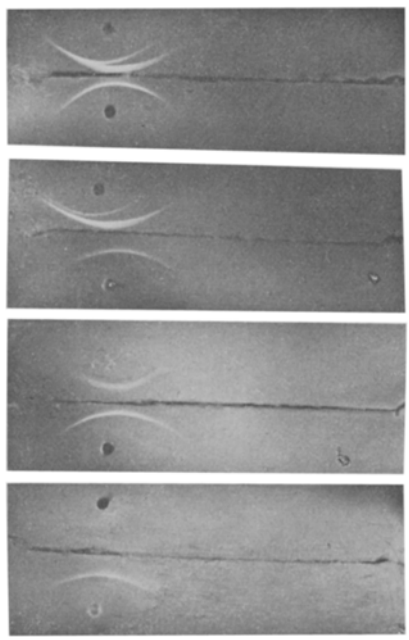

Fig. 3. Micro-immuno-electrophoretic analysis: Antiserum to crude extract (in trough) tested against the following antigens in the upper well (top to bottom): Crude extract, P1–1, ovine FSH (NIH-FSH-S7) and ovine LH (NIH-LH-S5), respectively. In all cases, lower well contained P1–2. Electrophoresis at pH 8.6, at 5 mA/slide for 2 h at room temperature. Migration towards anode (right).

With P1–2, on the other hand, a single sharp line was obtained. Micro-immuno-electrophoresis gave similar results (Figure 3). Multiple precipitin lines were obtained with all the antigens that reacted positively, with the exception of P1–2, which consistently gave only a single precipitin line.

These immunochemical tests, therefore, confirm that P1–2 is homogeneous. It is interesting that in tests for crossreactivity of the antiserum, a positive reaction was obtained with ovine FSH, but not with ovine LH, PMS or HCG. Further investigations are in progress[6].

Résumé. Des tests immunochimiques confirment l'homogénéité de P1–2, une glycoprotéide pituitaire d'origine ovine ayant une activité hormonale double, folliculostimulante et lutéinisante.

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Experimental Allergic Sialoadenitis III. Acute Parotitis Induced by Instillation of Antiserum to Rat Plasma into the Glandular Duct of Rats

Few investigations have been devoted to experimental allergic sialoadenitis[1–5]. We have recently developed a technique for cannulation of the parotid duct of rats enabling the easy introduction of fluids into the gland[6]. Using this technique acute sialoadenitis was induced by immune mechanisms. Challenge of the gland of previously sensitized animals with the homologous antigen resulted in an inflammatory reaction[7]. Intraductal instillation of antiserum to basement membranes caused a necrotizing sialoadenitis and vasculitis[8]. The purpose of this communication is to report on the production of parotitis with antiserum to rat plasma as an extension to our previous studies.

Rabbits were immunized with lyophilized rat blood plasma. 3 injections each of 100 mg of lyophilizate suspended in 2 ml of saline and emulsified in 2 ml of complete Freund's adjuvant were given at weekly intervals. 2 further injections without adjuvant were administered during the 5th and 6th week. Blood was drawn 10 days after the last injection. The antisera gave 5 to 8

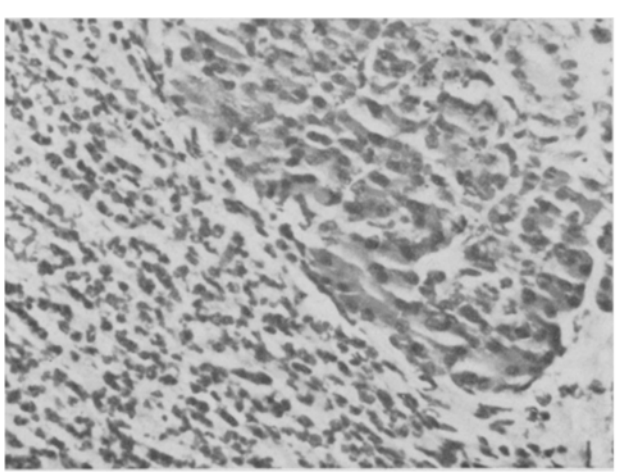

Fig. 1. Severe acute septal and moderate lobular inflammatory infiltration. Hematoxylin and eosin. ×670.

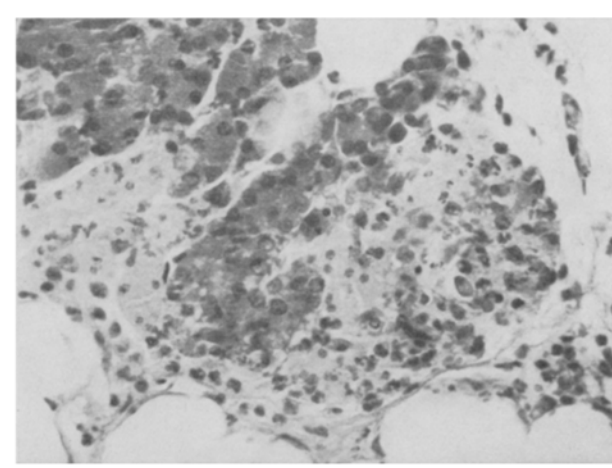

Fig. 2. Focal necrosis of glandular parenchyma and inflammatory response. Hematoxylin and eosin. ×440.

precipitation bands when reacted with rat plasma in the double immunodiffusion test. The duct of the right parotid gland of rats was cannulated with a polyethylene catheter[6] and 0.5 ml of the antiserum to rat plasma, normal rabbit serum, normal human serum or physiological saline were slowly introduced. The rats were killed 24 h after the intubation and the right gland was removed for histological examination. The extent of inflammation was evaluated on an arbitrary scale from 0 to 3+ as described elsewhere[7]. Since proteinous solutions per se cause mild inflammation (1+), moderate (2+) and severe lesions (3+) only were considered to be of significance.

Moderate to severe sialoadenitis developed in 8 of 12 rats following instillation of antiserum to rat plasma into the parotis. Histologically, the lesions were characterized by inter- and intralobular inflammatory infiltration and focal necrosis of the parenchyma (Figures 1 and 2). The infiltrate consisted of polymorphonuclear granulocytes with an admixture of lymphocytes, histiocytes and a few plasma cells. Moderate sialoadenitis (2+) was observed in 1 of 20 control rats given normal human serum. The glands of 10 and 9 rats receiving normal rabbit serum or physiological saline, respectively, were normal.

Blood plasma proteins are widely distributed in cells, connective tissues and interstitial spaces, lymphatics and blood vessels[9]. Instillation into the gland of antiserum to plasma components is apparently followed by local formation of immune complexes, resulting in an inflammatory reaction[10]. It is assumed that the plasma proteins residing in cells, connective tissues, interstitial spaces and/or vessels participate in the formation of the complexes. In view of the procedure employed, sialoadenitis is ascribed to a modified reversed Arthus type of reaction, since it is caused by precipitating antibodies introduced into a tissue harbouring the respective soluble antigens[11,12].

Zusammenfassung. Durch den Ausführungsgang der Ohrspeicheldrüse wurde Antiserum gegen Rattenblutserum eingeleitet, was zu einer schweren allergischen Entzündung der Parotisdrüse führte. Es wird angenommen, dass es sich bei dieser Entzündung um eine besondere Art von Arthusreaktion handelt.

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7α-Hydroxylation of Dehydroepiandrosterone in Human Testis and Epididymis in vitro

Steroid 7α-hydroxylase, known from various tissue of animals and lower vertebrates, was demonstrated in human adrenals and liver[1]. The testicular 7α-hydroxylation of steroids has so far been described only in human foetus when dehydroepiandrosterone was used as a substrate[2], and in rats where androstenedione was the preferred substrate for hydroxylation[3,4].

Considering the possible regulative role of 7α-hydroxylation in the androgen biosynthesis[3], we studied the in vitro formation of 7α-hydroxylated metabolites of dehydroepiandrosterone in human testis and epididymis of an adult man.

Human testes and epididymis were obtained from a sexually deviated but otherwise healthy man aged 38 years, who underwent the voluntary castration. The histological control confirmed the normal structure of both testis and epididymis as well as the ability of the testes for complete spermiogenesis. Immediately after operation the tissues were kept in an ice bath and then minced separately for approximately 30 min. The samples of minced tissue, 100 mg each, were incubated separately with 0.2 μC of [4-^{14}C] dehydroepiandrosterone ([4-^{14}C] 3β-hydroxy-5-androsten-17-one = ^{14}C-DHA; specific activity 14.0 mCi/m*M*), dissolved in 0.05 ml propylene glycol. The incubation was carried out in 3 ml Krebs-Ringer buffer, pH 7.4, containing glucose, in an oxygen atmosphere at 37 °C for 60 min. Both tissues were incubated in 2 parallel samples without addition of any cofactor, with the blank containing only a substrate and buffer.

After the incubation, 20 μg of each the expected non-radioactive metabolite was added to the samples and the incubation mixture was extracted with ethyl acetate and chloroform. The extracts were purified by chromatography on small columns of silica gel followed by paper chromatography in the system Bush B5. The more polar metabolites (7-hydroxydehydroepiandrosterone and 5-androstene-3β, 7α, 17β-triol) were separated in this system, whilst the less polar ones were rechromatographed in the system, cyclohexane: toluene: methanol: water = 9:1:8:2. 7α- and 7β-hydroxyepimers of dehydroepiandrosterone were further separated in the system n-heptane: toluene: methanol: water = 9:11:16:4 developed for 48 h. 5-androstene-3β, 7α, 17β-triol was rechromatographed on ethylene glycol impregnated paper, developed by dichloromethane for 10 h, respectively. The radioactive steroids on the paper were detected by autoradiography, the spots were then cut off and their radioactivity was measured using liquid scintillation spectrometer. Additional 500 mg of tissue was incubated with [4-^{14}C] dehydroepiandrosterone (2.5 μCi) and, in the ex-

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