was mit dem über die gegenseitige Stellung der Sulfanilamido- und der Methoxygruppen gesagten übereinstimmt. Der Ersatz des Methoxyls durch die Methylmercaptogruppe übte einen recht ungünstigen Einfluss auf die chemotherapeutische Wirksamkeit aus (I-3064, I-3068, I-3065).

Bei der Synthese der besprochenen Sulfonamide benutzten wir folgende Verfahren:

1. Sulfanilierung der betreffenden 2-Amino- bzw. 4-Amino-alkoxypyrimidine durch Einwirkung von *p*-Acetamidobenzensulfochlorid im Pyridin (Methode A).

2. Behandeln von Na-Sulfanilamid mit den entsprechenden Alkoxypyrimidyl-4-trimethylammoniumchloriden nach BRETSCHNEIDER und KLÖTZER[2] (Methode B).

3. Verschmelzen einer Mischung von Sulfanilamid, 4-Chlor-2-methalmercapto-alkoxypyrimidin und Pottasche (Methode C).

4. 2-Sulfanilamido-4-hydroxy-5-methoxypyrimidin wurde durch Kondensation von Sulfaguanidin mit dem Na-Enolat des Methoxyformylessigsäuremethylesters dargestellt (Methode D).

Summary. Some new 2- and 4-sulfanilamido-5-alkoxypirimidines and their hydroxy-, methoxy-, methylmercapto- and methyl-derivatives were prepared. All of the substances described were tested on white mice infected with *Streptococcus pyogenes*.

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cis-Cyclohexane-1,2-Diol in the Beaver Gland

We wish to report the isolation of the title compound from the scent glands of the Canadian beaver. The glands were extracted with alcohol and the 'basic' material, obtained from this extract by a conventional distribution between ether and aqueous sulfuric acid, was subjected to a fractional distillation. A fraction boiling at 80–125° (0.08 mm Hg) proved to be crude *cis*-cyclohexane-1,2-diol, while the following oily fraction was almost pure castoramine (125–135°/0.08 mm Hg)[1]. A clean separation of the two compounds could then be achieved by alumina chromatography. The appearance of the diol in the basic extract is due to the fact that its solubilities in ether and water are of a comparable magnitude. Additional amounts could therefore be obtained by a continuous ether extraction of the appropriate aqueous layers (total yield = 0.2% of semi-dried glands).

The diol, purified by distillation. chromatography and crystallization from ether (m.p. 96–97°) proved to be identical in all respects (m.p., mixed m.p., IR, NMR) with a synthetic specimen prepared by oxidation of cyclohexene with sodium chlorate and osmium tetroxide[2]. The identity was further confirmed by the preparation of the crystalline dibenzoate.

cis-Cyclohexane-1,2-diol has not been previously reported as a beaver gland constituent[3], and, to our knowledge, this communication describes its first isolation from a natural source. It is tempting to speculate that the diol is produced by the beaver by the reduction of catechol (or its derivative), a compound present in the beaver gland[3].

Zusammenfassung. Aus den Biberdrüsen wurde *cis*-Cyclohexan-1,2-diol isoliert und charakterisiert.

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Effect of Iproniazid on Pregnancy

From the observations that reserpine influences the vaginal cycle and reduces fertility in rats[1,2], it has been concluded that serotonin may interfere with pregnancy[3]. Thus POULSON et al.[3] have shown that serotonin can inhibit implantation and can terminate an established pregnancy in mice; they further demonstrated that implantation can also be prevented by injecting large doses of 5–10 mg of iproniazid per mouse and day over 6 days. It seemed important to know, if this effect of iproniazid on implantation can be considered specific or whether it is a side effect appearing only with high doses. We therefore tried to find inhibition of implantation in mice with smaller doses and with shorter treatment.

The results are given in the accompanying Table. It appears that, with the exception of a single series, the iproniazid treated mice showed the same frequency of pregnancies as their controls, irrespective of dose or time of administration of the drug during the critical period of implantation. Also the number of young per litter was the same in treated and in control animals. The table also shows that reduced fertility after 100 mg/kg on day 5 of pregnancy was only observed in one group of animals, but not in a second parallel series of mice.

From our experiments, we conclude that iproniazid does not interfere with implantation in our mice. The discrepancy between our results and those of POULSON et al.[3] is difficult to explain. The discrepancy could be accounted for by assuming a metabolic difference in the animals used. Or one can argue that treatment with 200 mg/kg of iproniazid on 6 consecutive days has unfavorably altered the endocrine balance by interference on the level of the hypothalamo-pituitary axis. TUCHMANN-DUPLESSIS' experiments[2] show that reserpine probably interferes with implantation by acting on the level of the pituitary. Typically, when placentation was complete, the effect of reserpine on fertility diminished, and this is just what happened also in POULSON's et al. experiments with iproniazid[3]: In the second half of pregnancy the effect of iproniazid was practically nil, while sensitivity to exogeneous serotonin was much greater than in the first half of pregnancy. SPECTOR[4] working with another M.A.O. inhibitor, chronically given to rats and mice with the effect of reduced fertility, also offers as an alternative to specific action at the site of implantation the same explanation, i.e. that M.A.O. inhibitors may well act *via* the central nervous system and the pituitary, and this is made very probable by the recent paper of KHAZAN et al.[5]. Our conclusion is, therefore, that M.A.O. inhibitors may under certain conditions indirectly interfere with implantation, but that this action is not to be considered a specific one.

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