et al., who noted a weight increment in several hypophysectomized male pigs with unsuccessful ocular pituitary grafts.† Moreover, examination of the data published by Allanson, *et al.*,[8] indicates a post-operative weight gain in 3 of their hypophysectomized males, although no special mention was made of the fact. A possible explanation of the gain in body weight after hypophysectomy may lie in the fact that, shortly after the guinea pig has recovered from effects of the operation, it eats as voraciously as the normal animal. The rat, on the contrary, has a diminished food intake after hypophysectomy. Thus it would appear that appreciable gain in weight in the guinea pig, at least, is not necessarily dependent on a growth factor from the pituitary.

Summary. Spermatogenesis was maintained by subcutaneous injection of 3 mg daily of testosterone propionate in 4 guinea pigs hypophysectomized for 60 to 155 days. Tubular atrophy in the pig may not be complete as late as 84 days after hypophyseal ablation. Adrenal atrophy occurs after removal of the pituitary, but increase in body weight does not necessarily cease.

13116 P

Acetylation of Optical Isomers of S-benzylcysteine in Rats and Humans.

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We reported the conversion of S-benzyl-*l*-cysteine to the corresponding acetyl derivative in the rat, rabbit, dog[1] and man[2] and suggested the possibility of the inversion of S-benzyl-*d*-cysteine to the acetylated *l*-derivative *via* Knoop's acetylation mechanism[3] which postulates the formation of a keto acid from the *d*-amino acid followed by the interaction of the keto acid with ammonia and pyruvic acid to give the *l*-acetyl amino acid. duVigneaud, *et al.*,[4] reported

† Gains in body weight have been observed to occur also in hypophysectomized female guinea pigs with unsuccessful ocular grafts of pituitary (Haterius, H. O., and Cutuly, E., unpublished data).

1 Stekol, J. A., *J. Biol. Chem.*, 1938, **124**, 129; 1939, **128**, 199.

2 Stekol, J. A., *Proc. Am. Chem. Soc.*, 100th meeting, Detroit, Mich., 1940.

3 Knoop, F., *Z. physiol. Chem.*, 1910, **67**, 489; Knoop, F., and Kertess, E., *Z. physiol. Chem.*, 1911, **71**, 252; Knoop, F., and Blanco, J. G., *Z. physiol. Chem.*, 1925, **146**, 267.

4 du Vigneaud, V., Wood, J. L., and Irish, O. J., *J. Biol. Chem.*, 1939, **129**, 171.

results which apparently indicated such an inversion of S-benzyl-*d*-cysteine in the rat. On feeding the *d*-isomer to rats, some *l*-acetyl derivative together with *dl*-acetyl benzylcysteine were isolated from the urine. When *d*-benzylcysteine was fed to rats and humans,[2] significantly smaller yields of the acetylated products could be isolated from the urine as compared to those obtained after the administration of comparable amounts of *l*-benzylcysteine. We drew the conclusion at the time[2] that these findings did not conform to the postulates of Knoop's acetylation theory which presupposes that if the acetylation of an amino acid takes place *via* the keto acid, then the amounts of the acetylated product excreted in the urine of animals which were fed *d*- or *l*- amino acid should be the same. Indeed, du Vigneaud and Irish,[5] who revived Knoop's theory, stated that "this would be expected if the theory were correct, since both enantiomorphs should be deaminated to the keto acid." The lower yields of the acetyl derivatives in the urine after the feeding of *d*-benzylcysteine to rats and man[2] we later attributed[6] to a possible destruction of the intermediate keto acid which was presumably formed from *d*-benzylcysteine prior to its conversion to the acetyl derivative.

Before accepting the acetylation mechanism of Knoop's as valid for the synthesis of mercapturic acids *in vivo,* we felt it advisable to ascertain experimentally the fate of the larger portion of *d*-benzylcysteine when it is fed to animals, since so far only a fraction of the substance fed has been accounted for in the urine as the acetylated product. We, therefore, repeated our earlier work on the isomers of benzylcysteine in rats and humans[2] and isolated from the urine the acetylated derivatives as before. In addition, however, we were able to isolate from the urine of rats and humans which were fed *d*- or *dl*-benzylcysteine, *unacetylated d-benzylcysteine* in 20 to 28% yields of the *d*-derivative fed. At no time comparable amounts of *l*-benzylcysteine yielded in the urine the unchanged substance, but only the *l*-acetyl derivative. Control experiments excluded the possibility of hydrolysis or racemization of the acetyl derivative during the isolations. The isolated *d*-benzylcysteine had a specific rotation of $(\alpha)_D^{24} = -24°$ for a 1% solution in 1 N NaOH. Analytically, the substance was identical with the original *d*-benzylcysteine fed.

This observation suggests that if both isomers of benzylcysteine are acetylated *in vivo* directly (which is the more likely probability) then the *l*-isomer is acetylated *in vivo* to a greater extent than the

[5] du Vigneaud, V., and Irish, O. J., *J. Biol. Chem.*, 1937-38, **122**, 349.

[6] Stekol, J. A., PROC. SOC. EXP. BIOL. AND MED., 1940, **45**, 693.

d-enantiomorph. If the acetylation is preceded by the formation of the corresponding keto acid from both isomers, as Knoop's theory postulates, then the *d*-isomer is apparently less readily deaminized than the *l*-isomer. *d*-Benzylcysteine is undoubtedly directly acetylated, as is indicated by the excretion of *dl*-acetylbenzylcysteine in the urine. We are not excluding the possibility of racemization of *d*-benzylcysteine *in vivo,* neither do we imply that some oxidative deamination of either or both isomers of benzylcysteine does not occur *in vivo.*

The work is being continued and extended to benzylhomocysteine and phenylaminobutyric acid, particularly with reference to the possibility of racemization and preferential rate of acetylation of the optical isomers of these amino acid derivatives *in vivo* as contrasted with Knoop's acetylation theory.

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Some Effects of Experimental Trichinosis in the Dog.*

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In view of the fact that encysted trichina larvae become calcified in the muscles of the host, it appeared desirable to determine the changes which occur in the blood picture of dogs suffering from experimental trichinosis. The present report presents the results of cytological studies in which concurrent determinations were made on the level of blood calcium, hemoglobin and inorganic phosphate at periodic intervals before and during the period of infection.

Methods and Technic. The 13 mature mongrel dogs, used in these experiments, were kept under observation for several weeks to determine whether or not they were suffering from any disorder. Each was treated with arecoline hydrobromide (Parke, Davis) to insure freedom of the gastro-intestinal tract of parasites. The dogs were kept in separate cages in a room in which the temperature fluctuated very little. Purina dog biscuit was supplied *ad libitum.*

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