

to the preference for the new solution. As Lester and Greenberg<sup>8</sup> have shown, a third bottle containing sugar solution strongly influenced the choice of alcohol. Only if the preference for a substance is strong in every set of alternatives can it be said that the animal has a specific craving for this substance.

The reason for the preference for, or aversion to, alcohol among animals is not known. It was shown earlier that a low original alcohol consumption in a rat can be changed into a strong preference in various ways: by a deficient diet causing metabolic block<sup>7</sup>, by damaging liver function<sup>8</sup> or by inducing an experimental conflict situation<sup>9</sup>. In our investigation all these conditions could be excluded. According to one of us<sup>10</sup>, the choice of alcohol can be explained by the metabolic type of the animal. Perhaps differences in metabolism can explain the differences in preference for alcohol in the various animal species.

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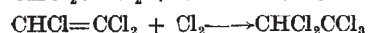
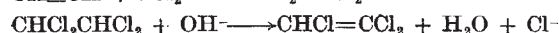
- <sup>1</sup> Mardones, R. J., Hederra, D. A., and Segovia, M. N., *Bol. soc. biol., Santiago, Chile*, **7**, 1 (1949).  
<sup>2</sup> Williams, R. J., Berry, L. J., and Beerstecher, E., *Proc. U.S. Nat. Acad. Sci.*, **35**, 265 (1949).  
<sup>3</sup> Richter, C. P., in *Neuropharmacology*, edit. by Abramson, H. A., **3** (Josiah Macy Jr. Foundation, New York, 1957).  
<sup>4</sup> Emerson, G. A., Brown, R. G., Nash, J. B., and Moore, W. T., *J. Pharmacol. Exp. Therap.*, **106**, 384 (1952).  
<sup>5</sup> Loiseleur, J., and Petit, M., *C.R. Soc. Biol., Paris*, **141**, 568 (1947).  
<sup>6</sup> Lester, D., and Greenberg, L. A., *Quart. J. Studies Alc.*, **13**, 553 (1952).  
<sup>7</sup> Mardones, R. J., and Onfray, B. E., *Rev. Chilena Hig. y Med. Prevent.*, **4**, 293 (1942).  
<sup>8</sup> Sirnes, T. B., *Quart. J. Studies Alc.*, **14**, 3 (1953).  
<sup>9</sup> Conger, J. J., *Quart. J. Studies Alc.*, **12**, 1 (1951).  
<sup>10</sup> Forsander, O., Kohonen, J., and Suomalainen, H., *Quart. J. Studies Alc.*, **10**, 379 (1958).

## RADIOBIOLOGY

### Urinary Metabolites of <sup>14</sup>C-Tetrachloroethylene in Mice

IN recent years tetrachloroethylene (perchloroethylene) has rapidly gained in importance as an industrial solvent. Its toxic effects have not been investigated very much, however, and its metabolism is almost unknown. The present work was carried out in order to facilitate the estimation of health hazards caused by exposure to this solvent.

Starting with <sup>14</sup>C-acetylene, tetrachloroethylene was synthesized essentially according to the industrial method<sup>1,2</sup>:

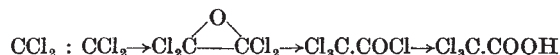


The crude <sup>14</sup>C-tetrachloroethylene was purified by preparative gas chromatography, and the specific activity of the purified product was 0.7  $\mu\text{c./mgm.}$

Five female mice, one at a time, were exposed for 2 hr. in a sealed flask (2.7 l.) to the solvent vapour in doses of about 1.3 mgm./gm. body-weight. 70 per cent (42–87 per cent) of the solvent was absorbed. In four days about 90 per cent of the absorbed activity was excreted, namely, 70 per cent in the expired air, 20 per cent in the urine and less than 0.5 per cent in the faeces. By chromatographic, auto-

radiographic and isotope-dilution methods, in part by Jondorf's method<sup>3</sup>, the following metabolites were identified in the urine (percentage of total urinary activity): trichloroacetic acid (52 per cent), oxalic acid (11 per cent) and dichloroacetic acid (traces). No monochloroacetic acid, formic acid or trichloroethanol was found. After hydrolysis 18 per cent of the urinary metabolites were still not extractable with ether.

These results indicate the formation of an epoxide further rearranged to trichloroacetyl chloride as one metabolic pathway for tetrachloroethylene:



This reaction has been previously shown to occur *in vitro* with the oxidation of tetrachloroethylene in the presence of non-biological catalysts<sup>4,5</sup>.

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<sup>1</sup> *Fiat Final Rep.* No. 843 (1947).

<sup>2</sup> *Bios Final Rep.* No. 1056 (1947).

<sup>3</sup> Jondorf, W. R., *The Metabolism of Certain Chlorinated Hydrocarbons* (University of London, 1956).

<sup>4</sup> Kirkbride, F. W., U.S. Patent 2,321,823 (1943).

<sup>5</sup> Frankel, D. M., Johnson, C. E., and Pitt, H. M., *J. Org. Chem.*, **22**, 1119 (1957).

### Action of Tetracycline on the Level of Properdin

THE influence of antibiotics on the formation of antibodies has been studied by many authors. Recently, Patočka *et al.*<sup>1</sup> published a paper describing the inhibitory effect of tetracycline on the formation of specific antibodies of various types in infections or after immunization with brucellæ, the influenza virus and herpes in laboratory animals. Clinical experience shows that in certain infections caused by pathogenic agents (viruses) tetracycline may have a favourable effect on the course of the disease. We tried to ascertain whether in these cases this is not due to the action of tetracycline on a non-specific defence mechanism.

We therefore investigated the properdin-levels in rabbits daily for one month with intramuscular application of 25 mgm. tetracycline per kgm. body-weight. Determination of properdin was carried out by Isliker and Linder's<sup>2</sup> modification of Pillemer's method. Properdin was determined before the beginning of the experiment and at intervals of 10 days. Before the experiment the average properdin-level was approximately ten units, which is regarded as the normal level. During the first ten days, properdin activity increased to a varying degree in all laboratory animals, on the average to more than double its original value. On the twentieth day we noted a marked fall, in spite of the continued application of tetracycline; on the thirtieth day the level stabilized at about the normal value. Fig. 1 shows the curve of the properdin-level in a group of nine rabbits after statistical evaluation. The experiment was then repeated in a similar manner with six rabbits which were also exposed to X-rays (500 r.). The results obtained in this group were similar, except that the decrease in the properdin-level between the tenth and the twentieth day after irradiation was much more pronounced, frequently below the original level. In the control group of four rabbits the familiar decrease in properdin activity was