lesions are much more abundant than after a single infection. Although the infected animals become skin sensitive to tuberculin and to the homologous strain, reinfection does not produce a local allergic reaction. The local nodule which develops promptly on primary infection, frequently fails to develop at the site of reinfection. The pathogenic action of these strains has been compared with that of known non-pathogenic acidfast bacilli, namely, one pigmented acidfast rod isolated from tap water and *B. phlei.* Both these strains produce a similar yellow pigment, but their colonies are rough and dry. Massive doses of these 2 strains produced abscesses at the site of injection, but never any visceral lesions.

The 11 strains of this group are apparently not identical; they vary in growth intensity, in pigment, in their growth on liquid media and in their pathogenic action. Whether they have any significance in human pathology remains to be studied.

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Observations on Various Insulin Mixtures Administered Per Os.

ELMER S. GAIS. (Introduced by L. Gross.)

From the Laboratories of the Mt. Sinai Hospital, New York.

At the time of publication of Stephan's paper[1] on the use of "cholosulin", a desoxycholic acid-insulin mixture, we were engaged in the preparation and study of a similar compound based on identical theoretical considerations. Mixtures of desoxycholic acid and insulin were prepared and administered by stomach tube to fasting rabbits, the experiments controlled by subcutaneous injection. The results in 4 experiments with 4 rabbits each, were inconclusive. The blood sugar curves closely paralleled the controls. The recent reports of Bronkhorst, Freud and Laquer,[2] and Wahncau and Bertram,[3] seem to show some slight effect on carbohydrate metabolism by this compound, but they ascribe it to the bile acid and not the insulin in the mixture. The clinical use of Stephan's preparation by Umber and Rosenberg,[4] and others has not proven successful. Our mixture also had no effect on human diabetes.

[1] Stephan, R., *Munch. Med. Woch.*, 1929, **76**, 1579; *Med. Klin.*, 1930, **26**, 228.

[2] Bronkhorst, A. J., Freud, J., de Jongh, S. W., and Laequer, E., *Nederl. Tijdschr. v. Geneesk*, 1930, **74**, 2185; abs. *Endokrinol.*, 1931, **8**, 43.

[3] Wahncau, E., and Bertram, F., *Klin. Woch.*, 1931, **10**, 486.

[4] Umber, F., and Rosenberg, M., *Deut. Med. Woch.*, 1930, **56**, 169.

Another line of approach to the problem of a practical and successful method of administering insulin by mouth suggested itself. In order to attempt the prevention of digestion of insulin in the gastro-intestinal tract, a preparation from the juice of ground and pressed *Ascaris lumbricoides* of the pig, yielding a potent, assayable anti-protease fraction, was made by the method of Weinland.[5] This substance, a fluffy, white, sticky powder, was assayed in potency by titrating it against active peptic and pancreatic proteases. In definite quantities it prevented the digestion of egg-white and casein. The action is inhibitory and not permanent, lasting about 24 hours.

When a known concentration of this anti-protease was added to mixtures of commercial insulin plus either peptic or tryptic proteases, the potency of the insulin as tested by subcutaneous injection into 8 fasting rabbits, was unimpaired. Typical hypoglycemic reactions resulted. Controls of the proteases, the anti-protease and digested insulin-protease mixtures, gave no hypoglycemic response. In other words, the anti-protease was shown to be capable of preventing the digestion and destruction of commercial insulin by the proteases *in vitro*.

When, however, the mixture of insulin and anti-protease was fed by stomach tube to fasting rabbits (2 groups of 4 animals) in an attempt to prevent the digestion of the insulin in the gastro-intestinal tract, no hypoglycemic reactions were observed. It cannot be stated whether absorption had taken place, for the recovery of the introduced insulin was not attempted. At least one may state that, although large quantities of anti-protease were used, there was no evidence of insulin absorption and action as judged by the blood sugar curve.

The handling of the Ascaris and its extracts causes sensitization phenomena, such as bronchospasm, rhinorrhea, urticaria, etc. Ransom, Harris and Couch[6] noted that the globulin fraction of the tissue juice does not contain the sensitizing substance. Whether this globulin fraction contains the anti-protease would be of interest in the utilization of the anti-protease in man, for the ingestion of the anti-protease powder by the author caused rather severe asthma and uveal and laryngeal edema in the one diabetic to whom the insulin anti-protease mixture was administered. An assistant developed urticaria at first contact with the powder.

Before further human study can be pursued, a sensitizer free ex-

[5] Weinland, E., *Z. Biol.*, 1902, **25**, 86.

[6] Ransom, B. H., Harris, W. T., and Couch, J. F., *J. Agric. Res.*, 1924, **28**, 577.

tract must be prepared. Rabbits, however, are not sensitive and can be used.

Further study of a sensitizer-free insulin anti-protease mixture seems to be warranted. Other sources of the anti-protease are being investigated. Perhaps, if combined with some substance aiding absorption such as desoxycholic acid or saponin,[7] the insulin polypeptide, protected from hydrolysis, might be absorbed and exert its effect when administered *per os.*

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Fermentation of the d- and l- Forms of Arabinose by Bacteria.

STEWART A. KOSER AND FELIX SAUNDERS.

From the Departments of Hygiene and Bacteriology and of Physiological Chemistry, University of Chicago.

Although the commoner sugars have found an every day use in bacteriological technic for the separation and characterization of different types of bacteria, it is only rarely that optical antipodes have been subjected to comparative fermentation tests for the purpose of correlating sugar structure with utilization.[1] In most cases it is difficult or impossible to prepare both the *d-* and *l-* forms of a given sugar. Arabinose is one of the few exceptions to this rule.

The common form of arabinose is *l*-arabinose[2] ($[\alpha]_D = +105°$), which occurs naturally in a combined form in many gums such as arabic and mesquite. It has been used in bacteriological work for a number of years. *d*-arabinose seldom occurs naturally and must be prepared from *d*-glucose by degradation. Recently we obtained* a supply of *d*-arabinose which had been prepared by the oxidation of calcium gluconate with hydrogen peroxide in the presence of ferric acetate. This afforded us an opportunity to study the fermentation of both *d*-arabinose and *l*-arabinose, which are exact opposites in configuration and rotation.

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[7] Collens, W. S., and Goldzieher, M. A., PROC. SOC. EXP. BIOL. AND MED., 1932, **29**, 756.

[1] Kendall and Gross, *J. Infect. Dis.*, 1930, **47**, 249. Lester, *Acta Path. Microbiol. Scandanavica*, 1926, **3**, 696.

[2] The symbols *d-* and *l-* in connection with sugars refer to family relationships and not to the sign of rotation. See Rosanoff, *J. Am. Chem. Soc.*, 1906, **28**, 114.

* We are indebted to Dr. W. C. Austin of the Department of Physiological Chemistry of Loyola University for the supply of *d*-arabinose.