# CURRENT LITERATURE

### Adrenal Cortical Hormones and the Formation of Histamine and 5-Hydroxytryptamine by R. Hicks and G. B. West (1958): Nature, 181, 1342.

Groups of rats were given daily doses of hydrocortisone (or cortisone) for 14 days and then killed for testing for histamine and 5-hydroxytryptamine activity. Control animals were treated with the solvent. Both histamine and 5-hydroxytryptamine content of skin in the hormone treated animals fell to about 50 per cent of the level of the control. Histamine depletion alone was observed in jejunum and spleen. Histologically degranulation of skin mast cells without disruption of cell membrane was seen. In contrast similar treatment with DCA failed to produce a decrease in the tissue levels of either of the two amines studied and did not cause any morphological change in skin mast cells.

Administration of cortisone (and hydrocortisone) in another group of animals who were pretreated with the histamine depletor, polymixin B retarded the recovery rate of histamine and remarkably enough caused a fall of 5-hydrotryptamine in the skin to 20 per cent of the control. The magnitude, but not the rate of depletion resembled that found with reserpine treatment. DCA had no effect on recovery rate of histamine nor had any effect on 5hydroxytryptamine content of skin.

The authors suggest that the above may be due to a difference between the action of gluco- and mineralo- corticoids, namely that while the former lower the activity of both histidine decarboxylase and 5-hydroxytryptophandecarboxylase the latter do not.

T. H. R.

An Inhibitory Effect of Acetylcholine on the Response of the Guineapig Ileum to Histamine by Mauren M. Dale (1958): Brit, J. Pharmacol. 13, 17.

On the isolated guineapig ileum if small doses of acetylcholine and histamine are alternately administered the response to histamine tends to diminish and may disappear. When the tissue does not respond to small doses of histamine, a contraction may be produced with larger doses. This diminution

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of the histamine response is reversible and the response reappears if the preparation is left untreated by drugs for varying periods of time. This phenomenon is exhibited even in presence of cocaine or atropine.

Y. K. S.

Continuous Recording of Acid Gastric Secretion in the Rat by M. N. Ghosh and A. O. Shild (1958): Brit. J. Pharmacol., 13, 54.

A new method for continuous recording of acid gastric secretion in the rat has been described. Rats are anaesthetised with urethane (0.5 to 0.7 m1/100 g of 25% soln). The stomach is perfused with a dilute NaOH solution through the æsophagus and the ph of the perfusate emerging from a cannula in the pylorus is registered graphically. This preparation is claimed by the authors as a suitable preparation for the bioassay of secreting stimulants. The authors have used ten or more drugs in succession in one preparation. Histamine, methacholine, carbachol and acetylcholine produce a graded reversible stimulation of acid secretion.

Y. K. S.

A Comparision of Antifibrillatory Drugs in the Heart Lung Preparation by M. J. Rand, and J. M. Walker (1958): Brit. J. Pharmacol., 13, 107.

Quinidine, amethocaine, trasentine, and atropine have been studied for their ability to prevent atrial fibrillation in the heart lung preparation of the dog by stimulating the right auricle in presence of acetylcholine. The authors suggest on the basis of their results that atropine acts by specifically inhibiting acetylcholine, but that amethocaine and transentine exhibit their action by virtue of their quinidine like properties.

Y. K. S.

Some Observation the Interaction of Reservine and LSD by M. Taeschler, and A. Cerletti (1957): J. Pharmacol. Exper. Therap. 120, 2, 179.

This study has been made in mice. The authors have found that LSD antagonised potentiating action of Reserpine on barbiturate hypnosis in mice.

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They have ascribed this effect of LSD to its central stimulant effects. Amphetamine was also found to have similar action like LSD.

They found that the stimulant action of LSD in mice was greatly increased by pretreating the animal with reserpine. BOL, another inhibitor of Serotonin (which lacks the central stimulant effect of LSD) did not inhibit the sedative and barbiturate potentiating action of reserpine although it prevented the serotonin induced potentiation of barbiturates to the same degree as LSD.

Y. K. S.

## Body Composition of Indians and Its Relation to Basal Metabolic Rate by S. Banerjee, and R. Sen, (1958). J. Appl., Physiol., 12, 29.

The authors determined basal  $O_2$  consumption and the body composition such as total body water, extracellular fluid volume, serum volume and body fat in 14 normal men and 6 women and calculated values for cell solids, cell mass and lean body mass. Basal oxygen consumption of these subjects had significantly greater correlation with cell mass or cell solids than the surface area of the body. The difference between the basal  $O_2$  consumption of these subjects and that of the people living in cold environment was much less when the oxygen consumption of these subjects was expressed in terms of cell solids or cell mass than when it was expressed in terms of the surface area of the body. The observations indicate the greater suitability of cell mass or cell solids as the reference standard for the expression of B M R.

S. B.

Urinary Excretion of Adrenal Steroids During Exercise in Hot Atmospheres by K. W. Robinson, and W. V. Macfarlane, (1958): J. Appl. Physiol., 12, 13.

The authors determined the urinary output of 17-ketosteroids, 17-ketogenic steroids, sodium and potassium in 3 healthy young males exercising at 40.5°C. With moderate exercise the excretion of the above steroids decreased by more than 50%. Urinary excretion of sodium decreased rapidly and the K/Na ratio rose during the heating period.

S. B.

**Duodenal Inhibitor Mechanism in Duodenal Ulcer Patients** by E. R. Woodward and H. Schapiro, (1958): J. Appl. Physiol., 12, 55.

Since acid in the duodenum inhibits gastric secretion as well as mobility, an impaired duodenal inhibitor mechanism might account for the hypersecretion of acid gastric juice characteristic of duodenal ulcer patient. The authors made a quantitative determination of this inhibition in patients suffering from duodenal ulcer and normal subjects. No significant difference was found between the normals and the duodenal ulcer patients. The authors concluded that a deficiency in the duodenal inhibitor machanism is probably not a factor in duodenal ulcer disease.

S. B.

Inhibiting Effect of Norepinephrine on Gastric Secretion in Human Subjects by A. J. Leonsins, and W. R. Waddell, (1958) J. Appl. Physiol., 12, 334.

The authors studied the effect of intravenous infusion of norepinephrine into human subjects on the normal gastric secretion stimulated by various agents. The principal effect noticed was reduction of the gastric secretion in all the conditions. The effect was due to vasoconstriction produced by norepinephrine. For gastric secretion, according to the authors, vasodilation in the stomach is necessary.

S. B.

Studies on Carbohydrate Metabolism in Scorbutic Guinea pigs by S., Banerjee, and D. K. Biswas, and H. D. Singh, (1958): J. Biol. Chem., 230, 261.

The authors observed that lowered glucose tolerance in scorbutic guinea pigs was improved by prolonged treatment with insulin. Liver and muscle glycogen which were greatly decreased in scorbutic guinea pigs strickingly improved when the animals received injections of insulin for a prolonged period of time. Tissue contents of citric, malic and lactic acids which were significantly increased in scorbutic guinea pigs, were lowered when the animals were treated with insulin for a prolonged period of time.

S. B.

Different Fractions of Serum Proteins in Normal and Scorbutic Guinea pigs by S., Banerjee, and K. Rohatgi, (1958): Proc. Soc. Exp. Biol. and Med., 97, 234.

The authors determined different fractions of plasma proteins by paper electrophoresis in scorbutic and paired fed normal guinea pigs. Guinea pig serum contained albumin and alpha<sub>1</sub>, alpha<sub>2</sub>, beta<sub>1</sub>, beta<sub>2</sub> and gamma fractions of globulins. There was a significant decrease in albumin, alpha<sub>2</sub> and beta<sub>2</sub> globulins; considerable increase in alpha<sub>1</sub>—globulin and no significant change in beta<sub>1</sub> and gamma—globulins in the serum of scorbutic guinea pigs.

S. B.

Nicotinic Acid—Tryptophan Metabolism in Certain Diseases by S., Banerjee, and P. S. Agarwal, (1958): Proc. Soc. Exp. Biol. and Med., 97, 65.

The authors estimated different metabolites of nicotinic acid and tryptophan excreted in the urine by normal subjects and by patients suffering from typhoid fever, cholera, small pox, cirrhosis of liver and infective hepatitis both before and after the feeding of tryptophan. After tryptophan urinary excretion of nicotinic acid metabolites increased in all diseases but the increase was minimum in patients suffering from small pox. Excretion of tryptophan diminished in all the diseases studied. Kynurenin which was initially absent appeared in the urine after the feeding of tryptophan in small pox, liver cirrhosis and hepatitis but not in cholera. 3-hydroxyanthranilic acid appeared in urine after tryptophan was fed in cholera, small pox and hepatitis but not in cirrhosis of liver. The authors believe that neither the liver, nor the gut but the body tissues are more concerned in the synthesis of nicotinic acid from tryptophan.

S. B.

## Pteroylglutamic Acid in Different Diseases by S. Banerjee and S. K. Pain, (1958): Proc. Soc. Exp. Biol. and Med., 97, 326.

The authors estimated the urinary excretions of pteroylglutamic acid (PGA) and citrovorum factor (CF) in patients suffering from cirrhosis of liver, typhoid fever, renal hypertension, essential hypertension, acuta malaria, influenza, tuberculosis, nutritional anemia and in normal subjects. Patients suffering from cirrhosis of liver, typhoid fever, renal hypertension, acute malaria, influenza, and nutritional anemia excreted diminished amounts of PGA and CF than normal persons. After feeding of PGA patients suffering from above diseases excreted less CF than normal persons under similar conditions. These excretions in patients suffering from essential hypertension and tuberculosis were similar to excretions by normal persons. The authors are of opinion that different tissues of the body seem to be concerned in conversion of PGA to CF in the body and suggest that PGA or CF should be administered in different diseased conditions.

S. B.

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