divided according to their perceptual-personality characteristics. Those with scores over 2000 on the MMPI drug reactivity (abscissa in Figure 5) display the highest amount of psychopathology at the peak of a psilocybin induced experience and are the most variable subjects on perceptual tasks without any drug. PETRIE's data[7] also suggest that certain perceptual-personality characteristics are related to the control of perceptual intake and processing[8].

*Zusammenfassung.* Die in gesunden, jungen Volontären durch Psilocybin hervorgerufene, reversible Veränderung der bevorzugten Helligkeit kann mit der Stabilität der Wahrnehmungspersönlichkeitsstruktur, nicht aber mit der mydriatischen Wirkung der Droge in Beziehung gebracht werden.

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## The Pharmacological Action of Gastrin Pentapeptide

Since the isolation and synthesis[1-3] of gastrin and gastrin pentapeptide it has become possible to study the pharmacology of gastrin action on the oxyntic cell. It has also become possible to study the puzzling relationships between gastrin, histamine and cholinergic stimuli.

Our experiments were carried out on 5 mongrel dogs with Heidenhain gastric pouches, weighing 14–16 kg. The animals were fasted for 18 h before each experiment.

Throughout the experiments gastric secretion was stimulated either by feeding or by the continuous i.v. administration of 2 μg of gastrin pentapeptide (GP5) or 10 μg of histamine/minute. When a control plateau of secretion had been reached atropine sulphate or ganglionic blocking agents (pentolinium tartrate, hexamethonium bromide or chlorisondamine chloride) were injected s.c. in various doses arranged randomly.

Collections were for 10-min periods. At least 3 were taken at each dose level and the last 2 at each dose were used for calculations. Acid was titrated to pH 7.

In Figure 1 it will be seen that pentolinium alone was without effect upon basal gastric acid secretion, but in Figure 2 it greatly enhanced the response to i.v. GP5. The same result exactly was obtained with the other 2 blocking agents. Atropine on the other hand profoundly depressed GP5 and pentolinium enhanced secretion (Figure 1). The acid response to histamine in contrast was significantly depressed by ganglionic blockade (Figure 2). This finding indicates that the mechanism by which histamine stimulates the oxyntic cell is different from that by which GP5 does. That endogenous gastrin acts in the same manner as GP5 cannot be estab-

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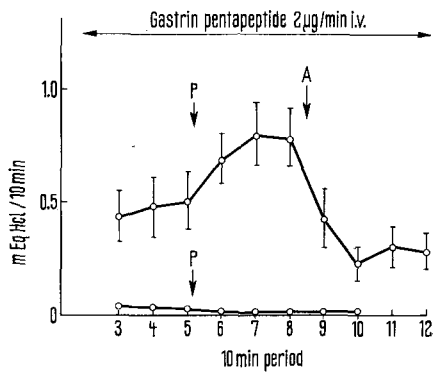


Fig. 1. The effect and standard errors of pentolinium tartrate 1 mg/kg s.c. at P on the acid secretion from unstimulated (lower curve) and GP5 stimulated Heidenhain pouches (upper curve). Atropine sulphate 1 mg s.c. was given at A. Each curve mean of 5 dogs.

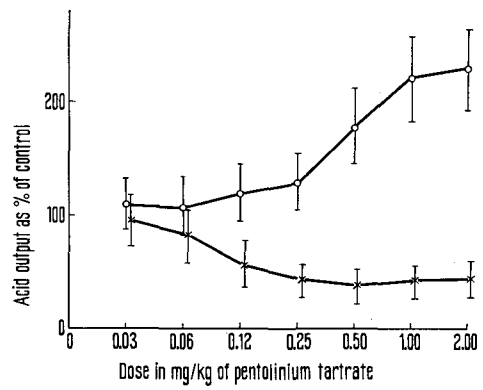


Fig. 2. The effect and standard errors of a ganglionic blocking agent on the secretion of gastric acid in response to GP5 (2 μg/min) upper curve and on histamine (10 μg/min) lower curve. Each curve mean of 5 dogs.

lished in these experiments since ganglionic blocking agents, in conformity with previous claims[4], depressed the secretion of gastrin by the antrum (Table). Dr. T. M. LIN (Eli Lilly) has obtained results (personal communication) which are in essential accord with ours.

These results suggest that gastrin pentapeptide stimulates the oxyntic cell via a mechanism which has several of the characteristics of muscarinic ganglionic stimulation (VOLLE[5]). Such ganglionic stimulation is accentuated by ganglionic blocking agents and blocked by atropine. The histamine mechanism appears to be different[6].

Effect of pentolinium tartrate (1 mg/kg s.c.) on the acid secretion mEq/10 min from Heidenhain pouches in fed dogs

| Dog | Control[a] | Pentolinium[a] |
|---|---|---|
| 1 | 0.32 | 0.21 |
| 2 | 0.79 | 0.55 |
| 3 | 0.39 | 0.12 |
| 4 | 0.12 | 0.02 |
| 5 | 1.11 | 0.57 |

S.E. of difference 0.081. $p < 0.01$. [a] Each figure mean of three10 min collections.

*Zusammenfassung.* Bei Stimulation der Magensekretion bei nichtnarkotisierten Hunden mit Heidenhain-Taschen durch Histamin und synthetisches Gastrin-Pentapeptid konnte die Wirkung des letzteren durch Ganglienhemmer vergrössert und durch Atropin vermindert werden. Die Wirkung des Histamins wurde durch Ganglienhemmer herabgesetzt. Die Versuche zeigen, dass Histamin und Gastrin-Pentapeptid verschiedene Wirkungsmechanismen besitzen.

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## Insulin-Induced Enhancement of Anaphylactoid Reaction to a Non-Carbohydrate Antigen

There is a growing body of evidence indicating that the glycemic state of an animal may influence its susceptibility to a variety of inflammatory and hypersensitivity reactions[1-5], as well as to certain physical stresses[5]. Thus, agents which lower blood sugar appear to enhance susceptibility to these stresses, whereas agents with a hyperglycemic effect may have stress-protective value[1-5].

The effect of blood glucose levels on susceptibility of rats to the anaphylactoid reaction produced by a single injection of dextran, ovomucoid and glycogen has been extensively studied. Several workers have reported that insulin-induced hypoglycemia greatly potentiates the inflammatory response elicited in rats by these agents[1,3]. It has been suggested that this effect is specific for polysaccharide antigens, or those which contain a carbohydrate moiety[1,3].

In mice a single injection of peptone elicits a severe anaphylactoid reaction which may culminate in death. Pertussis vaccine, which lowers the blood sugar levels of mice[6], enhances this reaction[7]. Propranolol, a $\beta$-adrenergic blocking drug, which potentiates insulin's hypoglycemic action[8,9], has a similar effect[7]. We therefore considered it of interest to determine the effect of exogenous insulin on the susceptibility of mice to the non-carbohydrate anaphylactoid agent, peptone.

Three groups of 10 CFW mice weighing 18–20 g (Carworth Farms) were injected i.p. with 0.8 U of regular insulin (Iletin, Lilly). 10 min later one of these groups was challenged i.p. with 37.5 mg of proteose peptone (Difco) another with 75 mg of the peptone; and the third group received no associated injection. Two additional groups of 10 mice received similar i.p. injections of peptone without insulin. Deaths were tabulated at 2 h. The results shown in the Table indicate that the preliminary injection of insulin markedly increased the susceptibility of the mice to peptone shock.

Insulin administered alone killed only 1 out of 10 mice. The low dose of peptone (37.5 mg) was non-lethal, whereas the high dose of 75 mg killed only 3 out of 10 mice. However, administration of 0.8 U of insulin followed in 10 min by an injection of either 37.5 or 75 mg of peptone resulted in 100% mortality.

The above experiment indicates that insulin, as had earlier been reported with pertussis vaccine, and propranolol[7], heightens the sensitivity of mice to peptone shock. This finding adds another to the lengthening list of mouse-sensitizing properties shared by both *B. pertussis* and insulin. In addition to their hypoglycemic effect, both agents are capable of inducing hypersensitivity to the pharmacological mediators, histamine and serotonin[6,10]. Both augment sensitivity to immediate[1,10], and

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