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 β-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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delayed-type[4,11] hypersensitivity states. KIND has shown that pertussis-inoculated mice are highly susceptible to the physical stress of reduced atmospheric pressure and low oxygen tension (hypoxic decompression)[12]. We have demonstrated a similar phenomenon in mice injected with insulin[13]. These findings are consonant with the thesis that there is an inverse relationship between the glycemic state of a host and its susceptibility to a wide variety of stressful stimuli[1-5].

Several workers have suggested that hypoglycemia, such as is induced by insulin, potentiates only those anaphylactoid reactions which are elicited by polysaccharide antigens, or those containing a carbohydrate moiety[1,3]. Evidence adduced here, and elsewhere[7], indicates that, at least in the mouse, the anaphylactoid reaction elicited by a non-carbohydrate agent, proteose-peptone, can also be exacerbated by prior administration of hypoglycemic agents[14].

Effect of insulin on susceptibility of CFW mice to peptone shock

Sensitizing agent	Dose U	Challenge agent[a]	Dose mg	Dead/ total[b]
Insulin	0.8	–	–	1/10
–	–	Peptone	37.5	0/10
–	–	Peptone	75.0	3/10
Insulin	0.8	Peptone	37.5	10/10
Insulin	0.8	Peptone	75.0	10/10

[a] Challenge agent injected 10 min after sensitizing agent. All injections i.p. [b] Deaths tabulated 2 h after challenge.

Résumé. Nous avons constaté que chez les souris l'insuline possède une sensibilité élevée au peptone anaphylactoïde non-carbohydraté. Ce résultat concorde avec l'hypothèse d'une relation inverse entre la quantité de glucose dans le sang et la sensibilité d'un hôte à une grande variété de «stressors».

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Effects of Excitatory and Tranquilizing Drugs on Visual Perception. Spatial Distortion Thresholds

While the 'excitation syndrome'[1] is the most characteristic physiological feature of the hallucinogenic (psychotomimetic or psychodysleptic) drug-produced state, the important perceptual manifestation of drugs – such as psilocybin, LSD and mescaline – concerns alterations of time and visual space. Specifically the subject not only experiences an increase in data content, that is, chronosystole or time contraction relative to the observer, but also perceives objects in nearby space as enlarged and those far off as diminished in size[2-5].

This report is concerned with alterations of visual space produced by the drug psilocybin in healthy subjects.

Changes in spatial distortion threshold (SDT) were monitored for 15 college student volunteers, median age 23 years, in response to 160–200 μg/kg doses of psilocybin. The perceptual and personality characteristics of these subjects, 9 men and 6 women, have been presented elsewhere[6].

Just prior to (T_1) at drug peak ($T_2 = T_1 + 110$ min) and following the time course of the drug ($T_3 = T_1 + 280$ min) each subject viewed a horizontal black line (15 cm by 0.7 cm) against a white 250 metercandle background placed 40 cm before the eyes in primary visual gaze. This target was viewed binocularly through a pair of counter-rotating prisms (30 mm apertures, 12 mm vertex depth). While prism power was added uniformly right and left, relative to his corrected vertical phoria the subject[7] was asked to report the first deviation of the line from 'flatness'. A set of 6 observations with prism bases down, followed by another set with prism bases up, were made at T_1, T_2 and T_3 with the mean and standard deviation of each set being used as SDT measures for those points during the drug time course.

Other measurements made on these 15 subjects included pupil diameter, brightness preference, increment threshold detection for light, the Minnesota Multiphasic Personality Inventory, and taste thresholds. These will be described in future reports[8].

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