Table 1. Isoagglutinin Changes during Influenza Infection

Influenza A_2							
	Anti-A						
No. of two-fold dilution steps	+3	+2	+1	0	−1	−2	−3
No. of cases	0	2	31	51	19	5	0
	Anti-B						
No. of two-fold dilution steps	+3	+2	+1	0	−1	−2	−3
No. of cases	1	4	36	104	48	4	1
Influenza *B*							
	Anti-A						
No. of two-fold dilution steps	+3	+2	+1	0	−1	−2	−3
No. of cases	1	0	7	16	18	0	0
	Anti-B						
No. of two-fold dilution steps	+3	+2	+1	0	−1	−2	−3
No. of cases	1	1	10	25	23	2	0

Table 2. Distribution of ABO Blood Groups

		A	O	B	AB	Total
Influenza A_2	Observed	105	93	15	8	221
	Expected	106	88	18	9	221
Influenza *B*	Observed	28	37	7	2	74
	Expected	36	30	6	2	74

influenza infection might therefore shed light on the antigenic content of this virus.

We have followed the isoagglutinin titres in paired sera collected from 221 patients with influenza A_2 and 74 patients with influenza *B* during 1961–64. The diagnostic criteria have been clinical symptoms accompanied by a 4-fold or greater increase in influenza antibody titres (complement fixation and/or hæmagglutination inhibition tests).

Our observations are summarized in Tables 1 and 2. Sixteen paired sera showing two or more titre-step difference from first to second sample have been retitrated. By this check only 3 showed the same difference, namely a 4-fold decrease in anti-A in one case and 4-fold decreases in anti-B in two cases.

It seems therefore justified to conclude that influenza infection does not produce any change in isoagglutinin titres.

The distribution of ABO blood groups among our patients with influenza A_2 corresponds to that of a normal control population, whereas the number of influenza B patients is too small for definite conclusions.

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PATHOLOGY

Effects of Adenine and Guanine on Hepatic Glucose Release and on the Action of Insulin on the Liver

We have recently obtained information from work on normal subjects and mild and severe diabetics indicating that insulin has an increasing effect on hepatic glucose metabolism in untreated diabetics with progressively elevated fasting blood sugar-levels and glycosuria implying greater degrees of catabolism and gluconeogenesis[1]. There is also evidence of a hepatic action of insulin when diabetic patients are treated with phenethylbiguanide[2]. In view of the similarity between the ethylbiguanide side-chain and the nucleotides adenine and guanine, we wished to test the effect of these compounds on hepatic carbohydrate metabolism and on the action of insulin on the liver.

Using the rat liver perfusion technique developed by one of us[3,4], the release of glucose over two successive periods of 1 h has been investigated. Control levels were measured during perfusions with glucose-free tyrode solution and the effects of added adenine (in two concentrations, designated as Adenine, 0·25 γ/ml., or adenine, 0·025 γ/ml.), guanine (designated as Guanine, 0·25 γ/ml., or guanine, 0·025 γ/ml.) alone or with insulin (in two concentrations designated as Insulin, 20 milliunits/ml., or insulin, 0·2 milliunits/ml.) on hepatic glucose release expressed in mg/h have been examined so far.

Table 1

Perfusion	No. of expts.	Hepatic glucose release (mean ± *S.D.*) 1st h	2nd h
Control	12	4·7 (1·2)	4·7 (2·9)
Insulin	8	3·7 (1·0)	3·0 (1·2)
adenine	8	5·5 (1·8)	5·1 (2·1)
Adenine	10	10·4 (2·7)	8·3 (4·0)
Adenine + Insulin	8	8·3 (1·8)	2·5 (1·1)
Adenine + insulin	8	3·6 (1·2)	3·9 (1·6)
guanine	8	4·4 (2·7)	2·7 (1·8)
Guanine	10	8·3 (1·7)	8·3 (2·0)
guanine + Insulin	8	4·2 (2·6)	1·7 (0·9)
Guanine + insulin	8	4·6 (2·5)	3·1 (2·3)

The nucleotides guanine and adenine caused statistically significant increases in hepatic glucose release. Insulin, in concentrations which in previous work[3] failed to reveal a statistically significant action in controlled circumstances, suppressed the glucose-releasing action of the nucleotides, even at the lower dose-levels, suggesting that its action is hormonal rather than stoichiometric.

The results (Table 1) appear to be in close harmony with, and provide some degree of explanation for, the clinical findings here. If we consider the state of affairs in the fasting untreated diabetic, increasing demands for gluconeogenesis to offset losses through glycosuria will be associated with increasing protein catabolism. It seems probable that the concentrations of nucleotides, no longer involved in protein synthesis, might increase in the tissues, diffuse into the blood stream and perfuse the liver as well as accumulating locally from the cells in that organ. From the present evidence, it would appear that in low concentrations nucleotides may increase hepatic glucose release and that, in such circumstances, insulin, in levels which approach the physiological for the portal circulation, has a powerful action restraining hepatic glucose release.

Further work on the role of these phenomena on glucose homeostasis is being carried out.

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Preparation of Antigens Specific of Human Breast Carcinoma by an Immunochromatographic Method

In spite of a rapidly mounting literature on the problem of specific antigens in human neoplasms, there have been few attempts to separate these antigens in amounts permitting biochemical and immunological analyses of the same. Such analyses not only would shed light on carcinogenic mechanisms, including possibly the serological back-tracking of eventual biological agents, but would make possible a much-desired immunological classification of tumours. Work from this laboratory has been concentrated on such efforts.