and that a preponderance of the latter effect in man accounts for the failure to obtain nitrogen retention with growth hormone in this species. This is entirely speculative since there is no direct evidence that growth hormone does stimulate the production of HGF.

Summary. Purified pancreatic hyperglycemic factor has been given to normal rats during a period of fasting or at the end of such a period. In the latter case HGF caused a fall in the level of ketonemia which had developed as a result of the fast, but did not affect the blood sugar. When given during the fast, HGF greatly inhibited the development of ketonemia, increased the nitrogen excretion, and inhibited to some degree the rise in liver fat; it did not affect the liver glycogen or the blood sugar. These effects differ from those of adrenal hormones and of growth hormone. It appears that HGF is capable of playing an active part in fasting metabolism.

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Influence of Smoking on Urinary Pepsinogen Excretion.* (21181)

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Whether smoking stimulates gastric secretion has not been definitely ascertained. The after-dinner smoke is believed to aid digestion and earlier workers reported that smoking stimulated gastric secretion and acidity. However, Schnedorf and Ivy(1) found that smoking produced no immediate effect on acid production but smokers had a higher gastric acidity (not statistically significant) than non-smokers. They reported that smoking had no effect on gastric evacuation but Dickson and Wilson(2) noticed a slight delay.

We have studied the effects of smoking on urinary pepsinogen excretion.

Method. To determine the excretion rate of urinary pepsinogen, the method of West, Ellis, and Scott(3) employing a casein substrate(4) was used. Twenty white male office and professional hospital workers between 18 and 45 years of age, who smoke 20 or more cigarettes per day, were examined on 5 not necessarily successive days. The results were compared with those of a control group of 20 non-smokers.

Results. The results are shown in Fig. 1. Aside from the diurnal variations(4) we found considerable day to day variation in healthy control individuals. These variations were not unexpected. They are usually within a limited range (though in rare instances they may exceed 100% of the mean value). These variations must be ascribed to the day to day

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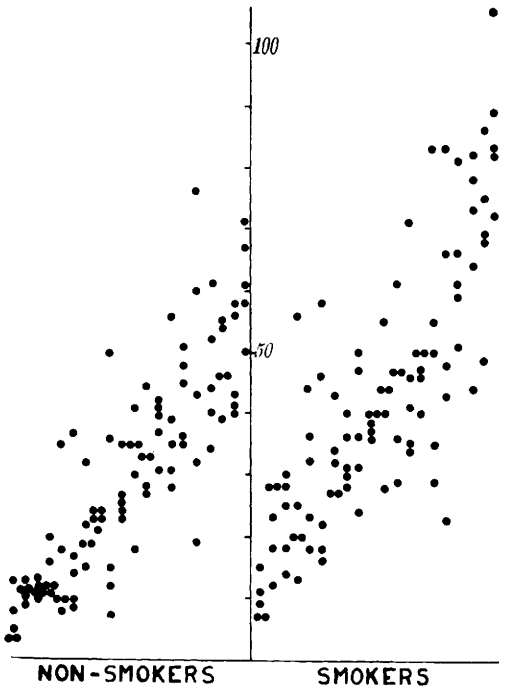

FIG. 1. Urinary pepsinogen excretion in units per hr. Each vertical column represents 5 test results on one individual.

variety of life situations. Smoking was suspected of increasing gastric secretion, either directly or indirectly.

The units of urinary pepsinogen excreted per hour in the group of smokers averaged 43 compared to the non-smokers with an average value of 30. This means that the average urinary pepsinogen excretion of smokers was 43% higher than that of non-smokers. Testing the significance of the differences between the means of the 2 samples by the Fisher "t" test(5) yields a "t" of 2.34, which is significant at $P = 0.03$. Since the direction of the difference was hypothesized, the use of a one-tailed test yields a highly significant value of $\frac{P}{2} = 0.015$.

In this study no special attention was paid to the question of whether or not the smokers did smoke immediately prior to or during the collection period. The test persons were given no instructions and it is assumed that most of them did smoke during the time in question. A group of 5 smokers submitted each to 8 further tests. Alternately, they did not smoke on the test day until after the final urinary specimen was collected, or they smoked at least 3 cigarettes during the collection period. Uropepsin excretion during smoking averaged 47 units per hour, as compared to 43 during abstinence.

Comment. The results indicate that the immediate effect of smoking on the urinary pepsinogen excretion rate, though statistically significant, is not of appreciable magnitude. This agrees with the findings of Schnedorf and Ivy, who found no immediate effect on acid production. No quantitative pepsin determinations in relation to smoking have to our knowledge been previously done. No influence of smoking on duodenal proteolytic enzyme production has been found(6). The mechanism for the prolonged effect of smoking on pepsin secretion is not clear.

Summary. The average excretion of urinary pepsinogen of habitual smokers was found to be 43% higher than that of non-smokers.

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