

as an inhibitor of hæmagglutination by heated influenza *B* virus. The *A* substance was much weaker with a titre of 1:20,000. The activity of both substances as inhibitors was destroyed by incubation with the receptor-destroying enzyme, but the *A* substance had lost none of its ability to block agglutination of human Group *A1* cells by a specific anti-*A* serum also provided by Dr. Morgan. No suitable anti-*O* serum was available for corresponding tests with the *O* substance.

Anderson (unpublished) has shown that unheated influenza viruses can produce significant destruction of the 'Francis inhibitor' in serum. Further, the receptors on the red cell which react with viruses of the group are destroyed like the inhibitor by the receptor-destroying enzyme and potassium periodate and not by any of a large number of other enzymes and chemical reagents that we have tested. This evidence seems adequate to characterize the substrate of the virus-enzyme as a mucin, and in all probability the same mucin as is responsible for the *ABO* blood group specificity. It should be stressed, however, that the serological specificity of the mucin in the red-cell surface, as in the soluble purified form, is not modified by the action of virus or the receptor-destroying enzyme.

Connective tissue mucin in the form of synovial fluid has only a slight inhibitory effect which is not affected by the receptor-destroying enzyme. In view of the importance which has been ascribed to hyaluronic acid mucins and hyaluronidases in the phenomena of bacterial invasion, it seems not out of place that those viruses which are characteristically pathogens of mucus-secreting surfaces or glands should possess an enzyme equivalent in many ways to the hyaluronidases of various pathogenic bacteria, but acting on glandular type mucin.

A full account of this work and of related studies on the enzymes produced by *V. cholerae* will be published elsewhere.

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¹ Stone, J. D., *Aust. J. Exp. Biol. and Med. Sci.*, in the press.

² Francis, T., *J. Exp. Med.*, **85**, 1 (1947).

³ Morgan, W. T. J., and van Heyningen, R., *Brit. J. Exp. Path.*, **25**, 5 (1944).

Control of Banana Wilt Disease

SOME years ago I reported in these columns an unusually interesting and important field experiment on the control of Panama (wilt) disease of bananas (*Fusarium oxysporum cubense*), which I had seen while travelling in Honduras¹. This consisted in flood-fallowing an area of about a hundred acres which had gone out of cultivation because of wilt disease. The area was empoldered (contained within earth embankments) and divided into sections, each of which was being kept submerged for a different period of time. This experiment was based on the observation that soil fungi, such as *F. oxysporum*, require oxygen to live, and that, when highly infected soil was submerged for one month under two feet of water, no living *Fusarium* could be found in it. At that time I wrote: "The outcome of this experi-

ment will be awaited with the greatest interest by all associated with the extensive alluvial banana lands of Central America". Already, at that time (1940), it had been found that in new land, built up by the sedimentation of controlled flood water, and therefore subjected to several inundations, the incidence of wilt disease was negligible.

Recently I had news of the experiment from a scientific correspondent, Dr. V. C. Dunlap, to whom I now make acknowledgment. Areas originally flooded for six, twelve and eighteen months have been producing fruit for nearly six years, and it is considered that several years of good production still lie ahead. Scattered cases of wilt disease did appear after ten to eighteen months, but the subsequent spread of the disease was slow. As in many good soils, a mat or stool may show disease in one plant while all the other plants apparently remain unaffected for two or three years. It is considered improbable that the method of flood-fallowing will ever be successful in acid soils (in which plants succumb readily and in which the fungus may spread with great rapidity), but the system is being extended to other areas considered to be suitable.

Much credit is due to the officers of the United Fruit Co. for their enterprising experimentation on a large scale.

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¹ *Nature*, **147**, 313 (1941).

A Pitfall in Correlation Theory

IT has often been assumed that when two quantities x and y are normally distributed by themselves, their joint distribution is given by the bivariate normal surface

$$f(x,y) = \frac{1}{2\pi\sqrt{1-\rho^2}} \exp\left\{-\frac{1}{2(1-\rho^2)}(x^2 - 2\rho xy + y^2)\right\},$$

x, y being expressed in standard units and ρ being their correlation coefficient. Further, for $\rho > 0$, it is supposed that the two quantities x and y increase or decrease together, while for $\rho < 0$, one quantity increases as the other decreases. I give below an example to show that both the above assumptions are ill-founded.

Suppose y is functionally related to x by a relation expressed in parametric form as

$$\left. \begin{aligned} x &= \varphi^{-1}(t) && \text{for } 0 < t < 1 \\ y &= -\varphi^{-1}(t + \frac{1}{2}) && \text{,, } 0 \leq t < \frac{1}{2} \\ &= -\varphi^{-1}(t - \frac{1}{2}) && \text{,, } \frac{1}{2} < t \leq 1 \end{aligned} \right\} \quad (1)$$

where $\varphi(x) = \frac{1}{\sqrt{2\pi}} \int_{-\infty}^x \exp(-u^2/2) du$.

Then the graph of y against x resembles the two branches of the hyperbola $xy = \text{constant}$, and if now x is normally distributed with mean 0 and standard deviation 1, it can be shown that y is normally distributed with mean 0 and standard deviation 1. The correlation coefficient between x and y is +0.3225, which is positive even though y increases as x decreases and *vice versa*. It can be shown that the joint distribution of x and y is given by

$$f(x,y) = \frac{1}{\sqrt{2\pi}} \cdot \frac{1}{\sqrt{e^{2x^2} + e^{y^2}}}$$