the solar constant of radiation, on account of the small amount of variation from month to month in the atmospheric coefficient of transmission.

Linke adopted the factor

$$T = \frac{\text{Total atmospheric depletion}}{\text{Depletion by pure dry air}}.$$

This factor becomes large in industrial cities, as is shown in the more equable climate of urban than of suburban regions.

Chapter VI treats of the total radiation received on a horizontal surface, directly from the sun and diffusely from the sky, and also from cloud sheets, either transmitted or reflected. This is an important subject, and Ångstrom's treatment of it is given in detail.

In this chapter, "Global radiation" is defined as the total radiation received from the sun, either directly, or diffusely from the sky; and in Fig. 11 are shown typical records, obtained on a day with sunshine, and also on cloudy days.

In chapter VII the radiation from the earth to the sky is considered, which is easily measured during the night hours, but which is taking place at all hours of both day and night, although under certain conditions of cloudiness its intensity becomes very low, or even negative in direction, when a cold surface is covered by warm clouds.

The seven chapters reviewed treat of the subject under consideration from the statistical standpoint. The remaining chapters consider it from the theoretical standpoint, which is quite apart from the treatment here reviewed.

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VITAMIN B_1 AND ITS USE IN MEDICINE

Vitamin B_1 and Its Use in Medicine. By ROBERT R. WILLIAMS and TOM DOUGLAS SPIES. The Macmillan Company Monographs. The Macmillan Company, 1938.

DEFICIENCY of vitamin B_1 (thiamin) is the cause of beriberi, which, since the dawn of medical history, has been one of the major health problems, as it is to-day, in populations subsisting largely upon polished rice. There is increasing evidence that deficiency of this nutrient occurs in the United States to a degree which impairs the health of a considerable number of people. It is, therefore, one of the most interesting of the food factors which are essential in the diet. Since its existence was first demonstrated by Eijkman, in 1897, thiamin has been investigated by many investigators. The greatest achievement in this field of research was the improvement of the yield of the vitamin by Dr. Williams from rice polishings, so that about an ounce of crystalline material was made available for chemical study. With this material, Dr. Williams and his associates succeeded in determining the structure of the molecule and in following up this knowledge in an astonishingly short time by its synthesis. The synthetic vitamin has now become available at moderate cost in any quantities which may be necessary for the prevention of beriberi and for clinical use.

In Part I of the book, "Vitamin B_1 and Its Use in Medicine," the authors have discussed in a concise way all aspects of thiamin deficiency which may be instructive to medically trained readers, and suggest the clinical conditions in which thiamin deficiency may be suspected. Every clinician should be familiar with the recorded experience with thiamin in therapy, and many will gain by reading this excellent discussion suggestions which they will want to apply in their practice.

Part II is devoted to the fascinating story of the researches which have yielded our present knowledge of the vitamin, its physiological role and its final synthesis.

This book should be read by every biochemist, physiologist and progressive physician. The reader may be assured that he will be entertained and instructed by it.

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SPECIAL ARTICLES

A FATAL DISEASE OF PIGEONS CAUSED BY THE VIRUS OF THE EASTERN VARIETY OF EQUINE ENCEPHALOMYELITIS

DURING the recent epidemic of equine encephalomyelitis in southeastern Massachusetts, a number of human cases of encephalitis were observed, which were shown by Fothergill, Dingle, Farber and Connerley[1] and by Webster and Wright[2] to have been due to the virus of the Eastern variety of equine encephalomyelitis. As part of our investigation of the human disease, it became of importance to consider the possibility of other host reservoirs of the virus. We were informed by a number of pigeon breeders residing in the area where the horse disease existed that during the period of the equine epidemic they had suffered unusual losses in their flocks both of special breeding stock and of the common, domestic pigeon, the species so numerous in our cities. Indeed, one of these breeders made the interesting observation to us by letter that his "losses of pigeons began to cease as soon as the cold weather set in."

[1] L. D. Fothergill, J. H. Dingle, S. Farber and M. L. Connerley, *New Eng. Jour. Med.*, 219: 411, 1938.

[2] L. T. Webster and F. H. Wright, SCIENCE, 88: 305, 1938.

We received a dead pigeon on September 20, 1938, from a breeder whose residence was adjacent to a farm on which two horses had died of the equine disease. The brain was removed, triturated in sterile sea sand, and suspended in 10.0 cc of infusion broth. After centrifugation, part of the supernatant liquid was filtered through a Berkefeld V filter. Young albino Swiss mice were injected intracerebrally with both filtered and unfiltered material (four animals in each group). All these mice died in 48 to 96 hours, having exhibited symptoms similar to those observed in mice infected with the equine virus of the Eastern variety. Bacteriological cultures of the pigeon brain suspension before injection and of the brains of the dead mice were sterile. Subsequent experiments confirmed the filterability of the infectious agent.

The identification of the pigeon virus was determined by intracerebral injection of guinea pigs previously immunized[3] against the Eastern and the Western varieties of virus, and in normal controls. Each animal received a dose of 0.15 cc of a 1:1000 dilution of mouse-brain passage virus. Of six guinea pigs immunized against the Eastern virus, two died, and from one of these we were unable to isolate the virus; the other four remained well and showed no symptoms. Two animals immunized against the Western virus and all six controls died. From one of the guinea pigs previously immunized with the Western virus, and from two of the controls, the virus was again recovered in mice. We conclude from this experiment that the virus isolated from the pigeon was the virus of the Eastern variety of equine encephalomyelitis.

Pigeons were shown to be susceptible to the virus obtained directly from the original pigeon, following intracerebral inoculation. In other experiments, pigeons were shown to be susceptible to the strain of the Eastern variety of equine virus isolated from the brain tissue of the human case elsewhere described.

Giltner and Shahan[4] first demonstrated the susceptibility of pigeons to experimental inoculation of equine encephalomyelitis virus (probably Western type). These authors and, later, Ten Broeck, Hurst and Traub,[5] suggested the possibility of pigeons as host reservoirs of the virus. The susceptibility of a number of species of birds to experimental inoculation of the virus was demonstrated by Remlinger and Bailly.[6] We have been unable to find any report of the isolation of this virus from pigeons dying from natural infection by it.

Summary: The virus of the Eastern variety of equine encephalomyelitis has been recovered from the brain of a pigeon[7] which had spontaneously contracted the disease in an area where equine encephalomyelitis was prevalent among horses.

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THE DEMONSTRATION OF PHAGE PRECURSOR IN THE BACTERIAL CELL[1]

IN their original study of the kinetics of the phage-bacterium reaction, Krueger and Northrop[2] found bacterial growth to be the essential conditioning factor for phage production. Under the conditions of their experiments any interference with bacterial growth was accompanied by a corresponding reduction in phage formation as stated in the equation:

$$\frac{P}{P_o} = \left(\frac{B}{B_o}\right)^n$$

where P = [phage] at any time, P_o = initial [phage], B = [bacteria] at any time and B_o = initial [bacteria].

More recently Krueger and Baldwin[3] have reported experimental evidence for the production of phage according to the reaction: phage precursor + phage→ phage. The precursor, apparently a normal cell metabolite, was obtained in ultrafiltrates of growing phage-susceptible cell suspensions. The essential reaction then is analogous to the autocatalytic transformation of inactive enzyme precursor into active enzyme upon the addition of small amounts of the active form to a solution of the precursor, *e.g.*, trypsinogen + trypsin→trypsin. The detection of phage precursor led to the conclusion that precursor formation, rather than bacterial growth as such, should condition phage production and that the apparent role of bacterial growth is due to the fact that both these expressions of cellular metabolism have nearly identical optimal milieus.

[3] We are indebted to Dr. R. W. G. Wyckoff and to Lt. Col. Raymond A. Kelser for providing some of the immune guinea pigs. Others were immunized by ourselves with chick-membrane vaccine kindly furnished by the Lederle Laboratories.

[4] L. T. Giltner and M. S. Shahan, SCIENCE, 78: 63, 1933.

[5] C. Ten Broeck, E. W. Hurst and E. Traub, *Jour. Exp. Med.*, 62: 677, 1935.

[6] P. Remlinger and J. Bailly, *Compt. rend. Soc. de Biol.*, 121: 146, 1936; *ibid.*, 122: 518; *ibid.*, 123: 562.

[7] Our findings in pigeons were briefly mentioned, with our permission, by Dr. Roy Feemster in an address before the American Public Health Association meeting in Kansas City, on October 26, and will be published in the *American Journal of Public Health*. They were likewise referred to by one of us (L.D.F.) in an address before a meeting of the the New England Veterinary Association in Boston, on October 26.

[1] The experimental work cited has been supported by grants-in-aid from the American Medical Association, the Research Corporation of New York and from various interested friends.

[2] A. P. Krueger and J. H. Northrop, *Jour. Gen. Physiol.*, 14: 223, 1930.

[3] A. P. Krueger and D. M. Baldwin, *Proc. Soc. Exp. Biol. and Med.*, 37: 393–395, 1937.