renal hypertension and also to determine the possible antihypertensive effects of other compounds chemically related to vitamin A.

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RENAL HYPERLIPEMIA IN DOGS

OBSERVATIONS made in studies on children suffering from nephrosis gave rise to the question whether or not the kidney itself may exert a regulatory influence on the blood lipids, disturbance of which could lead to the hyperlipemia manifested in nephrosis. This problem was studied by determining the content of total fat and of total and free cholesterol in the blood serum of 18 dogs which had been subjected to nephrectomies[1] or to subcutaneous injections of bichloride of mercury, uranium nitrate or potassium bichromate.

Bilateral nephrectomy, performed on three dogs, was followed by a continuous rise in the level of serum cholesterol. The effect observed in one of these dogs after the second kidney had been removed is shown in

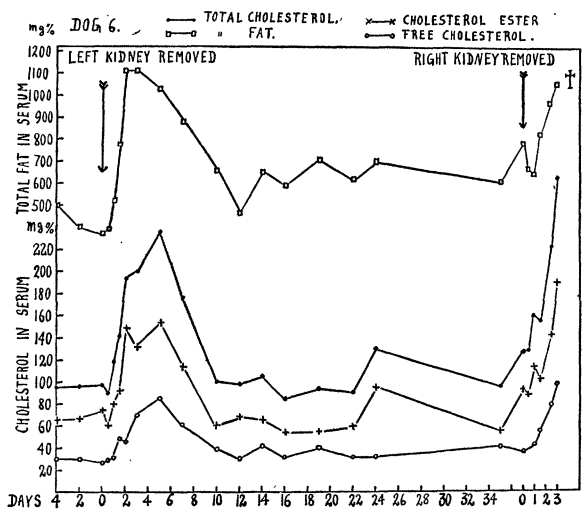

FIG. 1

Fig. 1. It can be seen that the total fat content increases beyond the extent that could be accounted for by the increase in cholesterol, thus indicating that lecithin and probably also fatty acids and neutral fat participate in this increase.

The effect of unilateral nephrectomy was studied in two dogs. In both animals the level of blood lipids rose for from 4 to 7 days and then returned to normal in 12 or 14 days after the operation (Fig. 1). The increase is obviously connected with the sudden removal of one kidney, while the return to normal level may well be due to the subsequent hypertrophy of the remaining kidney. A sham operation performed as control did not influence the blood lipid level.

In 10 dogs a single dose of bichloride of mercury administered subcutaneously was followed in every instance by an increase in the content of total fat as well as of free cholesterol and cholesterol ester. One lethal dose of 16 mg per kilogram of body weight led to a continuous increase until death. When a smaller dose was injected, however, the resulting hyperlipemia subsided and the values returned to normal. The results of one of the eight experiments carried out with a single injection of 5 mg of bichloride of mercury per kilogram of body weight are shown in Fig. 2. The resulting hyperlipemia is not

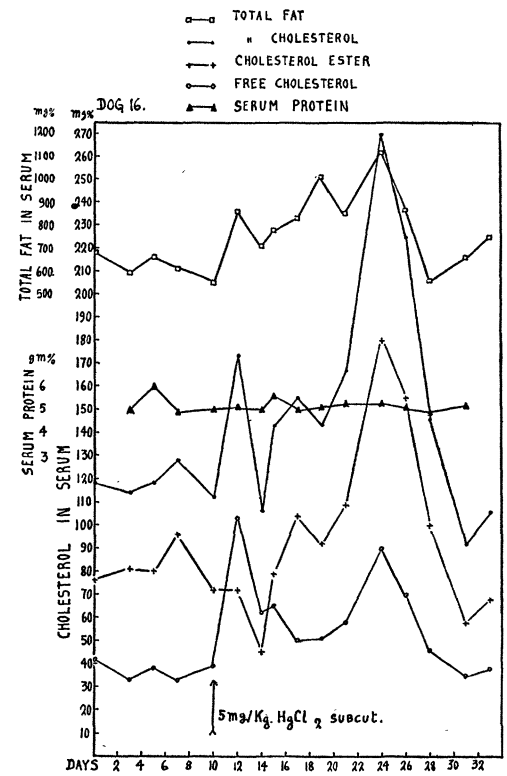

FIG. 2

accompanied by a decrease in the content of serum protein. This observation is of importance in connection with the theory which explains the hyperlipemia in nephrosis on the basis of hypoproteinemia. A smaller dose of bichloride (2 mg per kilogram of body weight) was injected intramuscularly in two other dogs twice a week for between three and four weeks. In these dogs hyperlipemia developed slowly and the level of total fat and of cholesterol continued to rise as long as the injections were given.

Subcutaneous injection of potassium bichromate (7 mg per kilogram of body weight) in one dog and of uranium nitrate (6 mg per kilogram of body weight)

[1] I am indebted to Dr. Harry Goldblatt and Dr. Joseph R. Kahn, of the Institute of Pathology, School of Medicine, Western Reserve University, for performing the operations on the dogs.

in another dog also brought about an increase in the content of total fat and cholesterol in the blood serum to over 100 per cent. of the original amount.

Conclusion: The tubular apparatus of the kidneys of dogs possesses a regulatory influence on the blood lipids such as has hitherto been unknown and is still unexplained. It is probable that the human kidney exerts the same function, but this has not yet been proved. A disturbance of this function would explain the hyperlipemia observed in nephrosis better than any hypothesis thus far advanced.

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ASSOCIATION OF TOBACCO LEAFSPOT BACTERIA WITH ROOTS OF CROP PLANTS[1]

SINCE the discovery, more than 20 years ago, of the causal agents of wildfire (*Bacterium tabacum* W. and F.) and angular leafspot (*B. angulatum* F. and M.) diseases of tobacco, no satisfactory explanation of the sources of inoculum in tobacco plant beds has been advanced. Even if a new plant-bed site is used, the plant-bed soil steamed or burned, a new cotton cover used, and disease-free seed planted, angular leafspot and, to a less extent, wildfire may appear throughout an entire Burley tobacco bed or large sections of it after a few hours of cool, wet weather. The fact that the amount of disease is usually so great seems to eliminate the possibility that the bacteria might have originated in trash from a previous infected tobacco crop. One fact is known which has a bearing on the source of inoculum; namely, that one or two applications of Bordeaux mixture sprinkled on the surface of the soil when the plants are very small or even before the seeds germinate will completely protect the leaves from infection in the bed.

In searching for the source of inoculum we found that,[2] in field soils naturally contaminated with these organisms from a previous infected crop, the organisms survived the winter at least until plant-bed time, and could be isolated by proper technic. The same was true of plots of soil out-of-doors artificially inoculated in the fall. Attempts to isolate the organisms from plant-bed soil in the spring of 1941, however, where subsequently one or the other disease developed, resulted only in failure. Occasional failure also resulted in attempts to isolate the organisms from artificially and naturally contaminated soil in which cover crops were growing. These erratic results suggested that the bacteria might be living on or in the roots of cover crops in the contaminated soil and that infection of leaves in the plant bed might follow multiplication of the bacteria on the roots of young tobacco or other plants growing in the bed.

In testing this hypothesis it was found that heavy infection frequently resulted when the roots of cover crops, including wheat, barley, rye, crimson clover and vetch, were washed free from soil in running water, ground in a mortar, diluted with water and poured over the surface of artificially water-soaked tobacco leaves. The roots were obtained both from artificially contaminated soils out-of-doors and from fields where the diseases were known to have been severe in 1941. Roots of tobacco from plant beds naturally infected with wildfire or angular leafspot also gave heavy infection when washed and used as inoculum. *B. angulatum* has also been isolated from the roots of seedling tobacco plants before the disease appeared on the leaves in untreated beds, and has been isolated from the roots of tobacco plants in beds treated with Bordeaux mixture. It is likely, therefore, that both organisms may be carried from the plant bed to the field on the roots of "healthy" plants and be the source of sudden outbreaks in the field following a protracted period of wet weather.

A microscopic examination of tobacco rootlets from naturally infected plant beds and from artificially inoculated tobacco roots growing in sand revealed masses of bacteria, at intervals on the roots, which appeared to be embedded in a matrix, for occasional bacteria which became separated from the surface developed motility while the others showed no movement whatever. Bits of roots bearing these colonies when used as inoculum produced heavy infection of either angular leafspot or wildfire, depending on the source, on water-soaked tobacco leaves.

The causal bacteria of these diseases can maintain themselves on the roots of several unrelated crop plants for at least six months, and can under certain natural conditions cause specific leafspot diseases of several unrelated plants, such as tobacco, tomato, morning-glory and cowpeas. These results seem to give support to the belief that the senior writer has had for many years; namely, that these bacteria are not primarily tobacco pathogens. They appear to be common (but specific) organisms present on roots, perhaps of native vegetation, which can and do, under special favorable circumstances, cause specific leafspots of tobacco.

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[1] The investigation reported in this paper is in connection with a project of the Kentucky Agricultural Experiment Station and is published by permission of the director.

[2] S. Diachun, W. D. Valleau, E. M. Johnson, *Phytopathology*, 32: 2, 1942.