coming even less than that for the normal subjects on high sodium intake. The $C_{\frac{1}{2}}$ periods in the control subjects were essentially the $B_{\frac{1}{2}}$ periods. This is less likely to be true for the abnormal subjects.

The $C_{\frac{1}{2}}$ and $U_{\frac{1}{2}}$ periods were influenced by measures which influenced sodium metabolism and excretion, such as sodium intake, desoxycorticosterone acetate, mercurial diuretics, water intake, and pitressin. Fig. 1 shows the influence of sodium intake on the $C_{\frac{1}{2}}$ and $U_{\frac{1}{2}}$ periods.

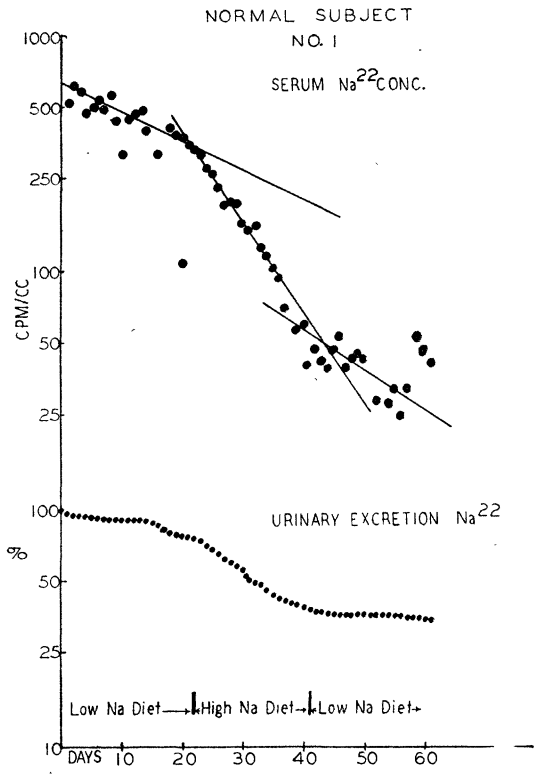

FIG. 1. Semilogarithmic graphs of changes in serum Na^{22} concentration (cpm/cc) and the rate of urinary excretion of Na^{22} (per cent of injected Na^{22} not eliminated in the urine) in Normal Subject No. 1.

The relationships of the changes in the rate of decrease in serum concentration and rate of urinary excretion to the changes in dietary sodium are shown. During the first 22 days while the subject was on a low Na diet (< 1.7 gm of NaCl/day) the rates were such that $C_{\frac{1}{2}}$ was 25 days and $U_{\frac{1}{2}}$ 100. During the next 19 days while the subject was on a high Na diet (13.7 gm of NaCl/day) $C_{\frac{1}{2}}$ was reduced to 8 days and $U_{\frac{1}{2}}$ to 19. During the last 19 days the subject was again on a low Na diet and antidiuretics. $C_{\frac{1}{2}}$ increased to 18 days and $U_{\frac{1}{2}}$ to 250.

These experiments have shown that the $B_{\frac{1}{2}}$ as well as the $C_{\frac{1}{2}}$ and $U_{\frac{1}{2}}$ for sodium are quite variable, being influenced not only by normal physiologic phenomena but particularly by disease and drugs. These variations must be taken into consideration when calculating the safety doses for radiosodium. $C_{\frac{1}{2}}$ and $U_{\frac{1}{2}}$ periods found for Na^{22} indicate the length of time required to turn over Na^{23} in man. The experiments will be published in detail elsewhere.

Isolation From Wild Bird Mites (*Liponyssus sylviarum*) of a Virus or Mixture of Viruses From Which St. Louis and Western Equine Encephalitis Viruses Have Been Obtained[1]

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In a previous communication by Reeves, Hammon, Furman, McClure, and Brookman (*1*) it was reported that in addition to three strains of Western equine encephalomyelitis virus isolated from mites, *Liponyssus sylviarum* (Canestrini and Fanzago),[2] found in the nest of a yellow-headed blackbird, another virus was isolated which was not as yet identified. Several months of laboratory work, including serial passages in several species of animals and extensive immunological tests, have led to the results summarized in this preliminary paper. When the studies are complete, they will be reported elsewhere in detail.

This agent, following isolation in mice and after several serial mouse passages, killed mice, guinea pigs, and chick embryos, but failed to kill guinea pigs which previously had been vaccinated with Western equine virus. It was not neutralized by hyperimmune Western equine serum or by St. Louis or Japanese B serum alone, yet a mixture of the three was effective in neutralizing the virus. A complement-fixing antigen prepared from the brains of mice infected with this virus reacted with specific antisera against Western equine, St. Louis, and Japanese B viruses, and antigens prepared from each of these three viruses in turn reacted with the sera from animals immunized against the mite virus. In cross-vaccination tests, however, there was no immunity in either direction in so far as Japanese B virus was concerned.

After 8 serial passages in mice the virus had only the immunological characteristics of St. Louis virus, and it would not kill guinea pigs. After 10 passages in chick embryos it had only the characteristics of a Western equine virus.

Two possibilities presented themselves: (1) that this was a simple mixture of two viruses or (2) that it was a stem virus maintained by mite-to-bird passage which could develop as either virus after passage in more selective hosts. The first possibility appeared more likely; but the second was challenging and not incredible, since these

[1] This investigation was carried out in collaboration with the Commission on Virus and Rickettsial Diseases, Army Epidemiological Board, Preventive Medicine Division, Office of the Surgeon General, U. S. Army, aided by a grant from the National Foundation for Infantile Paralysis, and under a contract with the California State Department of Public Health.

[2] These mites were collected and identified by entomologists and the ornithologist of the Neurotropic Virus Research Unit, including W. C. Reeves, D. P. Furman, B. Brookman, and H. E. McClure.

two viruses have been found so frequently in close association and appear to have the same vectors and vertebrate hosts. There is some further support for the latter possibility in the following results of two experiments. (1) Laboratory mixtures of known strains of these two encephalitis viruses, although not interfering completely in mice, are maintained with great difficulty through two passages. The Western equine virus is greatly inhibited, and attempts to recover it beyond the third passage have all failed. (2) Guinea pigs are killed promptly with signs typical of the Western encephalitis by a suspension of the brains of mice which die after an inoculation with 100 LD_{50} (third mouse passage) of the mite virus mixed and incubated with an equal volume of undiluted hyperimmune Western equine serum. The same suspension of mouse brain which killed normal guinea pigs failed to kill Western equine immune pigs. Thus, a high dilution of a third mouse passage of the mite agent, mixed with a great theoretical excess of Western equine immune serum, still maintained through passage a large amount of the equine-like component.

Until more convincing evidence can be presented that this agent represents a "new" virus, however, it appears to be more appropriate merely to report that a virus, or mixture of viruses, has been isolated from wild bird mites which, after serial passage in the brains of mice, may be identified as a strain of St. Louis encephalitis virus, and that a factor identifiable as the virus of Western equine encephalomyelitis is obtained after the agent is passed serially through chick embryos.

Reference

1. Reeves, W. C., Hammon, W. McD., Furman, D. P., McClure, H. E., and Brookman, B. *Science*, 1947. **105**, 411.

Periconia circinata, the Cause of Milo Disease

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The milo disease, which attacks the roots of susceptible milos and dwarfs and kills the plants, was first observed in Texas in 1924, and within a few years was found to occur also in other sorghum-growing states of the Southwest. For a time this disease threatened the growing of milo in certain areas of the Southwest. Resistant milos were developed, however, and now the severe losses formerly caused by the disease have been almost entirely eliminated.

For many years the exact cause of milo disease remained unsolved. The fact that susceptible milos grown in infested soil that had been sterilized by steam or chemicals remained free from the disease indicated that it was caused by a soil-borne organism. In 1937, Elliott, *et al.* (*1*) reported that the fungus *Pythium arrhenomanes* had been repeatedly isolated from the roots of susceptible milo plants grown in infested soil and affected with milo disease, and that this fungus was thought to be the causal organism. It had been previously found to be the cause of a common root rot of corn and sugarcane. It proved to be the cause also of a certain type of root rot in sorghum (*1*). It has never been demonstrated, however, that milo selections resistant to milo disease are resistant also to attack by *P. arrhenomanes*. The only logical conclusion, therefore, is that *P. arrhenomanes*, although able to attack the roots of sorghum, is not the fungus that causes milo disease.

Experiments have been in progress since 1942 at the Plant Industry Station, Beltsville, Maryland, to determine definitely the real cause of milo disease. Numerous isolations of fungi were made from the roots of susceptible milo plants grown in soil from infested fields and affected with typical milo disease. These fungi were grown on suitable media, and the cultures were used to inoculate separate portions of steam-sterilized soil. Selections of milo, resistant or susceptible to milo disease, were grown in these different lots of inoculated soil, and the growth of the plants was compared with the growth of these same selections in naturally infested soil and in steam-sterilized uninoculated soil. Some of these fungi reduced the percentage of emergence, caused damping-off, or produced other disease symptoms, but always with equal severity in selections resistant or susceptible to the milo disease. All isolates, except one, failed to produce results identical with those obtained in naturally infested soil. This one exception was a fungus that produced a mouse-gray mycelial growth on potato-dextrose agar. This growth later turned darker, due to the production of great numbers of large, black spores. The fungus was identified as *Periconia circinata* (Mang.) Sacc. When selections of milo susceptible or resistant to the milo disease were grown in the greenhouse in flats and beds of steam-sterilized soil inoculated with this fungus, the susceptible milo plants displayed symptoms identical with those shown by these same selections grown in naturally infested soil, while the resistant milos were not affected. Susceptible plants grown in lightly inoculated soil showed symptoms of milo disease similar to those seen in plants growing in lightly infested fields. Plants grown in heavily inoculated soil produced more severe symptoms similar to those seen in plants growing in infested fields that had been planted to milo for several years. Resistant and susceptible selections of each of 8 milo varieties yielded identical results when grown in flats of *Periconia*-inoculated soil and in flats of naturally infested soil. In each case the susceptible selections were killed, while the resistant selections were not affected. Typical milo disease was produced also in susceptible milo grown in an outdoor bed of steamed soil inoculated with *P. circinata*, while plants of resistant milo were not affected. Identical results were obtained in a similar adjacent bed containing naturally infested soil. In all these experiments in which susceptible milo was affected with typical milo disease when