significance. The excellent performance of the dogs on mineralized milk until reproduction indicates that milk is adequate in vitamin E during the growing period for which milk is intended.

Summary. A deficiency in pups produced from dogs maintained for long periods of time on mineralized evaporated milk has been described. The condition is undoubtedly identical with muscle dystrophy previously described in rats, guinea pigs and rabbits and is cured by synthetic α-tocopherol if therapy is initiated before the symptoms are too far advanced.

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Infection of Guinea Pigs by Application of Virus of Lymphocytic Choriomeningitis to Their Normal Skins.

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The virus of lymphocytic choriomeningitis has been found to be infective for animals by a variety of routes[1–4] including, in addition to the more common ones, the intranasal,[2] intravaginal, intraurethral[5] and intrarectal.[6] Furthermore, it has been reported by Findlay and Stern[3] that, when this virus was fed to mice or applied to their lightly scarified skins, the mice did not exhibit apparent infection but the virus could be recovered from their spleens and kidneys. They also showed that when the virus was rubbed on the lightly scarified skins of 2 Rhesus monkeys, one showed a slight febrile reaction, the other no response. Recently, Shaughnessy and Milzer[7] demonstrated that the virus caused typical symptoms of the disease in guinea pigs when placed on their very lightly scarified skins.

The W. E. strain[8] of lymphocytic choriomeningitis virus was employed in these studies. Its virulence was such that, when 0.25 cc

1 Armstrong, C., and Lillie, E. D., *Pub. Health Rep.*, 1934, **49**, 1019.

2 Traub, E., *J. Exp. Med.*, 1936, **63**, 533.

3 Findlay, E. M., Alcock, N. S., and Stern, R. O., *Lancet*, 1936, **1**, 650.

4 Lepine, P., Kreis, B., and Sautter, V., *Compt. rend. Soc. biol.*, 1937, **124**, 420.

5 Wooley, J. S., Armstrong, C., and Onstott, R. H., *Pub. Health Rep.*, 1937, **52**, 1107.

6 Shaughnessy, H. J., and Zichis, J., unpublished studies.

7 Shaughnessy, H. J., and Milzer, A., *Am. J. Pub. Health*, 1937, **29**, 1103.

8 Scott, T. F. M., and Rivers, T. M., *J. Exp. Med.*, 1936, **63**, 397.

of a 1% suspension of an infected guinea pig brain was injected intracerebrally into guinea pigs, they showed signs of infection within 48 hours and died 6 to 8 days after exposure. In these experiments the inoculum consisted of a 10% suspension of infected guinea pig brain in heart infusion broth.

The guinea pigs and mice used were obtained from healthy stocks and it was demonstrated that they were not carriers of nor immune to the virus. The animals were also examined and found to be free of ecto-parasites.

The guinea pigs were exposed by placing from 0.5 cc to 1.2 cc of the virus suspension on the normal skin of the lateral dorso-lumbar region. The hairs were spread apart by means of the tip of a glass Luer syringe. At the same time the desired amount of virus was carefully deposited upon the skin from the same syringe without even touching the syringe to the skin. The inoculum did not dry to form a crust which might abrade the skin. The skin of the animals was examined with a hand lens for abrasions but none could be found. In order to minimize the chances of infection by the many possible means resulting from such exposure, screw-top screen capsules[9] were employed. The hair of the animals was not cut and the capsules were attached by means of an adhesive tape girdle. The virus was placed on the skin through the opening of the capsule, the cover of which was then replaced. This procedure was controlled by exposing animals in the same manner without the screen capsules. In addition, attempts were made to infect guinea pigs by spreading the virus suspension on their food and the litter in the cages.

The animals were kept in individual cages of solid construction with wire mesh covers. The temperature and condition of each animal were recorded daily. Following the observations of each animal and before observing the next animal, the operator thoroughly washed his gloved hands in a 20% solution of cresol compound, followed by washing with soap and water. Temperatures were taken of each animal with individual thermometers which were kept between observations in individual bottles containing 10% formalin. This technic was controlled by placing guinea pigs, to whose skins virus was not applied, between the exposed pigs and by subjecting them to the same technic of observation. In addition, the experiment was controlled by exposing groups of guinea pigs in rooms other than the one containing the intracerebrally inoculated virus control. Each group was observed and cared for by a different person.

In each experiment an identification of the virus was established

[9] Jellison, W. S., and Philip, C. B., *Pub. Health Rep.*, 1933, **48**, 1081.

by cultural studies, autopsy findings, transfer of the virus by subdural injection to Swiss mice, and in selected cases by neutralization tests with a specific immune serum.

Fifty guinea pigs were employed in this study. Thirty animals were exposed by placing a suspension of the virus on their normal skins. Of this number, screw-top capsules were attached to 18. Five pigs were inoculated intracerebrally to establish the virulence of the virus, and 10 were used as non-exposed controls. The remaining 5 animals were exposed by spreading the virus on the food and the litter in the cages.

Thirteen of the guinea pigs with the capsules died as a result of virus infection, 2 died of unknown causes and 3 survived without showing any clinical signs of infection. Of the 12 animals without the capsules, 8 died of virus infection and 4 survived without showing any apparent infection. The 5 pigs that were injected intracerebrally showed typical signs of infection and died. None of the control guinea pigs became infected.

In these experiments it was not possible to produce clinical infection in guinea pigs when the virus was spread on the food and litter of the cages. This was possibly due to less intimate contact of the virus with the skin, and to attenuation of the virus by exposure to drying and other physical factors. Our results are in general agreement with those of Traub.[10]

It is realized that minute abrasions, not visible with a hand lens, may have been present in the skins of these guinea pigs. However, any circumstances of this nature would be a factor encountered in any normal skin.

In view of these facts, it is believed that these results indicate that the virus of lymphocytic choriomeningitis may infect guinea pigs through the normal, apparently intact, skin.

[10] Traub, E., *J. Exp. Med.*, 1936, **63**, 183.