the control group exhibited marked symptoms of distemper. The first death occurred on the twenty-fifth day, and at the end of 36 days all the animals in this group had died of typical distemper.

Group B. In this group, which was inoculated simultaneously with virulent distemper virus and distemperoid virus, 2 foxes showed symptoms on the sixteenth day. By the twenty-third day, 5 animals showed some symptoms of distemper. However, they recovered promptly, and by the thirty-first day all appeared well.

Group C. In the group receiving distemperoid virus 3 days after the inoculation of virulent distemper virus, only 2 foxes showed symptoms. These became sick on the twenty-third day and were normal again on the thirty-first day.

Group D. One of the animals receiving the distemperoid virus 12 days after the virulent distemper virus showed symptoms on the nineteenth day, and 4 of this group showed symptoms by the twenty-third day; all had recovered by the thirty-second day.

The decisive results obtained in this study are further accentuated by the fact that foxes seldom recover from distemper once they have exhibited any symptoms whatsoever. In this case, the distemper virus used was a fox-passage virus of high virulence for that animal. From the results, it seems definitely established that there is a marked antagonistic effect of the distemperoid virus on a virulent distemper infection, and this effect appears sufficiently significant to be utilized practically in the treatment of distemper. In fact, in this experiment the antagonistic effect of the distemperoid vaccine was just as great when the ferret-passage virus was used 12 days after inoculation of virulent virus as when it was used within an hour after such inoculation; and in both cases the distemperoid vaccine prevented any fatalities. Our results corroborate our earlier clinical observations on foxes and those of Stader and Slaughenhaupt on dogs, as well as the experimental observations of Schlotthauer. In addition, a definite therapeutic effect of the modified virus on the virulent infection seems demonstrated for the incubation period and early stage of the infection. Thus, it appears that a modified virus may be useful for both the prevention and the therapeutic treatment of a disease caused by a virulent strain of the same virus.

Summary

A distemper virus modified by ferret passage so as to become a harmless vaccine for foxes and dogs exhibits the interference or cell-blockade phenomenon with respect to a virulent distemper infection in foxes. Ten control foxes receiving virulent distemper virus died, while 30 foxes receiving distemperoid virus in addition lived.

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Availability of Carotene From Kale

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Measurement of the vitamin A value of vegetables presents considerable difficulty. Figures quite disproportionate to known biological values have been reported by various investigators for the chemical determination of the carotene content of certain vegetables. Different vegetables of equal carotene content have been found to vary in their vitamin A value as determined by biological assay. This fact has led to the assumption that when carotene is ingested in the form of vegetable foods, it is less effective than when taken in pure form. It has, at times, been suggested that the discrepancies may be explained by the known inaccuracies of the biological estimations, coupled with the problem of obtaining representative samples. However, it is unlikely that the errors of all workers in this field should be consistently in the same direction. Therefore, the true relationship between the carotene content of vegetables as measured by chemical analysis and bio-assay is of fundamental importance.

As a part of an investigation on the factors influencing the utilization of carotene from vegetable foods, experiments were devised for the study of kale to determine whether or not digestibility of the vegetable tissue is one of the factors causing these differences. These experiments were designed so that chemical chromatographic analyses for carotene were made of the kale parallel with bio-assays on both the kale itself and an extract of the carotenoids from the vegetable.

Sixty-four chemical analyses were made, and 138 rats were used in the biological assays. Eighteen different lots of kale, purchased on the Washington market, have been used in this study, one bushel being obtained for analysis weekly. Since kale is commonly eaten by human beings in the cooked form, the analyses were made on cooked kale. In addition, cooking the kale served to inactivate the enzymes destructive of carotene, thus eliminating one factor that is impossible to control in bio-assays. The kale was thoroughly washed with distilled water and steam cooked. It was then spread on trays, cooled, and a representative sample of appropriate size withdrawn. The leaves were stripped along the midrib so that none of either the midrib or petiole was included. One-half of each leaf blade was removed for the biological assays, and the corresponding half was used in preparing the extract which was analyzed biologically and chemically. The chlorophylls and xanthophylls were removed by saponification with potassium hydroxide, leaving the carotenoid pigments in the solution. Aliquots were taken from the extract for chromatographing and determining β-carotene spectrophotometrically; the remaining extract was concentrated in cottonseed oil containing hydroquinone and was analyzed biologically.

The measurement of β-carotene was accomplished by a procedure devised in our laboratories based on the methods of Moore and Ely (*1*), using acetone, absolute methanol, and Skellysolve F as extractants and magnesium oxide and magnesium carbonate (1:9) as adsorbents.

The biological assays were performed essentially according to the *U. S. Pharmacopoeia* (11th ed.) method using as the standard of reference pure β-carotene in cottonseed oil prepared in our laboratories.

The data shown in Table 1 represent the average vitamin A values of cooked kale as determined on the vegetable itself and on an extract of the kale.

TABLE 1

Description of sample	Chemical assay*	Bio-assay
	IU/gram	IU/gram
Kale, cooked	90	60
Extract of cooked kale	90	84

* Determined as carotene and converted to vitamin A value, using 0.6 μg. carotene as equivalent to 1 international unit.

From these figures it appears that the vitamin A value of kale as determined by bio-assay is about two-thirds of the value measured spectrophotometrically after chromatographing. On the other hand, when the carotenoid pigments are completely extracted from kale and this extract is used as the vitamin A supplement instead of kale itself, the bio-assay gives a vitamin A value of only 6 per cent less than that obtained by the chemical method. This is well within the experimental error of the bio-assay method and near the upper range of error of the chemical method. This evidence suggests that incomplete digestion of the kale, and therefore incomplete absorption of the vegetable carotene in the intestinal tract of the animal, may be an important factor causing the difference between the vitamin A values of kale obtained by chemical and biological methods.

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The Growth and Distribution of Murine Encephalomyelitis Virus in the Developing Chick Embryo[1]

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Gard (*2*) has reported the transfer of Theiler's (*5*) FA "mouse poliomyelitis" virus to the developing chick embryo and propagation through four passages. Using the chorioallantoic technique, Gard demonstrated the presence of virus only in the central nervous system (CNS) of embryos which had been inoculated at the age of five, six, and seven days. In eggs inoculated at eight days of age, no virus was recovered from any site. In no instance was virus discovered in membranes or in the corpus minus the brain and vertebral column. Also, CNS harvested 10 days after inoculation was infective for mice inoculated intracerebrally, but CNS harvested 5 days after inoculation was noninfective when tested in the same manner. In Gard's experiments, all eggs were incubated at 37° C. before, as well as after, inoculation. The inoculum consisted of 0.05 ml. of bacteriologically sterile 10-per cent suspension of infected mouse brain. Gard also was successful in transferring the UFI strain of "mouse poliomyelitis" to eggs.

Jungeblut and Sanders (*3*) reported the successful propagation of a murine neurotropic virus in embryonic mouse brain and embryonic guinea pig brain cultures. Using similar tissue cultures, Parker and Hollender (*4*) also were recently successful in propagating the GD VII strain of Theiler's virus through 20 passages.

In the present work the FA strain of Theiler's

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