the same techniques reported here, it is possible for large numbers of cells in the peripheral blood to take up tritium-labeled thymidine. With the normal individuals, increase of the incubation time with tritium-labeled thymidine up to 24 hours did not increase the percentage of labeled cells. It thus appears reasonable that in the normal individual only a very small percentage of the cells of the peripheral blood are synthesizing DNA, and these presumably are capable of division. However, the average human being of 60 kg with 7 percent blood volume and 7000 leukocytes/mm^3 has approximately $(0.06 \times 7000 \times 4200 \times 1000)/100$, or approximately 18×10^6 potentially dividing cells in the peripheral blood at one time and presumed to be in transit.

The findings that histiocytes, or specific cells of the reticuloendothelial system, may be multipotential in character, and may be transported to needed sites normally via the blood stream, is pertinent in connection with protection against X-radiation by parabiosis (*11*) and by regionally fractionated exposures (*12*). In the later experiments, exposure of one-half of the body only was followed in a few minutes by exposure of only the other half of the body. Mortality was less than it was in animals that received equivalent single-dose total-body irradiation. Normally circulating multipotential cells would explain these findings. The hypothesis is supported by estimates that only a very few intact cells are required to repopulate a radiation-depleted marrow population (*13*).

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26 February 1958

Two Syndromes Caused by Sweetpotato Viruses

Unpublished studies on the nature, host range, and seasonal development of the sweetpotato virus complex have distinguished two distinct virus syndromes, those of feathery mottle and internal cork.

In insect transmission experiments begun in 1954, of which a preliminary report was published (*1*), the aphids *Myzus persicae* (Sulz.) and *Macrosiphum solanifolil* (Ashm.) transmitted a virus from sweetpotato to sweetpotato that was distinguished by chlorotic spots on the foliage, which are typical of the internal cork syndrome. The aphids picked up the virus from two sources: (i) plants of the Porto Rico variety showing the internal cork virosis and (ii) plants of a Russian sweetpotato (plant introduction No. 105945) showing the feathery mottle virosis.

On the basis of the "flush of growth" indexing technique (*2*), which requires about 2 months, there was 100 percent transmission of the chlorotic spot symptom of the internal cork syndrome. Since the syndrome caused by the feathery mottle virus did not appear in plants inoculated with aphids fed on plants of the Russian sweetpotato, it appeared that the aphid was not the vector of the virus.

On the basis of the necrotic root symptom (*1*) of the internal cork syndrome (after storage of roots for 6 months at temperature of 75°F and at 90 percent relative humidity) there was approximately 20-percent transmission, both by insects and by grafting, under greenhouse conditions. The very mild root-symptom expression and the low percentage of incidence must be balanced against the fact that 100 percent of the roots produced some sprouts showing the same virus syndrome. It appears that the development of root symptoms requires a longer incubation period, or multiple inoculations such as undoubtedly occur in the field.

Further studies revealed that there is a definite but limited amount of natural transmission of the feathery mottle virus in the field. Such transmission is revealed only after harvest, in the sprout growth of the bedded, infected roots. The 1957 season was unusual because it stimulated an extended vegetative growth throughout September, after the growth had come to a halt during the summer drought in August, and there was positive evidence of current-season transmission of feathery mottle virus (Fig. 1*B*). Moreover, when the "flush of growth" technique (*2*) was used in the greenhouse with material harvested from the field, there was a much higher incidence of infection than is normally observed. The current-season symptoms, which appeared only on the terminal growth, were similar to those reported by Sheffield (*3*) and could be manifestations of the same disease.

A very important feature of the earlier transmission experiments involving internal cork was the discovery that the original source plant of feathery mottle virus (Russian sweetpotato, P.I. 105945) also contained the internal cork virus, or viruses, and that only the cork virus was transmitted by aphids. This feathery mottle source plant, which descended in a clonal line from plants used by Doolittle and Harter (*4*) in their work on feathery mottle and which was listed by them as No. 029878, also exhibited typical chlorotic spot of the internal cork virus syndrome, and this symptom has been constantly present in this seedling in subsequent studies.

That sweetpotato P.I. 105945 carried both viruses may possibly account for the fact that Webb (*5*) and Webb and Larson (*6*) were able to obtain chlorotic spotting in plants inoculated with virus from their feathery mottle source plants by means of the aphid vector and also by mechanical means. Even though sweetpotato P.I. 105945 has been used in numerous experiments, it has not yet exhibited the necrotic, internal cork root symptom, perhaps because of its immunity from expression of the necrotic root spotting phase of this syndrome.

During a sweetpotato virus disease sur-

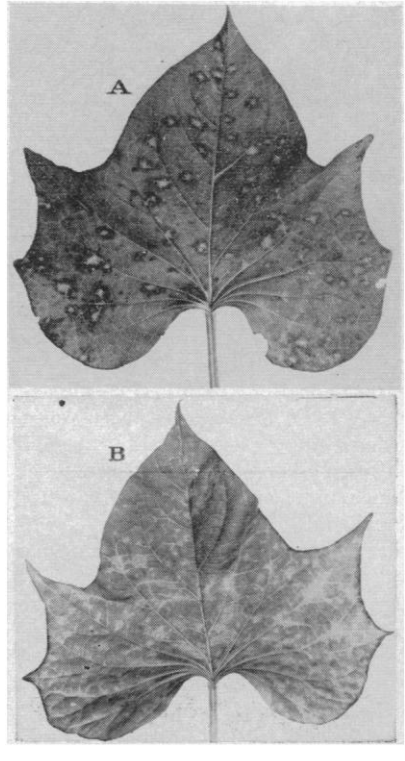

Fig. 1. Current-season symptoms on Porto Rico sweetpotato leaves in two syndromes: (*A*) internal cork; (*B*) feathery mottle.

vey in 1955–57, made in about 20 states, the syndromes here described were encountered side by side in the field in three states: in Georgia in 1955 and in California and New Jersey (*7*) in 1957. In Georgia the Porto Rico variety and several seedlings were observed to be infected with feathery mottle. In California, feathery mottle was found on Red Velvet, a pigmented selection from Porto Rico. In New Jersey the affected variety was Georgia Red. In all cases in other states the "yellow dwarf" symptom description of common use in California was found to be generally applicable.

Incidentally, two similar sweetpotato disease syndromes caused by viruses are known in other countries. The Russian sweetpotato P.I. 105945 exhibits both syndromes. Sweetpotato root material from Israel exhibited both the cork and the feathery mottle syndromes and, when indexed, was apparently identical with the United States material.

Sheffield (*8*) reported that at least two viruses, designated A and B, attack sweetpotatoes in East Africa. Virus A, found only in one locality, caused a relatively mild disease and was transmitted by *Myzus persicae*. Virus B was widespread throughout East Africa and was transmitted by a white fly (*Bemisia tabaci* Genn.). There are a number of strains of virus B, the mildest of which may cause no symptoms in some sweetpotato varieties, whereas the others cause extremely severe diseases. Infection with virus A did not protect a plant from infection by virus B, but infection with a mild strain of virus B protected against infection by a severe strain of virus B. Neither virus was transmitted mechanically to healthy sweetpotatoes. Sheffield conjectured that virus A might be the same as one of the sweetpotato viruses in the United States but considered virus B to be distinguished from them by its insect vector, the white fly. However, we do have an as yet unidentified insect vector for the feathery mottle American counterpart, to judge by the spread of this disease in nature.

In an earlier report Sheffield (*9*) noted that in some cases there was no obvious stunting by virus B, "the symptoms being confined to mottling, or one runner only may be affected or one branch only of one runner. It is probable that the stunted forms are primary infections (i.e., they arose from infected vines), the milder forms being secondary (or new) infections."

The appearance of similar current-season symptoms at Beltsville, Maryland, in 1957 in the syndrome caused by our feathery mottle virus confirms her finding on virus B. It would appear that differences in the climate or environment of the continents and the effect of these differences on the agents of dissemination might account for the apparent discrepancies in the disease syndromes caused by similar viruses, but the possibility of specific differences exists. The latter view is supported in the recent report by Sheffield (*3*), who describes the transmission of sweetpotato virus to other hosts (several species of *Ipomoea* and of the family Solanaceae) by mechanical inoculation, by graft, and by means of white flies.

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26 February 1958

Thorium Content of Ocean Water

Two samples of ocean water from the Pacific, one collected at the surface, near the coast, outside San Diego Bay in the summer of 1956, and the other taken at a depth of 3500 m from longitude 124° 41.0′ W, latitude 33° 54.5′ N on 25 March 1957, have been analyzed for total thorium alpha activity.

Each sample was approximately 25 liters in volume. Three hundred milliliters of concentrated HCl, 25 ml of 0.18*M* $Fe(NO_3)_3$ solution, and approximately 5000 count/min of Th^{234} beta tracer were added to each sample at the time of collection. The samples (in polyethylene containers) were stored for several weeks before they were analyzed.

The method of analysis was as follows. To carry the thorium, ferric hydroxide was precipitated by adding slight excess of ammonium hydroxide to the sample solution. The hydroxide precipitate was dissolved in dilute hydrochloric acid. Nitric acid and perchloric acid were added to the solution, which was then evaporated to fumes of perchloric acid. Addition of hydrofluoric acid and repeated fuming served to remove silica. The final solution was diluted to decrease the concentration of perchloric acid to 3*N* and was then transferred to a 4- by 100-mm Dowex-50 resin column to separate the thorium from carrier and extraneous ions (*1*). The thorium on the column was washed repeatedly with 3*N* hydrochloric acid. It was then eluted from the column with 0.5*M* oxalic acid. This eluant was treated with nitric acid and perchloric acid, heated to fumes of perchloric acid three times, diluted, and again transferred to the resin column. After washing with 3*N* hydrochloric acid, the carrier free thorium was eluted with 0.5*M* oxalic acid, and the eluant was collected on a tantalum plate. The plate was taken to dryness under a heat lamp, flamed, and counted for alpha and beta activity. The beta count served to determine the fraction of added Th^{234} which was recovered. From the observed alpha activity the total thorium alpha activity in the sample was calculated assuming complete exchange of thorium with the Th^{234} tracer.

In each of the samples analyzed, additional thorium was obtained from the first eluant and hydrochloric acid washings by taking these through further perchloric acid fumings and resin-column treatments. These second fractions of thorium which were separated from the surface water and deep ocean samples are designated below as S-2 and D-2, respectively.

Samples were counted on an alpha proportional counter at 51-percent geometry. The observed activities, corrected for yield, geometry, background, and reagent blank, are listed in Table 1.

The half-life values of Th^{232}, Th^{230}, Th^{228}, and Th^{227} (the only thorium alpha emitters which occur naturally) are 1.39×10^{10} years, 80,000 years, 1.90 years, and 18.6 days, respectively. Using these values, we calculate the following upper limits of concentration of thorium isotopes in the deep ocean sample (*2*): Th^{232}, less than 5×10^{-11} g/ml; Th^{230}, less than 3×10^{-16} g/ml; Th^{228}, less than 7×10^{-21} g/ml; Th^{227}, less than 2×10^{-22} g/ml.

Some information concerning the approximate isotopic composition of the thorium which was isolated from our surface water sample was obtained by observing the change in its alpha activity with time. The data for sample S-2 are shown in Table 2. The growth of alpha activity during the first 20 days

Table 1. Thorium alpha activity in Pacific Ocean water.

Sample	Th^{234} tracer recovered (%)	Th alpha activity (disintegration/hr liter)
	Surface	
Fraction S-1	10	9 (±2)
Fraction S-2	40	9.6 (±0.5)
	Deep ocean	
Fraction D-1	28	0.3 ±0.3
Fraction D-2	45	0.4 ±0.4