

(thus differing from the chlorosis caused by potassium deficiency in which the affected area acquires a dull yellow color with a bronze or copper overcast). The blanched tissues usually do not die as quickly as in potassium deficiency so that local specking or spotting of the leaf is commonly wanting except in advanced stages of the malady. In some instances, however, local dying of the tissues between the veins has been the first symptom. In plot tests conducted in several tobacco-growing districts, in which chemically pure sources of nitrogen, phosphorus, potassium, calcium and sulfur were used instead of the ordinary commercial fertilizer materials, the symptoms of magnesium deficiency have usually appeared where sandy and sandy loam soils were used and there was abundant rainfall. In all such cases addition of sulfate or chloride of magnesium to the fertilizer salts has prevented the chlorosis. In comparative tests, applications of the so-called high grade or relatively pure commercial forms of the sulfate and chloride of potassium have resulted in severe chlorosis while low grade sulfates and chlorides of potassium containing considerable quantities of magnesium, such as "double manure salt" and "kainit," have prevented the disease. In some cases use of the purer forms of sulfate of potassium has resulted in severer chlorosis than that caused by the chloride and, moreover, the severity of the chlorosis has been proportional to the quantity of sulfate of potassium used. Symptoms of the disease also have been prevented by applying dolomitic limestone to the soil while comparatively pure calcite has been ineffective. Certain organic fertilizer materials of vegetable origin which are commonly used as sources of nitrogen, notably cotton seed meal, tobacco stalks and stems and barn manure, tend to prevent the disease. These materials contain appreciable quantities of magnesium. Use of other common sources of fertilizer nitrogen which contain little or no magnesium, including nitrate of soda, dried blood and especially ammonium sulfate, has favored development of the disease. In pot cultures this chlorosis is readily induced by applying a nutrient solution containing all the usual plant food elements except magnesium, using an ex-

cess of the solution so as to produce a leaching action on the soil. Moreover, the disease if not too far advanced is readily cured by adding magnesium to the nutrient solution. In view of the relation of the sulfur supply to the symptoms of magnesium deficiency it is worthy of note that an inadequate supply of sulfur *per se* results in a mild, diffuse type of chlorosis of tobacco, affecting all green parts of the plant and thus differing from the symptoms of magnesium deficiency. Since more or less sulfur is constantly added to the soil through rainfall, while there is loss of magnesium through the leaching action of the rain water, it is to be expected that symptoms of magnesium deficiency will be especially pronounced in wet seasons. In dry seasons the likelihood of relative deficiency of sulfur is increased. Experiments with corn, a crop plant differing widely from tobacco in many respects, show that it also is subject to the "sand drown" disease, the symptoms and characteristics of the disease in corn corresponding rather closely with those found in tobacco. It seems likely, therefore, that other crop plants are subject to injury from an inadequate supply of magnesium in the lighter, more sandy type of soils. Apparently the quantity of magnesium required in the fertilizer to prevent the symptoms of magnesium deficiency is small, probably less than 50 pounds per acre. These investigations suggest that the element magnesium needs to be taken into account both in the general problem of liming and in the proper choice of commercial fertilizer materials for making up so-called complete fertilizers.

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#### TRANSFERENCE OF THE BEAN MOSAIC VIRUS BY MACROSIPHUM SOLANIFOLII

It has been assumed by pathologists that the virus of bean mosaic is transferred from diseased to healthy plants by insects. No experimental proof has been submitted to substantiate this claim. The only satisfactory ex-

planation of the widespread occurrence of the disease in certain seasons seems to be that occasional plants which show mosaic early in the season serve as the sources of infectious material which is carried throughout the field by virus-carrying insects. The probability of this being the correct explanation is strengthened by the known facts concerning the spread by insects of mosaic diseases of other plants.

Indirect proof of the relation of insects to the dissemination of bean mosaic was obtained during the summer of 1921. Plantings in late May of seed collected from plants showing mosaic the previous season were made in rows four feet apart. Another plot was planted at the same time seventy-five feet away with seed from plants grown for two previous seasons under insect-proof cages. These plants had shown no symptoms of mosaic. By the middle of July, practically all of the plants in the mosaic seed plot were showing symptoms of the disease. None of the plants in the clean seed plot had developed any signs of mosaic up to the time of the appearance of the second pair of leaves. Weekly inspection was made and the first case of mosaic was found in one plant of the Long White variety on June 12. A count was made each week of the new plants which had developed mosaic and on September 17, when the results were summarized, 19.5 per cent. of the total number of plants in the plot, which included twenty varieties, were affected with the disease. On July 15, clean seed from the same source as the above was planted in hills between the rows of diseased plants in the mosaic plot, half of the seed under insect-proof cages. By September 1, every plant outside of the cages was affected with mosaic, while not a single diseased plant appeared in the cages up to the time the plants were killed by frost. The diseased and healthy plants at all times during their growth were sufficiently far apart so that they did not come in contact with one another. This experiment indicated that insects are directly responsible for the dissemination of the disease.

In May, 1922, a planting of beans in the greenhouse was found to be infested with aphids. Several of the plants had early shown symptoms of mosaic. Growing some

distance from the aphid-infested plants were a group of bean plants in water cultures. Practically every plant in these cultures developed a severe case of mosaic, and examination showed that the aphids were also abundant on them. Golden Wax and Green Pod Stringless beans were planted in pots in the greenhouse and the pots placed in cages. Brittle Wax beans were planted in the field and three cages placed over a portion of the row. In addition, a twenty-foot row of the same variety was planted and left uncaged for observation as to freedom of the seed from mosaic. As soon as the greenhouse and field plants had developed the first pair of leaves, aphids from the mosaic plants were transferred by means of a camel hair brush to watch glasses, and then placed on the leaves of three plants in one of the field cages, and on five plants of Golden Wax and three of Green Pod Stringless in cages in the greenhouse. Two cages of plants in the field as well as the additional twenty foot row were held for field checks. A large number of plants in pots were held in separate cages in the greenhouse as checks. The plants were inspected daily. After five days, three plants of Golden Wax and one of Green Pod Stringless showed apparent symptoms of mosaic as water-soaked areas along the veins which gradually involved the entire leaf. In addition there was a distinct down-curling of the leaves, usually characteristic of mosaic. This was contrary to usual observations which have been that when mosaic is transferred to healthy plants by aphids, symptoms of the disease do not appear on the inoculated plant until the new leaves have developed. However, with both of the varieties used the mottling was very distinct on the original leaves. Contrary observations on other plants have probably been due to the fact that it is difficult to detect mosaic symptoms on old leaves. After twelve days new leaves had appeared and these showed marked mosaic symptoms. These plants were held for eighteen days and the succeeding leaves continued to present characteristic mosaic markings. None of the plants in the check cages had developed any signs of mosaic one month after the beginning of the experiment, when they were discarded.

After seven days one plant of Brittle Wax in the field cage reacted similarly to the plants in the greenhouse. When the second pair of leaves appeared, mosaic markings were not apparent and it was thought that weather conditions had checked the progress of the disease in the plant. However, upon examining the plants one week later, two plants of the three upon which the insects had been placed had developed typical mosaic symptoms. All of the plants in the check cages, as well as in the twenty-foot row outside of the cages, have remained healthy up to this time, one month from the time of the appearance of the first pair of leaves.

The species of aphid used in these experiments has been identified by Miss Eugenia McDaniel of the entomology department as *Macrosiphum solanifolii*. This species has been collected on beans at other times, especially early in the season. *Phaseolus vulgaris* is one of the known hosts of this very polyphagous species.

#### SUMMARY

The spread of bean mosaic was observed during 1921 under conditions which strongly suggested transfer by insects. The sudden appearance of the disease in the water cultures of beans growing in the greenhouse, and infested with aphids, indicated even more definitely the mode of dissemination. Definite proof of the transfer of the virus by *Macrosiphum solanifolii* was obtained under controlled conditions, both in the greenhouse and in the field.

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#### THE EXTENSION OF THE X-RAY INTO THE ULTRAVIOLET SPECTRUM

It was found that when thermions liberated from a tungsten filament were accelerated and allowed to impinge on a metal grid maintained at a variable positive potential, secondary electrons were emitted from the grid. The number of such secondary electrons emitted were measured by means of a galvanometer in series with the grid and a plate maintained at a constant positive saturation potential.

On plotting the secondary current as a func-

tion of the accelerating voltage, acting on the primary electrons, a sudden change in the slope of the curve occurring at critical potentials was interpreted in the usual way. The energy-quantum relation  $V(\text{volts}) L (\text{\AA}) = 12320$  was used to compute the equivalent wavelengths.

The following table gives the preliminary results thus far discovered. The quantities bracketed are still doubtful. Those preceded by an (a) are not found by the usual breaks in the curves but are positions on the continuous curves where the ratio of the number of secondary (s) electrons per primary (p) were such as indicated in the s/p column. At present it appears as if the convergence wavelength ( $s/p = 3$ ) for tungsten ended at 91.2  $\text{\AA}$  and is followed by an absorption band extending probably down to 14  $\text{\AA}$ . This is then followed by the  $M\alpha$  line, here extrapolated as 7.04  $\text{\AA}$  from the above measurements.

#### TUNGSTEN

Volts	Wave-length ( $\text{\AA}$ )	S/P	Remarks
4.4	2800		Hull found 2700 shortest spark spectrum.
(17)	(725)		Suspected
35.0	352		
(60)	(205)		
135	91.2	3	Doubtful
144	a85.6	2.5	
181	a68.0	2.0	
295	a41.7	1.5	
435	a28.3	1.0	
( )	( )	0.0	
1750	7.04		Extrapolated X-ray data gives $M\alpha = 7.007$

#### IRON

Volts	Wave-length	Remarks
3.3	3763	
8.5	1450	Millikan's iron spectrum shows 1430 and 1409. Also 1184.
10.4	1184.6	
24.3	507.0	Also 506 and 552.1. Intensity 7. $M\alpha$ computed from Sanford's formula gave 484 $\text{\AA}$ . Iron shows spectrum 271.6 $\text{\AA}$ .
45.8	269	Doubtful.
( )	a( )	
200	a61.6	

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