# Virus Diseases of Broad Beans.

# $B_{\mathbf{v}}$

## Patricia I. Klesser.

Broad beans are not grown extensively in South Africa. The viruses described here were found on naturally infected *Vicia faba* plants grown in isolated areas of the Transvaal, and they are possibly not characteristic for the whole country.

In the field, the symptoms are: mosaic, mosaic with stunt, or severe necrosis and collapse.

The viruses causing mosaic symptoms are found frequently. An unusual feature of this mosaic is the fact that the chlorotic areas are depressed below the normal surface of the leaf.

The systemic necrosis is caused by the tomato spotted wilt virus, which is described in detail in the paper on pea virus diseases. The incidence of this disease can be fairly high, with severe crop losses; but usually it occurs only sporadically thoughout a planting.

# REVIEW OF LITERATURE.

Although Elliot transmitted a mosaic from clover to broad bean in 1921, the first descriptions of naturally occurring mosaic diseases of broad bean were those of Böning 1927, van der Meulen 1928, Merkel 1929 and Fukushi 1930.

Imai 1934 successfully transmitted a virus causing mosaic on broad bean, to broad bean, pea and sweet pea, by both aphids and sap.

Further reports of broad bean mosaics came from Gigante in Italy (9); Yu in China (25); d'Oliviera in Portugal (5); and Quantz in Germany (17).

Murayama (14) determined the physico-chemical properties of a mosaic of broad beans in Japan.

Pierce (16) described the broad bean local lesion virus which he isolated from red clover, and which Weiss (22) considers to be the same as the pea wilt virus of Johnson (12).

Weiss regards the mosaic virus of Böning as being identical with the common pea mosaic virus. Further, he groups the viruses of Merkel, Murayama and Yu together, and suggests a relationship with either the bean yellow mosaic or the pea mottle viruses.

Other types of viroses found naturally on broad bean are the mild mosaic and rosette of Yu (25, 26); a vascular wilt—Stubbs (20); a mottle—Bawden, Chaudhuri & Kassanis (1); a soil-borne necrotic mosaic—Fujikawa (7) and tomato spotted wilt, Yu (27).

Rubio & van Slogteren studied the X-bodies associated with the mottle virus (18).

When artificially inoculated, broad beans develop systemic mosaic or necrotic symptoms with most of the legume viruses (10, 15, 16, 28, 29, 30, 31, 32) and also with the tobacco ringspot virus (24) and the curly top virus (19).

With celery virus 1, only local lesions develop (23).

# METHODS AND MATERIALS.

Naturally infected broad bean plants were collected from several parts of the Transvaal. The viruses were inoculated to a number of species of *Leguminosae*, and also to several Solanaceous plants. (None of the latter was susceptible).

Carborundum power was used for the sap inoculations and *Aphis fabae* and *A. craccivora* for the insect transmission tests.

#### 1. Broad Bean Mosaic Virus A.

Physical properties: Thermal inactivation point, 55–56°C. Longevity in vitro, 2–3 days. Dilution end point, 1: 1,000.

Transmission: Mechanical sap inoculation. Aphis fabae.

Host range: Arachis hypogaea L., Crotalaria juncea L., C. spectabilis Roth., Glycine max (L.) Merr., Lathyrus odoratus L., Lupinus albus L., L. luteus L., L. mutabilis Sweet., Medicago lupulina L., Melilotus officinalis Willd., Phaseolus acutifolius Gray var. latifolius Freem., P. lunatus L., P. mungo L., P. vulgaris L., Pisum sativum L., Trifolium hybridum L., T. incarnatum L., T. pratense L., Vicia faba L., Vigna sesquipedalis (L.) Wight., V. unguiculata (L.) Walp., Voandzeia subterranea Thouars.

### REACTION OF SUSCEPTIBLE SPECIES.

Arachis hypogaea.

Local. No reaction.

Systemic. There is a diffuse chlorotic mottle.

Crotalaria juncea.

Local. There are necrotic spots after 8 days. These fuse and the leaves drop.

Systemic. In 12 days necrosis has spread up the stem and into the veins of the young leaves (Fig. 1b). The plant may collapse or may have malformed rolled leaves, with dark green blisters.

C. spectabilis.

Local. No reaction.

Systemic. After 3 weeks the young leaves develop irregular chlorotic areas, which cause a malformation of the leaves, due to uneven growth of the laminae. Soon necrosis sets in in these areas. Later formed leaves have chlorotic line patterns which become necrotic to produce an "etched" effect. Older leaves may show necrotic rings without any chlorosis.

Glycine max.

Local. No reaction.

Systemic. A few leaves show a chlorotic mottle, the young leaves being symptomless. Some old leaves have vivid yellow spots with central necrotic specks.

Lathyrus odoratus.

Local. No reaction.

Systemic. Most leaves are rolled and have dark green streaks.

Lupinus albus.

Local. Chlorotic spots with dark green rings can be seen after 11 days. The leaflets drop.

Systemic. The young leaves show a vein clearing, and they remain folded and have wavy margins. Later leaves develop small chlorotic spots and then dark green blisters. The latter leaves are crinkled and may be malformed. The growing point is rosetted and stunted.

#### L. luteus.

Local. No reaction.

Systemic. In 8 to 9 days small chlorotic spots develop on the young leaves. After 3 weeks most leaves are small, malformed and mottled. The plant is stunted. There may be enough necrosis to cause the collapse of the plant.

#### L. mutabilis.

Local. No reaction.

Systemic. The young leaves have chlorotic specks. Later ones are chlorotic with dark green blisters and malformation. The plant is rosetted.

Medicago lupulina.

Local. No reaction.

Systemic. The young leaves develop chlorotic streaks and later leaves are mottled and slightly puckered.

Melilotus officinalis.

Local. No reaction.

Systemic. The plant is stunted and the leaves have chlorotic vein streaks.

Phaseolus acutifolius.

Local. In 4 days there are chlorotic spots and the veins become necrotic.

Systemic. After 12 days the young leaves develop a vein clearing with small chlorotic spots. There is necrosis in the chlorosis (Fig. 1e) and the growing point may collapse. Later formed leaves are mottled.

#### P. lunatus.

Local. In 7 to 8 days necrotic lesions develop and there are large chlorotic areas.

Systemic. The necrosis spreads quickly into the stem and veins of the young leaves, and the plant collapses.

P. mungo.

Local. No reaction.

Systemic. A diffuse spot mottling develops.

P. vulgaris var. Canadian Wonder.

Local. There are irregular chlorotic areas which fuse to a general chlorosis.

Systemic. In 6 days the young leaves show a vein clearing. Later leaves develop a diffuse chlorotic spotting and the older leaves have large chlorotic blotches.

var. Haricot.

Local. Within a week there are chlorotic blotches.

Systemic. At the same time the young leaves curl down severely. They are crinkled and chlorotic.

Pisum sativum.

Local. In 8 days the leaves show small necrotic specks.

Systemic. Soon after, the young leaves develop a vein clearing with small necrotic specks. Later leaves and stipules are all similarly speckled and the stem develops purple necrotic streaks which may result in the collapse of the plant. If not, the leaves are rosetted and folded with irregular dark green areas and the tendrils are abnormally curled.

Trifolium hybridum.

Local. No reaction.

Systemic. Most leaves show a chlorotic streak mottle and slight crinkle after about 3 weeks.

T. incarnatum.

Local. No reaction.

Systemic. After 9 days the young leaves show a vein clearing. Later leaves have irregular chlorotic areas and are crinkled. Some may have necrotic spots or veins (Fig. 1c).

T. pratense.

Local. No reaction.

Systemic. A chlorotic streak mottle develops (Fig. 1d).

Vicia faba.

Local. No reaction.

Systemic. In 9 days the young leaves show a vein clearing. Later formed leaves develop chlorotic spots or dark green streaks and then a mosaic. A characteristic of this mosaic is that the chlorotic areas are sunken below the leaf surface, resulting in the dark green streaks being apparently raised. (Fig. 1a). The leaves are also rolled and the plant is slightly stunted.

Vigna sesquipedalis.

Local. Isolated chlorotic areas develop.

Systemic. Only the first and second trifoliates show a diffuse chlorotic mottle.

# V. unguiculata.

Local. There are chlorotic spots in 4 days, and the leaves become flaccid and drop.

Systemic. After 9 to 10 days the young leaves develop a vein clearing. Later ones are diffusely mottled.

#### Voandzeia subterranea.

Local. No reaction.

Systemic. After 3 weeks the leaves show a chlorotic network and irregular chlorotic areas.

Natural source of virus: *Vicia faba*. The plants were slightly stunted and the leaves were rolled and elongated. They had a mosaic mottle with the chlorotic areas depressed below the dark green or normal coloured areas.

#### IDENTIFICATION.

The characteristic symptom of this virus on broad bean, is a mosaic with the chlorotic areas depressed. This apparently occurs with only one other described virus.

Böning (3) mentions a similar effect with his virus, which was later identified as the common pea mosaic virus. However, the pea mosaic is not transmissible to bean (which is a host of this virus) and causes a mosaic on pea and not a necrosis which is usual with this virus.

Although pea virus 2 Osborn (15) is able to infect bean, the symptoms on pea are again chlorotic and not necrotic.

This necrotic reaction on pea can be compared with several others:

- (a) The alfalfa mosaic virus 1B Zaumeyer (29) causes a similar local and systemic necrosis on the pea, but only a local necrotic spotting on the bean, whereas this virus induces a mottle only. Moreover this alfalfa mosaic results in a necrotic collapse of the broad bean.
- (b) The pea American streak virus Zaumeyer (28) is non-infectious to the bean, causes a local reaction on broad bean and withstands a temperature of 10° higher than this virus.
- (c) The pea Wisconsin streak virus Hagedorn & Walker (10) is not able to infect either bean or broad bean.
- (d) The pea New Zealand streak virus Chamberlain (4) causes a local and systemic necrosis of the bean (whereas this virus induces a mottle only), and broad bean is not included in the host range. Further, there is a great difference in the physical property values of the 2 viruses.
- (e) The alsike clover mosaic virus Zaumeyer & Wade (32) results in a conspicuous reflexing of the bean leaves, which does not occur with this virus.
- (f) The white clover mosaic virus complex Zaumeyer & Wade (31) induces a necrosis on both the bean and broad bean, both of which show a mottle with this virus.
- (g) Although the symptoms of the pea stunt virus Zaumeyer (30) on pea and bean are similar to this virus, symptoms on other hosts, the host range and the physical property values are different.

Pierce's broad bean local lesion virus (16) causes only a local necrosis on broad bean, while Stubbs' virus (20) induces a severe systemic necrosis.

The mild mosaic virus of Yu (25) is unable to infect bean, cowpea and soybean (all of which are hosts of this virus) and does not withstand more than 3 hours ageing, whereas this virus lasts 2-3 days *in vitro*.

The broad bean mottle virus Bawden, Chaudhuri & Kassanis (1) induces symptoms similar to this virus on bean, pea, sweet pea and the clovers, but differs on soybean and broad bean. (During the initial subinoculations with the mottle virus, broad bean developed a systemic necrosis as well, but later, only a mottle). However, Bawden's virus withstood heating up to 95°C.. and ageing up to 20 days. Further it was not transmissible by any of the *Aphis* species.

As this virus cannot be fully identified with any previously described, it may be a new virus or strain. It is named broad bean mosaic virus A.

#### 2. Broad Bean Mosaic Virus B.

Physical properties: Thermal inactivation point, 56-58°C. Longevity in vitro, 1-2 days. Dilution end point, 1: 1,000.

Transmission: Mechanical sap inoculation. Aphis craccivora.

Host range: Crotalaria juncea L., C. spectabilis Roth., Lupinus albus L. vars. sweet and bitter, Phaseolus lunatus L., P. vulgaris L., Pisum sativum L., Trifolium hybridum L., T. incarnatum L., Vicia faba L., Vigna sesquipedalis (L.) Wight., V. unguiculata (L.) Walp., Voandzeia subterranea Thouars.

#### REACTION OF SUSCEPTIBLE SPECIES.

Crotalaria juncea.

Local. No reaction.

Systemic. In 8 days the young leaves develop a vein clearing and spotting. The next leaves have chlorotic streaks which lead to a malformation. Later leaves are small and rolled and have dark green blisters. (Fig. 2e). The plant is stunted.

C. spectabilis.

Local. No reaction.

Systemic. In 13 days there are chlorotic spots and vein flecks on the young leaves. Later leaves have broad alternate chlorotic and green veinbands (Fig. 2c). There may be necrotic stem streaks.

Lupinus albus var. Sweet.

Local. After 13 days the leaves have chlorotic spots with necrotic veins, and they soon drop.

Systemic. At the same time the young leaves remain folded and have small chlorotic spots. Later formed leaves are small, malformed and mottled. The growing point is rosetted and the plant stunted.

var. Bitter.

Local. No reaction.

Systemic. The first leaves are mottled. Later the growing point becomes rosetted with small chlorotic leaves. There may be necrotic stem streaks.

Phaseolus lunatus.

Local. No reaction.

Systemic. After 3 weeks chlorotic vein flecks develop (Fig. 2b).

P. vulgaris var. Canadian Wonder.

Local. In 11 to 12 days large chlorotic blotches appear.

Systemic. The first few trifoliates develop large chlorotic areas (Fig. 2d). New leaves are symptomless.

var. Haricot.

Local. In 10 to 12 days there are small chlorotic areas.

Systemic. Soon after, the young leaves curl down and have a chlorotic network. Later formed leaves are small, puckered and mottled.

Pisum sativum.

Local. No reaction.

Systemic. After 11 to 12 days the young leaves show a vein clearing and spotting. The next formed leaves develop a mosaic, and the tendrils are abnormally curled. Necrosis of the growing point leads to the eventual collapse of the plant.

Trifolium hybridum.

A symptomless carrier.

T. incarnatum.

Local. No reaction.

Systemic. The young leaves show a chlorotic network and flecking in 11 days. Later leaves are crinkled and have a mosaic mottle. (Fig. 2a). The plant is severely stunted.

Vicia faba.

Local. No reaction.

Systemic. In 18 days chlorotic spots develop on the young leaves. Later leaves show a mosaic with the chlorotic areas depressed, leaving raised dark green islands.

Vigna sesquipedalis.

Local. No reaction.

Systemic. There is a chlorotic network followed by a chlorotic spotting.

V. unguiculata.

Local. No reaction.

Systemic. The young leaves show small chlorotic specks after 18 days. Later ones are chlorotic with short dark green veinbands, and are rolled downwards.

Voandzeia subterranea.

A symptomless carrier.

Natural source of virus: Vicia faba. Although the mosaic symptoms on the leaves were similar to those on the plants from which the A virus was isolated, there was little stunt or rolling. The general appearance of the plants was normal.

Crotalaria spectabilis. Most leaves had bands of vivid yellow and dark green. There was also a slight veinal necrosis.

#### IDENTIFICATION.

This virus is very similar to the previous one, but differences in host range, physical property values and symptoms on some plants are too marked for it to be classified as the same.

As this virus also results in the typical sunken chlorotic reaction on the broad bean, it is named broad bean mosaic virus B.

## SYMPTOM DIFFERENCES BETWEEN BROAD BEAN MOSAIC VIRUSES A AND B.

Host plant.	Virus A.	Virus B.	
Crotalaria juncea	Loc. necr. spots. Syst. necr. coll.	Loc. no reaction. Syst. chl. str., d.gr. bl., malf.	
C. spectabilis	Loc. no. Syst. irreg. chl., malf., necr. patterns.	Loc. no. Syst. chl. spots, d.gr. veinb. necr. s.s.	
Phaseolus lunatus	Loc. necr. lesions. Syst. necr. coll.	Loc. no. Syst. chl. flecks.	
Pisum sativum	Loc. necr. specks. Syst. necr. specks, coll.	Loc. no. Syst. mos., necr. coll.	
Trifolium hybridum	Loc. no. Syst. chl. streak mot.	Symptomless carrier.	
Vigna sesquipedalis	Loc. chl. spots. Syst. chl. mot.	Loc. no. Syst. chl. spots or veinb.	
Voandzeia subterranea	Loc. no. Syst. chl. veins and areas.	Symptomless carrier.	
Abbreviations used:—	-		
bl.—blisters.	malf.—malformed.	n.s.s.—necrotic stem streak.	

bl.—blisters.	
---------------	--

chl.—chlorosis/chlorotic.

mos.—mosaic.

str.—streaks.

veinb.—veinbands.

coll.-collapse.

mot.--mottle.

d. gr.—dark green.

no.-no reaction.

irreg.-irregular.

necr.--necrosis/necrotic.

# PHYSICAL PROPERTIES AND METHODS OF TRANSMISSION.

Virus.	Thermal inactiva- tion	nactiva- Longevity	- Longevity Dilution	Dilution	Transmission.		
			point.	Sap.	Seed.	Aphis.	
A	55–56 56–58	2–3 1–2	1: 1,000 1: 1,000	‡	_	++	

# HOST RANGES.

Host plant.	Virus A.	Virus B
Arachis hypogaea	-	
Crotalaria juncea	-	
C. spectabilis	-	-
Polichos lablab		_
Glycine javanica		
G. max	-	- C?
athyrus odoratus		<u> </u>
upinus albus	+	-4-
. luteus		_
L. mutabilis		_
Medicago lupulina		
1. sativa	_	
Melilotus officinalis		
Phaseolus acutifolius		
P. lunatus	-	-4-
P. mungo		
P. vulgaris		-41
Pisum sativum	-	-+-
Trifolium fragiferum	_	
Г. hybridum	-	
T. incarnatum		
C. pratense		
repens	-	_
'icia faba		
'igna sesquidedalis		
igna sesquataans	-	
Voandzeia subterranea		

# LITERATURE CITED.

1.	BAWDEN, F. C., CHAUDHURI, R. P. AND KASSANIS, B. (1951)	Some properties of the broad-bean mottle virus. Ann. Appl. Biol., 38: 774–784.
2.	Berkeley, G. H. (1947)	Alfalfa mosaic on pepper in Ontario. Phytopathology (Abstr.), 37: 3.
3.	Böning, K. (1927)	Die mosaikkrankheit der Ackerbohne ( <i>Vicia faba</i> L.) Ein Beitrag zu dem Mosaik der Papilionaceae. Forsch. auf dem Gebiet der Pflanzenkrankh. und der Immunität im Pflanzenreich, 4: 43–111.
4.	CHAMBERLAIN, E. E. (1939)	Pea Streak ( <i>Pisum</i> virus 3). N.Z. Jour. Sci. & Tech., 20: 365A-381A.
5.	d'Oliviera, Maria de L. (1946)	O "mosaico" de Faviera e suas relações com outras Leguminosas. Brotéria, 15: 90-94. (Abstr. R.A.M., 26: 372).
6.	Elliott, J. A. (1921)	A mosaic of sweet and red clovers. Phytopathology, 11: 146-148.
7.	<b>Г</b> ИЛКАWA, <b>Т</b> . (1955)	Studies on the necrotic mosaic of broad bean. Agric. & Hort., 30: 721-722. (Abstr. R.A.M., 35: 742.)
8.	Fukushi, T. (1930)	On the mosaic disease of broad bean. Jour. Pl. Prot., 18: 707–712, 779–784. (Abstr. R.A.M., 10: 700.)
9.	GIGANTE, R. (1937)	Il mosaico della Fava ( <i>Vicia faba</i> 'L.) in Italia e comportamento di alcune Leguminose di fronte ad esso. Boll. Staz. Pat. veg. Roma., 17: 497–529. (Abstr. R.A.M. 17: 645.)
10.	HAGEDORN, D. J. & WALKER, J. C. (1949).	Wisconsin pea streak. Phytopathology, 39: 837-847.
11.	IMAI, Y. (1934)	Studies on the transmission of broad bean mosaic. Trans. Sapporo Nat. Hist. Soc., 13: 241-245. (Abstr. R.A.M., 14: 4)

12.	Johnson, F. (1942)	The complex nature of white clover mosaic. Phytopathology, 32: 103–116.
13.	Merkel, L. (1929)	Beiträge zur Kenntnis der Mosaikkrankheit der Familie der Papilionacee. Zeitschr. Pflanzenkr. u. Pflanzensch., 39: 289-347.
14.	Микауама, <b>D</b> . (1938)	Physico-chemical properties of the virus of broad bean mosaic. Jour. Fac. Agric. Hokkaido Univ., 44: 1-32 (Abstr. R.A.M., 18: 224).
15.	OSBORN, H. T. (1937)	Studies on the transmission of pea virus 2 by aphids. Phytopathology, 27: 589-603.
16.	PIERCE, W. H. (1935)	The identification of certain viruses affecting leguminous plants. Jour. Agric. Res., 51: 1,017–1,039.
17.	QUANTZ, L. (1953)	Untersuchungen über ein samenübertragbares Mosaikvirus der Ackerbohne ( <i>Vicia faba</i> ) Phytopath. Zeitschr., 20: 420-448.
18.	RUBIO, M. AND VAN SLOGTEREN, D. H. M. (1956)	Light and electron microscopy of X- bodies associated with broad bean mottle virus and <i>Phaseolus</i> virus 2. Phytopathology, 46: 401-402.
19.	Severin, H. H. P. and Henderson, C. F. (1928)	Some host plants of curly top. Hilgardia, 3: 339-393, 595-636.
20.	Stubbs, L. L. (1947)	A destructive vascular wilt virus disease of broad bean ( <i>Vicia faba</i> L.) in Victoria. Jour. Dept. Agric. Vict. Austr., 45: 323–332.
21.	van der Meulen, J. G. J. (1928)	Voorlopige onderzoek naar de specialisatie en die infectie- bronnen der mozaiekziekten van landbougewassen. Tijd- schr. Plantenz., 34: 151–176.
22.	Weiss, F. (1945)	Viruses described primarily on leguminous vegetable and forage crops. Pl. Dis. Reptr. Suppl. 154.
23.	WELLMAN, F. L. (1934)	Identification of celery virus 1. The cause of southern celery mosaic. Phytopathology, 24: 695–725.
24.	WINGARD, S. A. (1928)	Hosts and symptoms of ringspot: a virus disease of plants. Jour. Agric. Res., 37: 127–153.
25.	Yu, T. F. (1939)	Mild-mosaic virus of broad bean. Phytopathology, 29: 448–455.
26.	(1939)	A suspected virosis of the severe leaf-curl or stunt type occurs in China near Nanking. Phytopathology, 29: 459-461.
27.	(1947)	Spotted wilt of broad bean. Phytopathology (Note), 37: 191–192.
28.	ZAUMEYER, W. J. (1937)	Pea streak and its relationship to strains of alfalfa mosaic. Phytopathology, 27: 144.
29.	(1938)	A streak disease of peas and its relation to several strains of alfalfa mosaic virus. Jour. Agric. Res., 56: 747-772.
30.	(1940)	Three previously undescribed mosaic diseases of pea. Jour. Agric. Res., 60: 433-452.
31.	ZAUMEYER, W. J. AND WADE, B. L. (1935)	The relationship of certain legume mosaics to bean. Jour. Agric. Res., 51: 715–749.
32.	ZAUMEYER, W. J. AND WADE, B. L. (1936)	Pea mosaic and its relation to other legume mosaic viruses. Journ. Agric. Res., 53: 161-185.

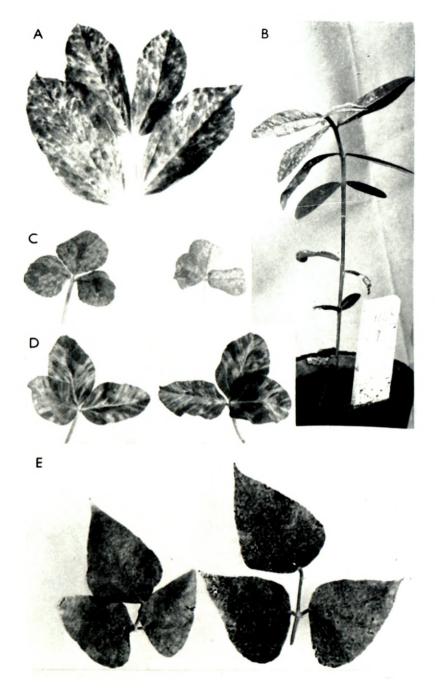


FIG. 1.—BROAD BEAN MOSAIC VIRUS A.

A. Vicia faba—showing depressed chlorotic areas. B. Crotalaria juncea.
C. Trifolium incarnatum. D. Trifolium pratense. E. Phaseolus lunatus.

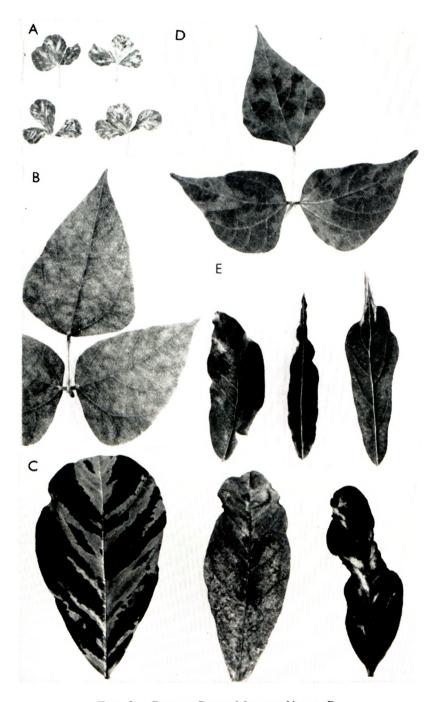


FIG. 2.—BROAD BEAN MOSAIC VIRUS B.
A. Trifolium incarnatum. B. Phaseolus lunatus. C. Crotalaria spectabilis.
D. Phaseolus vulgaris. E. Crotalaria juncea.