Virus Enemies of Gladiolus

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When a virus infects the gladiolus plant the probability is that it will soon become a permanent resident of the plant and of all its vegetative progeny.

Thus, the recent great increase in production of gladiolus, the growing of corms in very many places, and the interstate and international commerce in them offer unusual opportunities for transporting viruses and for increasing them in the crop. Such factors are partly responsible for the increase in number of virus diseases as the growing of gladiolus has expanded. Before 1928 no one had described a virus disease of this crop. By 1952 workers had demonstrated four diseases of this class and had described several other suspected virus diseases of gladiolus.

The first report of a suspected virus disease of gladiolus was by Louise Dosdall at the Minnesota Agricultural Experiment Station in 1928. She described a disease of Gretchen Zang and other gladiolus varieties. It was distinguishable by wartiness and mottling of the corms, mottling of leaves and flower bracts, breaking of the flower color, and bunching of the blossoms. The disease reappeared the following season when affected corms were replanted. Miss Dosdall therefore considered it a degeneration disease, although she presented no proof of transmissibility of a causal virus. Later students of gladiolus diseases have not recognized a virus disease with distinctive wartiness of corms. Colin D. McKeen, of the University of Toronto, in 1943, described ring patterns in gladiolus corms, but found no proof of virus as a causal agent. We have seen corm symptoms resembling McKeen's disease, but the causal agent is still unknown.

A. W. Dimock, of Cornell University, in 1940 described a damaging mosaic disease of gladiolus, characterized by short spikes, fewer florets than normal, and bleaching of the petals of colored varieties. We have termed this disease white break to distinguish it from the more common and milder mosaic caused by bean yellow mosaic virus. In white break the flowers are blotched with white or with yellow rather than streaked. The bleached areas are sometimes recognizable in the buds as they begin to show color. Affected flowers open irregularly and fade early. Bleaching is often so extensive that the flower is no longer recognizable as to variety. The flower bracts are streaked or bleached and may wither while the flowers are still opening. Affected plants are often notably shorter than normal plants. No leaf symptoms are consistently linked with white break.

White break caused some concern among producers of gladiolus when it was first recognized. Affected plants are unsalable, and such a disease could assume major importance if it would spread rapidly. It appears commonly along the eastern seaboard and is known in southern Oregon. Natural spread at Beltsville, Md., has reached 15 percent in one season. There has been little evidence of natural spread in other areas in which glads are produced commercially, so that white break is feared less than formerly. The disease is a nuisance because it is not recognizable in plants that do not bloom and is commonly overlooked in flowers that are cut for shipment when the first floret shows color. It thus persists in field plantings and frequently enters the wholesale markets in cut blooms. No insect vector has been found for the white break virus, al-

though aphids, leafhoppers, and thrips have been experimentally tested as suspects.

The virus of common mild mosaic of gladiolus is transmitted by the green peach aphid and by the crescent-marked lily aphid. The mosaic is expressed as angular light- and dark-green mottling in young leaves and as stripe breaks in the flowers.

Similar mosaic mottling appears in plants of related genera of Iridaceae, such as *Babiana*, *Freesia*, *Ixia*, *Sparaxis*, *Streptanthera*, *Tritonia*, and *Watsonia*, grown from corms from commercial sources. Those plants, like gladiolus, however, are free from leaf mottling and from flower breaks if grown from seed. Extensive cross-inoculations, mostly by means of the green peach aphid, showed that the mosaic diseases occurring in those plants are the same as the mild mosaic of gladiolus or much like it. Flower breaks were observed in affected plants of *Babiana*. The effects of this mosaic on the other hosts are usually mild mottling comparable to the symptoms in gladiolus, but freesias die back when infected with some strains of the virus from gladiolus.

A VIRUS DISEASE caused severe loss in commercial pole beans in 1946 in Oregon, especially when the beans were grown next to gladiolus fields. The gladiolus were shown to be the source of the virus when the virus was experimentally transmitted from gladiolus to beans and reproduced the bean disease in question. Next, abnormalities characteristic of the previously known bean yellow mosaic, or bean virus 2, were found also within the cells of bean infected experimentally from gladiolus. Thus gladiolus were shown to be carriers of this bean virus—a wholly unexpected relationship, for gladiolus is remote from bean in the plant kingdom and the bean yellow mosaic had not been detected previously in plants other than members of the pea family. Finally we transmitted the mild mosaic of gladiolus to beans by means of the green peach aphid. Symptoms typical of bean yellow mosaic resulted, and the virus, on further transfer by this aphid from these beans to gladiolus seedlings, produced the mild mosaic symptoms again. This showed that mild mosaic in gladiolus is a form of bean yellow mosaic.

Proof that the mild mosaic of gladiolus is the same as bean yellow mosaic made it possible to apply earlier studies on the bean virus to the problem in gladiolus. The virus was found not to be seed-borne in gladiolus; it had been shown not to be seed-borne in legumes. Other species of aphids, notably the pea aphid, were known vectors. The virus is of the nonpersistent type; it is acquired by the vector aphids in a few minutes feeding on a diseased plant, but is soon lost by the vectors if they later feed on nonsusceptible plants or if they do not feed for a time. Thus susceptible crops are usually safe from infection when grown at relatively short distances from sources of virus. Furthermore, many strains of bean yellow mosaic are known to occur; and strain variation in the causal virus may account for minor variations found in the mosaics from several iridaceous plants. Commercial gladiolus now take their place along with red clover, sweetclover, and alfalfa as reservoirs of bean yellow mosaic as sources of infection for crops like beans, peas, and the sweetpeas. Considered solely as a gladiolus disease, bean yellow mosaic is of minor importance, for few varieties are seriously disfigured by infection. On the other hand, gladiolus have consistently proved bad neighbors for beans in the West.

CUCUMBER MOSAIC virus was detected in gladiolus in Tasmania and in England before it was found in North America. It and the tobacco ring spot virus were isolated from gladiolus in Ontario and Wisconsin in 1951. Subsequently at Beltsville, Md., we isolated cucumber mosaic virus and to-

bacco ring spot virus from a number of variously diseased gladiolus submitted from several States for diagnosis. In our experiments the tobacco ring spot virus has been successfully introduced into gladiolus seedlings and isolated again from them, but no symptoms occurred in leaves or flowers. Gladiolus seedlings experimentally infected with cucumber mosaic showed white streaking in petals and white or yellowish streaks in the leaves. Cucumber mosaic evidently is responsible for some of the streaking not infrequently encountered in gladiolus. Gladiolus also must be recognized as a reservoir of these viruses as well as the bean yellow mosaic virus.

ASTER YELLOWS, a common virus disease well known in a wide range of plants, also affects gladiolus. Diseased gladiolus develop green flowers and uniformly straw-yellow leaves in Eastern States. The plants usually shrivel and die down rapidly after the symptoms appear. We proved in 1948 that aster yellows causes these effects. The disease is damaging but usually is infrequent in gladiolus, even in areas of high intensity of aster yellows. The reason for this low prevalence in gladiolus is unexplained, for the six-spotted leafhopper, the vector of the virus, feeds readily on gladiolus.

A DISEASE called grassy-top developed in up to 50 percent of the plants of some commercial fields of gladiolus in Florida and Alabama in 1952. The corms that gave rise to the disease were all grown the previous season in one locality in Oregon. Affected plants developed several weak yellow shoots instead of the single vigorous shoots of normal plants. Roots also were thin and weak. Corms had rough, hard bases, small warts at the nodes, and enlarged and irregular cores. From such grassy-top gladiolus the six-spotted leafhopper transmitted western aster yellows to China aster, celery, and zinnia. When the virus was returned to gladiolus in early stages of growth, the plants were killed. Gladiolus infected in later stages of growth, after new corms were well developed, survived to the next season. Such gladiolus, infected after flowering, apparently give rise to grassy-top symptoms when planted again. This outbreak is the only one known in which aster yellows has assumed commercial importance in the gladiolus crop.

OTHER GLADIOLUS DISEASES possibly caused by virus are mentioned briefly in gladiolus publications. They have symptoms similar to known virus diseases. Visible pathogens are lacking and proof is lacking that virus is the cause.

R. O. Magie, of the Gulf Coast Experiment Station, Bradenton, Fla., mentioned green petal stunt in the varieties Spic and Span and Golden Arrow, in which stunted petals show green veins, florets often fail to open, and plants are stunted or killed.

The disease called white pitting, known in Florida and sent to workers at Beltsville, has been found to persist from year to year in the corms. White pitting, characterized by short, stiff brittle leaves, white pitting of petals, and white or brown streaking in leaves and stems, is known only in Florida thus far.

GLADIOLUS STUNT, distinguished by plants and spikes much shorter than normal, with no mottling, streaking, or distortion, may prove to be a virus disease. Stunt is difficult to recognize until the symptoms are fully expressed. It is said to be widely distributed and responsible for much of the decline in vigor of old varieties, and, therefore, is feared more by growers than the other known or suspected virus diseases.

The gladiolus thus is known to be subject to four well-known viruses of wide host range—bean yellow mosaic, cucumber mosaic, tobacco ring spot, and aster yellows viruses—and to several suspected virus diseases. But we do not have all the information we should have about the agents that

transmit them, their host range, and varietal reactions. Until more detailed knowledge is available there is little basis for suggesting a sound program of control.

Dr. Magie, in the North American Gladiolus Council Bulletin No. 20, December 1949, suggested the value of certifying planting stock of gladiolus, comparable to the program of certifying white potatoes. The stocks, he wrote, might be "produced in parts of the country where disease spread is naturally light or absent." He was concerned mainly with fusarium rot control, but he also mentioned virus control. Such a program would be good if it could be put into practice. But no one knows now which areas are favored by light spread of gladiolus viruses; more research is necessary before the gladiolus industry can follow the path of the potato industry; and the number of gladiolus varieties in the trade, already enormous, is continually augmented by the new productions of hundreds of amateur breeders. Therefore we find less interest in protecting the present commercial varieties than in developing new sorts. Of course the situation would improve if breeders of gladiolus would take all possible care to avoid exposing their seedlings to sources of infection so that only disease-free new varieties might enter the trade.

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Blights of Lilies and Tulips

C. J. Gould

Whether one grows tulips or lilies for fun or for profit—in Brooklyn, Keokuk, or Seattle; in the garden, field, or greenhouse—he probably has more grief from botrytis blights than from any other cause.

The blights, or "fire" as they are sometimes called, are world-wide in distribution and most prevalent in cool, moist areas. They are caused by similar, but distinct, fungi. Most common on tulips is *Botrytis tulipae*. Most common on lilies is *Botrytis elliptica*.

Tulip blight begins with a diseased bulb or with contaminated soil in which the fungus is living on old remains of the tulip plant. As the young tulip shoot pushes through the ground, it becomes infected if it comes in contact with the fungus and may be changed into a distorted, grayish-brown body covered with powdery masses of spores. Sometimes only part of a leaf is diseased. Often the entire shoot is affected. Soon the leaves of the nearby tulip plants become spotted with small, circular, yellow or brown dots. Many of the spots remain small and dry up, but others, (especially if the weather is cool and moist) enlarge rapidly. Their color becomes a grayish brown or brown, with a dark water-soaked margin. Powdery masses of spores often form in the center. These, when blown to other plants, initiate more spots, which may become visible within 24 hours.

Large spots near the leaf base often