

14. PATHOLOGICAL ASPECTS OF SORGHUM DOWNY MILDEW DISEASE (*SCLEROSPORA SORGHI*) ON MAIZE AND SORGHUMS

R. G. Kenneth* and G. Shahor**

For a number of years, a new method, with various modifications, has been known for inducing systemic infection of maize and sorghums without resorting to the use of oospores, which all too often fail to give high infection rates even on susceptible hosts. Germinating seeds are usually sandwiched between sporulating leaf pieces to bring about infection. In a series of experiments, we elucidated certain facts concerning this method (3). Conidia from either systemically—infected or local-lesion-infected leaves of a sorghum×sudangrass hybrid (cv. Vidan), rather than hyphae or other structures arising from the leaves, could bring about systemic infection when inoculated on 1-14-day-old Vidan and maize. Oospore formation (oogonia) occurred on lower leaves of both hosts, at times within 14 days after seed germination in Vidan, 28 days in maize. Inoculation of 1-2-day-old seedlings during the first two days almost invariably resulted in systemic chlorosis already on the first leaf, whereas inoculation of older seedlings caused chlorotic symptoms to appear first on later-forming leaves. In early-inoculated seedlings, shoot inoculation resulted in much higher disease incidence than did inoculation of the coleorhiza, and delayed the symptoms less. In two-week-old Vidan in which the third leaf was 2/3 its maximum size, inoculation of (a) the coleoptile, (b) the area from just below the first leaf till just below the second leaf blade, (c) the second and third leaf junction, all resulted in systemic infection, with highest incidence by coleoptile inoculation; partial leaf chlorosis first appeared in (a) on the 4th or 5th leaf, in (b) on 5th or 6th leaf, and in (c) on 5th leaf. This shows that penetration, without symptoms, had occurred as far as the meristematic tissues of young leaves still within the leaf tube. Inoculation of mid-blade of the second and third leaf resulted in local lesions on these parts. Conidial inoculation of young Vidan tillers which had sprouted after cutting down healthy mother shoots resulted in systemic infection with chlorosis starting on the third leaf. Two sweet corn hybrids—NK1304, considered resistant in the field, and Buttersweet, very susceptible, proved equally susceptible when inoculated at 5 days of age. As spore showers could initiate systemic infection of maize in the field, as shown by one field in which there was 68% infection in the row adjacent to heavily stricken Vidan, dropping gradually to 5% seventeen rows away, it is important to clarify whether resistance of maize cultivars to oospores may differ from resistance to conidial inoculum. The reasons for the precocious production of oospores in these greenhouse experiments as against their late appearance in fields should be investigated, for they remain a necessary link in the oversummering and initial infection by the organism, barring seed transmission, which has yet to be demonstrated in Israel. If the factors controlling oospore formation were better understood, it is conceivable that means for preventing their appearance could be devised, thus eliminating this all-important inoculum for initial infection. As it is, apical leaves emergng completely blanched in maize and sorghum, yielded neither

* Associate Professor and

** Graduate Student, Department of Plant Pathology and Microbiology, Faculty of Agriculture, Hebrew University, Rehovot, Israel.

oospores nor conidia (those leaves emerging already with oogonia produced no conidia). Safeeulla has shown that the more there is sporulation from the leaf, the less oospores form subsequently on that leaf. I do not see much possibility of inducing greater sporulation (and thus, less oospores), nor is it desirable, except perhaps in *S. graminicola*, where sporangia normally are of very little importance. Perhaps, however, other factors can practicably prevent oospore formation, such as plant nutrition, light conditions, etc.

On the level of basic research, it would be worthwhile to find out if conidial inoculum derived from a single conidium will allow oospore formation in the plant. In other words, is the fungus homothallic? Or if heterothallic, can it be induced to produce oogonia and antheridia, as in some species of *Phytophthora*, by using triggering agents, such as *Trichoderma* contaminants?

Although local lesions have definitely been found by us on lowest leaves of sweet corn in the field a few years ago, and sporulation was evident, they did not result in plants which later displayed systemic infection, and were not at all important until this summer. In one field of Jubilee sweet corn with 50% systemic infection, we found almost 40% of the plants with short to long narrow, sporulating local lesions. They appeared on any part of the blades of the middle-tier of leaves of plants just prior to and during tasselling, and also on apical areas of leaves with half-leaf (systemic) symptoms. Sometimes single lesions appeared on a leaf, but often a great many lesions found on a leaf.

In one field of forage maize (cv. MITZKI LAVAH), displaying 2.8% lodged plants, are found that 11.4 times as many mildewed plants lodged as did non-mildewed plants. It would be worthwhile to know whether there is a tendency to lodge in other downy mildew diseases.

As mentioned by Bain (1) for sorghum, and Kenneth & Shahor (3) for sorghum and maize, sorghum downy mildew may be accompanied by seedling blight. Bain showed a strong correlation between systemic mildew infection by way of oospores and a blight caused by *Fusarium moniliforme*, with blight far more frequent in plants showing downy mildew symptoms than in those free of symptoms. As blight appeared a few days after downy mildew symptoms (2 weeks after emergence), it was possible that the mildewed plants were more susceptible to the blight. He stated, however, that the *Fusarium* inoculum in seed or within the oosporic material could possibly have become active much earlier, predisposing the seedlings to downy mildew infection, and furthermore suggested that such an action "could account in part for results reported by Clark *et al* (2) relative to low quality seed and incidence of downy mildew". There is no denying that the latter possibility exists. Our experience, however, in greenhouse experiments shows that systemic downy mildew, in sorghum and maize, can very frequently be lethal in itself, when inoculum consists of conidia and infection occurs early. In such case, chlorosis already appeared on the first leaf, whereas with oosporic inoculum we have never found chlorosis to begin earlier than the second leaf. (It would be well to ask this point as to when chlorosis first appears in seed-borne downy mildew.) Such seedlings suffer from lack of chlorophyll and should be weak, and wilting very often occurs by the 3-leaf stage, followed by death at 4-leaf stage. We found that wilting Vidan seedlings, grown in sterilized soil, as yet showing no discoloration of lower part of shoot, often yielded no colonies of fungi when uprooted and plated out on agar medium. It is probable that plants weakened from the start by systemic downy mildew would also be subject to invasion by various parasitic and adventitious saprophytic organisms which could hasten their death. When inoculated late with conidia to yield systemic downy mildew chlorosis from the 3rd leaf onwards, they never showed wilting symptoms.

The low infectivity of oosporic inoculum we and others sometimes experienced may possibly result from continued dormancy of oospores, or from non-viable oospores, or

from the results of yet unknown processes connected with soil or seed conditions (perhaps depth of sowing, soil moisture, nutrition, etc.). One factor that could possibly provide an explanation for a series of failures is mycoparasitism of oospores by a chytridiaceous fungus. Sieved, sandy-loam soil used for our experiments was shaken in suspension with oospores at different temps.; within a few days what appeared to be germination of oospores by vesicles producing zoospores was observed. These were in fact the double-walled, hyaline epibiotic sporangia of a species of *Phlyctochytrium*, tentatively determined to be *P. lippii*. whose rhizoids were within the oospore, and which grew within a few hours to maximum size; the cytoplasm within divided into many small uniflagellate zoospores, which then escaped through 3–8 small openings. The zoospores are capable of germinating on oospores, effecting penetration and themselves turning into epibiotic sporangia from which zoospores are released. Infection occurred within 2½ days, with 90% infection within 6 days at 28°C, although slower infection occurred at 17° and 34°C. The fungus was isolated and grown (coll. #2741) on peptone-malt extract-glucose agar, and remained alive at 6°C for two years, and still infective. It grew at 17–34°C (opt. 23–27°C). The fungus is capable of infecting some species of live plant-parasitic and free-living nematodes as well as various pollen grains (Kenneth, Cohn and Shahor, unpublished data), and could thus probably thrive in many soils and reduce oosporic inoculum. Fungi of the *Rhizophydium-Phlyctochytrium* complex had been found in the past to attack oospores of *Sclerospora effusa* and *Albugo* (4) and *Peronospora tabacina* (5), and in the latter case were first erroneously thought to represent germination of the oospore. We are at present working on the dynamics of this fungus in soil; maize and sorghum pollen showers are so heavily deposited on the soil late in the growing season when many oospores reach the soil that it is conceivable that numerous oospores are immediately eliminated by this fungus.

References

1. Bain, D. C. (1973). Association of *Fusarium moniliforme* with infection of sorghum seedlings by *Sclerospora sorghi*. *Phytopath.* **63**: 197–8.
2. Clark, L. E., Frederiksen, R. A. and Glueck, J. A. (1970). Effect of seed quality on reaction of sorghum to downy mildew (*Sclerospora sorghi*). *Assoc. So. Agr. Workers, Inc.* 67th Ann. Conv. Proc. 47.
3. Kenneth, R. and Shahor, G. (1973). Systemic infection of sorghum and corn by conidia of *Sclerospora sorghi*. *Phytoparasitica* **1**(1): 13–21.
4. Melhus, I. E. (1914). A species of *Rhizophydium* parasite on the oospores of various *Peronosporaceae*. *Phytopath.* **4**: 55–62.
5. Person, L. H., Lucas, G. B. and Koch, W. J. (1955). A chytrid attacking oospores of *Peronospora tabacina*. *USDA Plant Dis. Repr.* **39**: 887–8.