# 10. EPIDEMIOLOGY OF MAIZE DOWNY MILDEWS WITH SPECIAL REFERENCE TO THOSE OCCURRING IN ASIA

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In considering the caution of an epidemic, three aspects need to be taken into account: circumstantial, biological and mathematical (Lal, 1973). The third aspect only emphasises a facet, albeit an important one, of the research approach. The former two encompass what in plant pathological literature has been familiarly described as host-pathogen-environment triangle (Bourke, 1970). Gäumann (1950) has listed 8 conditions for the establishment of an epidemic. Three pertain to the host, 4 to the pathogen, and the last defined as "optimal weather conditions for the development of the pathogen" was designated as 'metereopathology'. The accent on pathogen may be noted. 'Meteopathology' of Bourke (1970) *sensu stricto* is not congeneric with 'metereopathology' of Gäumann (1950).

Van der Plank (1963) in the only book on plant disease epidemics does not discuss a single from the group of diseases to which we have to pay attention in this exposition. Nonetheless he has brought to bear mathematical rigour on our thinking about epidemics. In such attempts the underlying objective has been to know in advance whether a disease outbreak in a given situation would occur of an intensity sufficient to damage the crop and if so what timely action of a preventive kind (application of fungicides, etc.) can be undertaken. Such action in the present state of our knowledge of maize downy mildews is out of question because: (a) no economical chemical control means has yet been devised and (b) the monetary value of maize crop in many parts of the world, at least in Asia, is not remunerative. There will be thus no incentive for the farmer to resort to such a means even if it becomes known or available in the forseeable future.

It will be the endeavour of this exposition to indicate that the number of decisive factors in downy mildew outbreaks is not large. Furthermore, identification and mobilization of host resistance—an international activity undertaken in recent years with some measure of success—offers the best means of combating downy mildews in maize.

**General Considerations:** Weston (1920, 1923a, 1924) has made notable epidemiological observations which apply to downy mildews in general. An important aspect that possibly sets this group of diseases apart is the nocturnal production of propagules (conidia/sporangia) and host infection by germ tubes/zoospores also in dark. Gäumann (1950) has alluded to this phenomenon more vividly: "It is a popular belief that downy mildew arises 'overnight', a statement expressing two facts: that light normally inhibits the formation of asexual frectifications and that the few hours of a warm summer night are sufficient for the formation of a mat of conidiophores". Safeeulla and Thirumalachar (1956) have, however, pointed out that this activity of the pathogens is linked with favourable conditions that prevail in night time only (especially around midnight) in nature. If these conditions are made available to the pathogen, it is able

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to reproduce asexually independent of the diurnal day-night rhythm. The crucial factors seem to be a temperature range of 20–25°C, a condensed film of moisture on leaves for a duration of at least 12 hours (which results when relative humidity in the crop environment is above 90%) and lastly light.

Here I wish to draw your attention to a phenomenon which possibly explains some of the data presented not only by Safeeulla and Thirumalachar (1956) but others also. The former authors noted that in *Sclerospora graminicola* a "full crop of sporangia" was produced on one lot of excised leaves of *Pennisetum typhoideum* after one hour's incubation (8–9 P.M.). In *S. andropogonis—sorghi* production of a "full crop of conidia" was achieved when excised sorghum leaves were incubated for a duration of 3 hours (8–11 P.M.). Exconde *et al.* (1967), in *S. philippinensis,* observed production of as many as 10,282 conidia/sq. cm. in 'natural darkness with moisture' while in 'artificial darkness with moisture' the number did not exceed 49/sq. cm. Similarly in *S. maydis* conidial production reached its peak on a diseased plant wetted by dew at 10 P.M. (Semangoen, 1970) and in *S. sacchari* more conidia were produced on leaves excised in the evening than those clipped in the morning (Sun, 1970). Chang and Wu (1970) have indeed observed a diurnal periodicity of conidial production in this pathogen. In Queensland also, "Daily exposure to sunlight seemed to be necessary to produce daily sporulation" (Hunt, 1940) in the sugarcane downy mildew. Shah (1973) in Thailand, also obtained peak conidial production within a shorter incubation period when maize leaves infected with *S. andropogonis-sorghi* were collected in night time. This periodicity was noted to be influenced by 6 hours' of light exposure, Sclerosporas thus do appear to possess a diurnal rhythm of asexual reproduction even though the rhythm's full expression is conditioned somewhat by temperature, moisture and especially light. The data of Chang and Wu (1970) in particular suggest the existence of a circadian rhythm which seems to be endogenous in nature. This biological clock or physiological oscillation of asexual reproduction has been utilized in carrying out artificial inoculations in maize during a period past midnight with successful results. In *Sclerophthora rayssiae* var. *zeae* the rhythm does not resemble that observed in species of *Sclerospora* in that peak sporangial germination occurs in material collected at 4 P.M. rather than in collections made in night time (Singh *et al.,* 1970). Accordingly, inoculations in the afternoon lead to adequate infection.

The following diseases will be considered which are the major ones in Asia: (a) Philippine Downy Mildew (*Sclerospora philippinensis*), (b) Java Downy Mildew (*S. maydis*), (c) Sorghum Downy Mildew (*S. andropogonis-sorghi*), (d) Sugarcane Downy Mildew (*S. sacchari*) and (e) Brown Stripe Downy Mildew (*Sclerophthora rayssiae* var. *zeae*). Records of crazy top (*Sclerophthora macrospora*) on maize in the Orient have remained unsubstantiated although the pathogen does occur on rice, *Triticum, Eleusine* and other graminaceous hosts. Moreover, information on its epidemiology can be found in the excellent review of Ullstrup (1970).

**Philippine Downy Mildew (*Sclerospora philippinensis*):**

Historically, the disease was first recorded in India in 1912 even though it was, through a strange quirk of inadvertency, misdetermined first as *S. maydis* (Rac.) Bult. (not Palm) and later as *S. indica* Butl. The error has been perpetuated to this day. However, it has never appeared in an epidemic form on maize. In a field a certain number of individual plants alone appear to be severely affected. In one area of its occurrence (Delhi) infected clumps of *Saccharum spontaneum* (*Kans* in vernacular) have been found to be the main disease source (Chona and Suryanarayana, 1955; Suryanarayana, 1961; Suryanarayana *et al.,* 1970). Maize plants get infected through conidial transfer from *Kans* in the crop season. The appearance of the disease on the grass is one

week or 10 days earlier than on the adjacent maize crop. In the current year (1974) because of more than average rainfall in July infection on the grass was first noticed around 22nd July while on the adjacent maize plants it appeared on 28th July or so. Whether the survival of the pathogen in *Kans* takes the form of some kind of perennating mycelium or some other means remains to be determined.

The sources of infection in the Philippines appear to be diverse. Weston (1921) undoubtedly found it among others on *Saccharum spontaneum* but its role in perpetuation is confounded because of the co-existence of *Sclerospora spontanea*. Exconde *et al.* (1968a) have stated: "Because of abundance in some corn fields of *Saccharum officinarum, S. spontaneum, Sorghum bicolor, S. halepense* and *S. propinquum,* any of these could easily serve as an important reservoir of *S. philippinensis*". Severe natural infection in "field grown sorghum" was observed in 1971 in Mindanao where maize is grown almost throughout the year.

The occurrence of the disease in epidemic proportions exhibiting highest virulence among the Asian Sclerosporas in the Philippines is a phenomenon to be reckoned with. The situation is possibly ascribable to the continuous cropping of maize both in wet and dry seasons. While in the latter the incidence may not be high, survival of the pathogen in a non-resting phase (conidial stage) is assured. The cyclical chain of conidial production, because of the assured availability of the living host throughout the year, permits the pathogen to multiply in astronomical proportions and in doing so it is also able to maintain its virulence at a high level. Weston (1923a) has estimated that in a single night 758,033,400 to 5,946,069,000 conidia may be produced from one infected maize plant.

The occurrence of oospores was recorded by Acedo and Exconde (1967). Although their role in local disease outbreaks has not been fully elucidated, Thurston (1973) has alluded to the danger that these may pose in long distance dissemination. Presently not much is known regarding the role of seed in the perpetuation of the disease, although Weston (1920) could trace the mycelium in the cob along the funiculus of attachment in absortive kernels and occasionally in the seed coat and the endosperm. Indeed Weston (1923b) recommended a method of treating maize seed to destroy adherent spores of downy mildew.

Epidemics of Philippine Downy Mildew, as indicated in the preceding discussion, result from infection chains developing through several successive conidial generations. These last for some weeks to more than two months (Weston, 1923a). The temperature, moisture and light conditions governing conidial production, germination and infection do not appear to differ markedly. We may agree with Gäumann (1950) that "The same ecological factors that favour sporulation of the downy mildew also favour the germination of its conidia and the success of its infection".

According to Exconde *et al.* (1967) a maximum number of conidia (upto 20,160/sq. cm.) developed on leaves which were "continuously wet for 12 hrs". While the optimum temperatures for conidial formation have not been precisely determined those for germination are 19–20°C (Exconde, 1970). The range is 16–28°C. Germination was considerably enhanced in dew water and in plant whorl exudates. While investigating the relation of disease incidence with planting time and metereological factors, Exconde *et al.* (1968) have reported that highest incidence was observed in July, November and December. The rainfall, temperature and relative humidity for these 3 months respectively were 13.5, 40.5 and 13.2 cm., 27.5, 27.0 and 26.3°C and 88.0, 88.0 and 88.4%.

Interruption of these infection chains should lead to reduction in disease incidence. Alternate spray of Dueter and Dithane M 22 or M 45 within the first month of crop growth (Dalmacio and Exconde, 1971) and combination of seed treatment, plant covering and foliar spray with chloroneb (Demosan) (Schultz, 1971) have proved effective on

this disease. However, as stated earlier, such measures can not be recommended for large maize hectarages. The more feasible alternative is to reduce the inoculum level by spreading the cultivation of resistant host cultivars. Indeed it has been found that the overall disease incidence as well as sporulation on resistant materials such as Aroman white and Aroman 206 is significantly lower (incidence 21.3–42.1%, sporulation 7,409 conidia/ml) than on the susceptible types (incidence 93.9%; sporulation 22,172/ml) such as sweet corn, pop corn and PH 801 (Exconde *et al.*, 1967, 1968; Barredo and Exconde, 1973). New varieties possessing resistance have been released in the Philippines and they have spread approximately in 10–15% of the country's maize area (Renfro, 1973). The prospects are bright that the disease damage would be reduced significantly within the next five years or so.

In Nepal, the disease was first observed in 1966; epidemics occurred in 1967 and 1970 (Shah and Tuladhar, 1971). The disease severity was highest (76.3%) in crop planted between 16th and 30th June during which time the total precipitation was 24.7 cm., the average temperature was 28.3°C and relative humidity 83.2%. The disease did not develop in the crop sown "after August and prior to May" (Shah and Tuladhar, 1971).

**Java Downy Mildew (*Sclerospora maydis*):**

Information on epidemiology of this disease has been reviewed by Semangoen (1970).

Although through artificial inoculation, disease could be induced on *Euchlaena mexicana,* its role in perpetuation is negligible for the grass is not native to, nor widespread in, Indonesia (Semangoen, 1970).

Purakusumah (1965) obtained as much as 36.5% disease incidence when seeds from infected plants of variety 'Metro' were planted. As Semangoen (1970) has commented, immature seeds from diseased plants showed evidence of mycelium but it was not traceable in mature seeds. Planting of "wet fresh" seeds led to disease outbreak but the same when air dried before planting produced only healthy seedlings. Thus Semangoen (1970) concluded that "perennation of the fungus in seeds seemed not to play an important role".

Attempts by Semangoen (1970) and earlier investigators have failed to show the occurrence of oospores. Studies on survival in soil also have given negative results.

In Java, the dry season extends from May to October. During this period farmers grow maize in "sawab" or irrigated ("wet-paddy") fields. In the wet season, however, maize is planted in "tegal" or non-irrigated fields. Semangoen (1970) has stated that "downy mildew infection on early rainy season maize comes from dry season maize in the wet-paddy-field area". Greater the proximity of the two types of fields, higher is the disease incidence. The situation becomes comparable to that of the Philippine Downy Mildew in the Philippines in that the host is available throughout the year for the pathogen to maintain itself through successive conidial generations. And it is the conidial stage which is the culprit in the epidemic.

The relationship of temperature, moisture and light conditions to asexual reproduction is also identical. Peak conidial production occurs at 3–4 A.M. "when plant surfaces are covered by dew and the temperature is lower than 24°C" (Semangoen, 1970). Conidial germination was highest (95.4%) in guttation water. Infection also averaged 74.0% when conidial suspension prepared in guttation water was inoculated. In comparison, the average infection for "leaf leaching water" and distilled water was 13.1 and 20.2% respectively (Semangoen and Soemadi, 1971). The amount of infection did not increase significantly with prolongation of the humid period. Thus what is crucial is that the host surface should be wet at the time of release of conidia in the early morning. Advent of dry weather on the following day will not materially affect the resulting disease

incidence (Semangoen, 1970). Perhaps in this respect it differs from *S. philippinensis* where a 12-hour period of continuous wetness is required for optimum infection.

**Sugarcane Downy Mildew (*Sclerospora sacchari*):**

Epidemics on maize due to this pathogen are known to occur in Taiwan where it is transmissible to sugarcane and *vice versa* (Change, 1970, Sun, 1970). Although the fungus occurs in Australia, Fiji Islands, India, New Guinea and the Philippines, outbreaks of the disease in these countries are sporadic and not so damaging as in Taiwan. Furthermore, the damage to the maize crop is more than to sugarcane.

In India, Singh (1968) recorded it from the Tarai area of Uttar Pradesh where sugarcane and other species of *Saccharum* are common. However, natural infection on these hosts has not been found so far. Artificial inoculation with conidia does lead to infection in sugarcane. Subramanian (1931) in Pusa, Bihar, found it on sugarcane and he was successful in transferring it to maize. But the more common disease on the latter host in that area is the Philippine Downy Mildew. It is possible that there might be occurring two strains or pathotypes—one which freely hops from maize to sugarcane and back; the other perhaps prefers only maize.

InTaiwan, the first serious outbreak occurred in 1954 (Chang, 1965). The disease became destructive since 1962 possibly because of the cultivation of a highly susceptible double cross hybrid, Tainan No. 5. The epidemic of 1964 was devastating; it continued to rage till 1967 but has declined since then.

The main infection source is the presence of diseased sugarcane plants in the vicinity of maize fields. The physical proximity of infected sugarcane (in a radius of about 0.8 km) is *sine qua non* for successful infection of maize (Sun, 1970). Although *Tripsacum dactyloides* and Broom corn do get infected on inoculation, in nature their role in transmitting the disease is negligible (Chang, 1966).

Oospores do get formed under Taiwan conditions whose role in epidemiology, according to Sun (1970), remains to be determined. In other countries also, they occur and their role in initiating infection or in establishing disease in new areas should not be overlooked. They are produced more readily and in greater abundance on sugarcane then on maize.

Infected seed can act as a carrier of disease only if it is planted immediately after harvest without dehydration and without storage—a practice which is not common (Chang and Twu, 1965). Chang and Chen (1969) have observed that reduction of seed moisture under 20 percent inactivates the pathogen and the disease does not develop in plantings of such seed.

Conidial production, like other downy mildews is nocturnal, between 1 and 4.30 A.M. (Chang and Wu, 1970). The optimum temperature is 22–25°C (Chang, 1970; Sun, 1970). Leaves collected from 30-40-day-old plants in April-May produced the highest number of conidia at 25°C(Chang and Wu, 1969). The minimum relative humidity required for conidial production was 86 percent; the optimum range was 95–100 percent (Chang and Wu, 1969). On sugarcane leaves incubation for 8–10 hours around 20°C and 8–11 hours around 25°C is required for optimum conidial production which lasts for about 6 hours (Chang and Wu, 1968). On maize, it may be 7–8 hours for leaves detached at 6 P.M. In this respect it is more akin to Philippine Downy Mildew than to Java Downy Mildew.

Conidial production is stimulated by exposure to incandescent light of 200 Lux intensity on maize and of 600 Lux on sugarcane leaf. The duration of exposure varied from 3–12 hours (Chang and Twu, 1972).

Conidial germination ranged from 81.1 to 100 percent in the temperature range of 16–31°C; between 19–28°C it was invariably 100 percent (Chang and Wu, 1969).

The time necessary for penetration of the host by the conidial germ tubes in a temperature range of 19–28°C was 3 hours or less (Chang, 1972a). The incubation period was 15–18 days in a temperature range of 19–22°C and 11–13 days between 25–28°C (Chang, 1972b).

An important host factor in disease development is plant age. Sun *et al.* (1966) observed that highest incidence of disease (86.0%) was in 13-day-old plants. Disease would not develop on plants more than 5 weeks old.

Two plantings each month throughout 1966 and 1967 of susceptible hybrid Tainan No. 5 showed that the disease incidence ranged from 78.8% to 100% from March to October. The minimum mean temperatures that prevailed during this period ranged from 12–28°C in 1966 and 12–25°C in 1967 (Chang and Cin, 1968).

A noteworthy aspect is that the period favouring disease development in Taiwan extends for as much as 8 months! Given the cultivation of susceptible hybrid like Tainan 5 especially in areas of infected sugarcane, the epidemic outbreaks are sure to ensue. A noticeable decline in disease incidence since 1967 has been ascribed to (a) vigourous and systematic roguing out of diseased plants in sugarcane fields and planting of resistant sugarcane cultivars, (b) discouraging maize cultivation in diseased areas (Putze and Ichu of Chiay and Yen Shin of Tainan) and (c) extension of cultivation of resistant hybrids such as Tainan Nos. 11 and 12.

**Sorghum Downy Mildew (*Sclerospora andropogonis-sorghi*):**

It is noteworthy that outbreaks of the disease occurred in the sixties in several parts of the world: in 1964 in Texas (Frederiksen *et al.*, 1970), in 1968 in the State of Guerrero, West Mexico (de Leon, 1970), in the States of Karnataka (Govindu *et al.*, 1970) and Rajasthan, India and Nakorn Sawan Province of Thailand (Boon-long *et al.*, 1972). It was confined to North-East Israel on Sudan grass upto 1966; in 1968 it broke out as far as 110 km away on Sorghum×Sudan grass hybrids as well as in an irrigated field of dent corn (Kenneth, 1970). Why 1968 should have been an auspicious year for this pathogen to appear in such far flung areas of the globe is a problem which merits investigation.

As indicated earlier (Payak, 1973), there appear to occur two races or pathotypes. For the purpose of this discussion we may designate them as Sorghum race and Maize race. They are characterized as follows. Sorghum Race: Oospore production in maize tissues common, accompanied by malformation/deformation of ears and tassels, prevalent in areas where the source of infection is traceable to sorghum. Distribution: Mexico, USA, Israel, India (Karnataka, Maharashtra, Tamil Nadu). Maize Race: Pronounced yellowing of maize foliage, stunting, with or without malformation/deformation, oospore production absent, infection source not traceable to sorghum. Distribution: Thailand, India (Rajasthan), Dange *et al.* (1973) have recently attempted to show that in Rajasthan, *Heteropogon contortus* acts as a collateral host of this race. This requires confirmation but it does indicate that the infection source is not traceable to sorghum.

Jones *et al.* (1972) have observed hyphae of the pathogen in the carpellate flowers (style, ovary wall and nucellus) and mature seeds (pedicel and pericarp but not endosperm and embryo) of maize. Disease developed only in plants arising out of seeds in soft dough stage. The mycelium was inactivated when seed moisture was reduced to 9% and by storage of seed for 40 days. Safeeulla (unpublished) has observed occurrence of oospores in the persistent glumes of sorghum which move along with the seed and may be responsible for infection to break out in new areas.

The factors affecting conidial production in this downy mildew are well documented (Safeeulla and Thirumalachar, 1956; Kenneth, 1970 and Shah, 1973). Both Kenneth (1970) and Shah (1973) have found that the time of conidial production is between

midnight and 5 A.M. Peaks are obtained at 2–3 A.M. The relative humidity should be above 89% (leaf surface wetting for a duration of 3–4 hours), temperature between 20–24°C. An exposure of light for 5–6 hours was considered necessary for each crop of spores to be produced (Shah, 1973). Kenneth (1970) observed *in situ* conidial germination between 3–5 A.M. Possibly conidiophores and conidia get detached *en masse* and "group" or "community" infections may follow.

The optimum temperature range for infection was 21–24°C. Infection was optimum when the inoculum density was 135,000 conidia/ml over a temperature range of 18–24°C. The most suitable time of infection was determined to be 3–4 A.M. (Shah, 1973).

The survival of the sorghum race is assured through the agency of oospores. Kenneth (1970) found that in sorghum oospores developed after 4 weeks of sowing. They germinate and cause infection in sorghum×sudan hybrids between temperatures of 24–29°C. One-year old oospores were as infective as the newly-formed ones. However, Kaveriappa and Safeeulla (unpublished) noted that they require a wethering period of at least 4 weeks before they can cause infection. The widespread outbreaks, mentioned earlier, possibly could be ascribed in a large measure to the versitality in dissemination and survival of the pathogen in the oosporic stage.

**Brown Stripe Downy Mildew (*Sclerospora rayssiae* var. *zeae*):**

An account of life cycles and epidemiology has been presented earlier (Payak *et al.*, 1970; Singh *et al.*, 1970). The infective source responsible for initial outbreaks has been determined to be the oosporic stage. This stage in maize develops more readily and more abundantly where higher temperatures prevail (possibly over 28°C). In such areas the number of sporangial generations that are completed in the crop season seems to be less than in areas where lower temperatures prevail. The disease severity also gets accordingly reduced. It has been shown earlier (Payak and Renfro, 1970) that the disease severity varies with the rainfall. Where the annual rainfall varies from 40–60 cm the disease intensity was described as low, moderate in areas with 60–100 cm rainfall and high in regions receiving 100–200 cm of rains.

The disease is primarily soil borne. As indicated earlier (Payak *et al.*, 1970; Singh and Renfro, 1971) the disease could be induced in locations known to be free of it by putting powdered infected leaf débris (with oospores) along with the seed in the soil. The disease in these areas has been appearing year after year. Irrespective of the type of cultivars that have been planted in these areas the disease has remained confined to the original sites.

By 1968 resistant cultivars such as Hybrid Ganga 5, Composites Kisan and Jawahar had been released. Wherever plantings of such cultivars has been made disease severity and consequent damage has been noted to be insignificant. It is believed that extension of cultivation of such materials should prove instrumental in reduction of inoculum. Epidemics of the disease were common in the mid sixties. There is a gradual decline towards moderate intensities of the disease. Possibly cultivation of resistant cultivars may be one of the factors.

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## Question and Answer

**R. A. Frederiksen,** U.S.A.: In Texas, we find tassel infection in some inbreds and hybrids in some years, and not in others. In some years, maize infection is high and sorghum infection is low, whereas in others, maize infection is low and sorghum infection is high. Similarly, oospores occur in maize in some years and not in others. They always occur, however, in sorghum. Consequently we have no evidence for races.

**Answer:** I postulated the existence of two races in Sorghum Downy Mildew with my limited experience in India. In Rajasthan, in Hybrid Ganga 5, oospores do not develop nor is there distortion or proliferation of tassels or ears, while in the southern state of Karnataka (Mysore) and Tamil Nadu, oospores develop readily and distortion of floral parts also occurs in the same hybrid. In the latter case infection is traceable to infected sorghum, but in Southern Rajasthan to date, sorghum has not shown infection even though planted side by side with infected maize.

**Sangam Lal,** India: You mentioned that peak sporangial production occurs in nature at 4:00 p.m. rather than in early morning hours. At laboratory conditions, we get sporangial production only when we keep the infected leaves near>95% humidity in the dark. Also, we get copious growth with sporangia—in morning hours on naturally infected plants. It suggests that sporangial production occurs mainly in early morning hours in nature. However, we get infection from the infected leaf bits inoculated in the evening in the whorl of test plants.

**Answer:** I should clarify that sporangial production was at its peak in material collected at 4 p.m. when inoculated in a moist chamber. It may be noted that in this pathogen, inoculations in daytime lead to infection, but this is not so in Sclerosporas.

To that much extent the rhythm of spore production is different from that occurring in Sclerosporas.

**T. Yamaguchi,** Japan: 1) Are there any data concerning spore dispersion? Are the spores caught by spore traps before the first outbreak of downy mildew in some districts?

2) Do downy mildew diseases spread gradually from the field where the first outbreak occurs? How many days does it take for the surrounding fields to get infected from the first disease outbreak under the most favourable conditions?

**Answer:** We have little information on spore dispersal, but field observations show that the distance is not large. In *Sclerospora Sacchari* according to Sun (1970), it is 0.8 kilometers.

No, studies with spore traps have not been carried out, as far as & know.

If conditions continue to be favourable, the Downy Mildew diseases may spread rapidly.

About 10 days are required for *Sclerospora philippinensis* to spread from the grass *Saccharum spontaneum* to an adjacent maize field.

**B. L. Renfro,** IACP: You stated on p. 9 that, according to work in the Philippines, a 12 hour continuous wet period is required for infection of maize by *S. philippinensis*. This seems to me to be about 2 times to 3 times as much time as really required for the germination and infection processes to be completed. Can we have a comment from you and/or either Dr. Exconde or Dr. Dogma on this point for clarification?

**Answer by O. R. Exconde:** It may appear long, but the 12-hour period starts from the time of inoculation. It is really difficult to pinpoint the exact time when actual infection takes place, that is, the exact time between germination and infection.

A somewhat related finding we had is that post inoculation moisture affected disease development. For instance, after 12 days, inoculated plants covered with plastic bags for 0, 1, 2 and 4 hours had 35%, 70%, 82.5% and 97.5% infection, respectively. Significantly, a higher infection percentage was obtained for those plants that were covered for 4 hours than those covered for 1 hour and 0 hour.