

Review Article

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Sorghum Downy Mildew of Maize – A Review

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ABSTRACT

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Corn ranks one of the four principal crops of the world. It has greater adaptability and is grown throughout the world, over a range of climatic conditions. Maize breeding programmes generally focus on yield improvement. However, several diseases are responsible for major economic losses in maize. Sorghum downy mildew is one of the most serious diseases in maize producing areas throughout the world. Although effective chemical measures are available, breeding resistant cultivars is more cost effective and environmentally safe alternative for controlling sorghum downy mildew. Effective breeding methods for producing sorghum downy mildew resistant inbreds and hybrids would depend primarily on the mode of inheritance of resistance or susceptibility to the disease.

Introduction

Maize or corn (*Zea mays* L.) is an important cereal crop of the world after wheat and rice. It is an annual, herbaceous, monoecious and protandrous plant (Dhillon, 1998).

It probably originated in Mexico and evolved from teosinte (*Zea mexicana*) (de Wet and Harlan, 1972). Being a C4 plant, it is physiologically more efficient and has higher grain yield and wider adaptation over a range of environmental conditions (Dowswell *et al.*, 1996). Maize has a wider range of uses than any other cereals as animal feed, human food and for hundreds of industrial purposes (Dhillon, 1998).

The average area under this crop of the world is 177.37 m ha with a world average production and productivity is around 872.06 million tonnes and 4.9 tonnes per hectare respectively (FAO, 2012). After the discovery of America by Europeans in Columbus' time its cultivation spread rapidly to all suitable parts of the world. The important maize growing countries are the USA, China, Brazil, Mexico, India, Philippines, South Africa and Indonesia. Maize is a relatively new crop to Asia, compared with other important cereals. This crop was introduced in the sixteenth century by European traders to Asia as an ornamental garden plant and did not become agriculturally important until 200 or 300 years later (Moore and Renfro, 1971).

In India, maize is grown in an area of 9.43 million hectares and the annual production is about 24.35 million tonnes with a productivity of 2.58 tonnes per hectare (Agricultural Statistics at a Glance, 2014). In Tamil Nadu, maize occupies 3.21 lakh hectares with an average production and productivity of 2.64 lakh tonnes and 8224 kg per hectare respectively (Department of Economics and Statistics, Chennai, 2014). It is expected to increase in future to meet the growing demands of poultry and other animals feed industry, industrial utilization and human consumption. The demand for maize is increasing every year. According to recent study (The Hindu Survey of Indian Agriculture, 2000), in 2015 the domestic demand for maize will be about 12 million tonnes and in 2030 it will be 13.5 million tonnes and for Tamil Nadu state it is about ten to twelve lakh tonnes. The commercial exploitation of single cross hybrids in maize, initially suggested by Shull (1909), is emerging again because of their uniformity in plant ear characters than other types of hybrids and high yield potential. Frey (1971) reported that yield increases in the USA after 1957 were mainly due to the wide cultivation of single cross hybrids. Due to yield advantage, hybrids dominate in maize cultivation over varieties. Hence, high yielding single cross hybrids are the need to meet the growing demand. To meet this demand it is necessary to increase the productivity of maize. One of the major factors limiting productivity in maize is the increasing incidence of pest and diseases. Of these, sorghum downy mildew (SDM) caused by *Peronosclerospora sorghi* (Weston and Uppal) C.G. Shaw is the most prevalent downy mildew in the tropical and subtropical areas of the world (Frederiksen *et al.*, 1969; Pupipat, 1975; Frederiksen and Renfro, 1977 and Williams, 1984).

Though the disease can be controlled by cultural practices such as eradication of

infected plants, deep ploughing and adjusting time of planting and by systemic fungicide, Metalaxyl either through seed treatment (Odvoidy and Frederiksen, 1984a) or foliar application (Odvoidy and Frederiksen, 1984b), their effectiveness on disease incidence is variable and in most cases, offer incomplete control. Moreover the economic costs of chemical control in maize production have been a barrier. Considering the cost of chemicals and the emergence of chemical resistance in the downy mildew pathogens (Raymundo, 2000) use of host plant resistance seems to be the most effective, economical and environmentally safe way of controlling SDM disease in maize (Rathore and Jain, 2000). The genetic information relating to host resistance is vital for making breeding decisions.

Downy mildew pathogens of maize

Among the various maize diseases, downy mildews are considered to be the major diseases. In origin, the downy mildews are “old world” diseases that now are very damaging and prevalent on the “new world crop” – maize (Shaw, 1975). None of the downy mildew diseases originated on maize (Shaw, 1975) but they possessed the ability to attack maize when maize was introduced from the new world to old world. Heavy losses (as high as 100 per cent) in maize due to downy mildew pathogens have been recorded in Philippines, Taiwan, Indonesia, Thailand, India, West Africa, Venezuela, Japan, Australia, Europe, North America and other parts of the world (Bonde, 1982 and Rifin, 1983). Twenty one species of downy mildew pathogens have been reported to attack the gramineae family (Shaw, 1975). Of these, ten species of fungi belong to three genera (seven species of *Peronosclerospora*, one species of *Sclerospora* and two species of *Sclerophthora*) have been reported to cause different types of downy mildews in maize

(Frederiksen and Renfro, 1977; Siradhana *et al.*, 1980 and Rathore *et al.*, 2002). The details are given in Table 1.

Downy mildews such as Sorghum downy mildew, Philippine downy mildew, Sugar cane downy mildew, Brown stripe downy mildew and more recently identified Rajasthan downy mildew were reported from different agro-ecological regions in India (Payak, 1975a; Siradhana *et al.*, 1980). Sorghum Downy Mildew (SDM) is particularly prevalent in the Peninsular India, in the states of Karnataka, Tamil Nadu and Andhra Pradesh reportedly causing yield losses of 30 per cent and higher (Payak, 1975a, 1975b and Krishnappa *et al.*, 1995). SDM became an internationally important disease of maize during the years of rapid expansion of the use of sorghum for grain and forage (Sabry *et al.*, 2006; Lukman *et al.*, 2013; Muis *et al.*, 2015).

It infects both maize and sorghum in warm and humid areas of the world (Frederiksen, 1980). The pathogen infects the roots primarily by oospores and the leaves by conidia and reaches the meristem causing systemic infection. Systemically infected plants do not produce cobs. If at all produce cobs they have only a few seeds and it causes severe yield loss in maize. It is an obligate parasite (Cardwell *et al.*, 1997) that cannot be cultured in the laboratory. The disease occurs in both maize and sorghum in warm and humid areas of the world (Sabry *et al.*, 2006).

Taxonomy of *P. sorghi*

SDM has two crop hosts, sorghum and maize. It is a member of subdivision Mastigomycotina, class Oomycetes, order Peronosporales and family Peronosporaceae. The widely prevalent SDM was first reported by Butler (1907) in Tamil Nadu and Maharashtra states of India. He considered it to be *Sclerospora graminicola* at that time.

Subsequently, Kulkarni (1913) in India observed that, in the asexual phase, germination was direct by means of a germ tube from conidia (rather than through zoospores from sporangia) and primarily on the basis he recommended designation of varietal rank as *Sclerospora graminicola var Andropogonis-sorghii*. Weston and Uppal (1932) described this fungus in English and further investigation of morphology and host range he named it as *Sclerospora sorghi* in 1932. In 1978 C.G. Shaw proposed the new genus *Peronosclerospora* as subgenus of *Sclerospora* based on conidial germination (by a germ tube) and he transferred genus name from *Sclerospora* in to *Peronosclerospora* that produce conidia which germinate directly by a germ tube (Shaw, 1978). From 1978 this SDM pathogen has been named as *Peronosclerospora sorghi* [(Weston and Uppal) C.G. Shaw].

Symptoms of sorghum downy mildew on maize

Systemic infection in maize seedlings is characterized by chlorosis which normally appears two weeks after sowing (Safeeulla, 1974). The leaves of infected plants tend to be narrower and more erect than those of healthy plants. Plants infected early usually die approximately four weeks after infection (Ajala *et al.*, 2003). In late infected plants, the chlorosis may be more noticeable on the lower half of the leaf which is often called half-leaf symptom. This chlorosis gradually covers the entire leaf surface at later stage (Safeeulla, 1974). Under warm humid conditions, a white downy growth is produced on the lower leaf surface some times on both surfaces also. This growth is a combination of conidia and conidiophores (Jeger *et al.*, 1998). In maize, leaf shredding is rare but it is common in sorghum (Jeger *et al.*, 1998). At flowering stage of growth, infected plants produced a bushy top, referred to a crazy top in place of

the tassel. Systemically infected maize plants generally do not form cob. In some cases when cobs formed, these are small and poorly filled (Ajala *et al.*, 2003).

Sorghum downy mildew – pathogen variability

Variability for pathogenicity in *P. sorghi* increases the potential for damage from SDM. Breeders should attempt to diversify their sources of DM resistance as quickly as possible to reduce the vulnerability of corn (Craig and Frederiksen, 1980). The first report of pathogenic variability of *P. sorghi* was found on sorghum crop from the USA in the late 1970s when a previously resistant and popular sorghum hybrid was infected with SDM (Craig and Frederiksen, 1980). Three distinct pathotypes were identified on sorghum in the USA from the differential reaction of the inbred lines Tx412, Tx430, CS3541 and QL-3 (Craig and Frederiksen, 1983) and their mode of inheritance of resistance was determined by Craig and Schertz (1985) and Sifuentes and Frederiksen (1988). Pawar *et al.*, (1985) screened 75 sorghum varieties against sixteen isolates of *P. sorghi* from different geographic regions and found that a differential reaction identified in each isolate as a different pathotype. Bock *et al.*, (2000) sampled nine isolates of *P. sorghi* from maize, sorghum and wild sorghum in Southern and Eastern Africa and they were compared for pathogenicity on different sorghum and maize cultivars which provided evidence for the existence of pathogenic variability among geographically distant populations of *P. sorghi* in Africa (Bock and Jeger, 2002). The isolates from Africa and Asia were more pathogenic than those from Americas (Jeger *et al.*, 1998).

Although *P. sorghi* typically infects both sorghum and maize (the sorghum strain), there are reports of strains that infect only maize

(the maize strain) (Bock *et al.*, 2000). Payak (1975b) postulated that three races of the pathogen *viz.*, sorghum, sorghum–maize and maize races differentiated by pathogenicity to sorghum and maize, occurred in India. The form of *P. sorghi* found in Rajasthan, India, is pathogenic to maize and *Heterpogan contortus* (L.) Beauv. but not to sorghum (Dange, 1976; Frederiksen and Renfro, 1977; Kothari *et al.*, 1980 and Siradhana *et al.*, 1980). In contrast, *P. sorghi* in Southern India (the states of Karnataka, Tamil Nadu and Andhra Pradesh) attacks maize and sorghum but not *H. contortus* (Payak, 1975b; Safeeulla, 1976 and Frederiksen and Renfro, 1977). The downy mildew prevalent in the Udaipur district of Rajasthan State in India, with *H. contortus* as the collateral host has been recently designated as ‘Rajasthan downy mildew’ (Siradhana *et al.*, 1980) caused by *P. heteropogoni*. Earlier, this pathogen was considered as a variant of *P. sorghi* (Nair *et al.*, 2004).

Epidemiology of sorghum downy mildew

A good knowledge of the disease epidemiology is required to design an effective screening method and also to recommend a control measures. *P. sorghi* has both a sexual and an asexual phase (Jeger *et al.*, 1998). During the sexual phase oospores are formed which enable the pathogen to survive the long, hot, dry and crop free periods (Williams, 1984). The asexual phase occurs in periods characterized by moderate temperature and high relative humidity (Lal, 1981). The primary sources of infection are oospores capable of surviving several seasons in the soil even up to ten years (Williams, 1984 and Bonde, 2008). Oospores germinate by germ tubes (Safeeulla, 1976 and Pratt, 1978) in the presence of host (Pratt, 1978) and infect underground parts of the plant. No germination was observed in the absence of plant roots (Pratt, 1978). Schuh *et al.*, (1987a)

determined the optimum soil condition for incidence of SDM on sorghum. A soil temperature- soil moisture combination of 25°C and -0.2 bar and a soil texture-inoculum density combination of 80 per cent sand content and 5 gram of oospore power per 100 gram of soil gave the highest disease incidence. Saturated soils and dry soil were both suppressive for disease (Balasubramanian, 1974). This was substantiated by a Principal Component Analysis of weather data (Schuh *et al.*, 1987b). The number of oospores dispersed increased exponentially with increasing wind speed (Bock *et al.*, 1997). Leaf shredding and oospore production were comparatively higher on sorghum than on maize (Bigirwa *et al.*, 1998). Infection by oospores results in systemically infected plants (Schuh *et al.*, 1988).

In the case of asexual phase conidia are produced between 15 and 23°C (Bonde *et al.*, 1985) through the stoma (Lal, 1981) In India, conidia of *P. sorghi* are produced between midnight and 5.00 AM when temperature are about 20°C and relative humidity greater than 85 per cent (Shenoi and Ramalingam, 1979 and Bock *et al.*, 1998). After conidial production and dispersal, germination occurs through one or more germ tubes and subsequent penetration occurs within a few hours (Bonde, 1982). Mature conidia begin to germinate within one hour after dislodging from conidiophore (Craig, 1987 and Cardwell *et al.*, 1994). Germination and germ tube growth took place in the conidial temperature range of 10 to 34°C (Bock *et al.*, 1999) and it declined with the increase in temperature (Bonde *et al.*, 1978 and Rao *et al.*, 1987). Conidia germinate by a germ tube (Lal, 1981) under saturated air (Lal, 1981; Bonde *et al.*, 1978 and Bonde, 1982).

In India, the optimum temperature range for conidial germination was reported to be 21 to

25°C (Safeeulla *et al.*, 1974). Plant age affects the incidence of infection. Maize plants older than 15 days were resistant to systemic infection by conidia (Bock *et al.*, 1999; Yeh and Frederiksen, 1980 and Siradhana *et al.*, 1978b). Normally conidiophores arise through stomata and these conidiophores are stout, dichotomously branched and terminating in sterigmata and conidia borne on their tips. Conidia are hyaline, thin walled and they detach easily from conidiophores (Lal, 1981) and enter into air. A very low wind speed is sufficient to disperse conidia (Bock *et al.*, 1998) and this forms secondary infection in maize field. Generally, crop escaped infection when the sowing was done before or on the onset of monsoon (Siradhana *et al.*, 1975 and 1978a). *P. sorghi* may be transmitted internally in maize seed either as mycelium (Jones *et al.*, 1972, Safeeulla and Shetty, 1977, Rao *et al.*, 1985, and Adenle and Cardwell, 2000) or as oospores (Rao *et al.*, 1984). Seed transmission appeared to only occur with freshly harvested seed or immature seeds (Jones *et al.*, 1972).

Sorghum Downy Mildew – Screening methods

SDM is one of the destructive diseases and has the capacity for epiphytotics on susceptible genotypes under favorable conditions in the epidemic areas of Karnataka, Maharashtra, Andhra Pradesh and Tamil Nadu states in India (Sundaram, 1977 and Anahosur, 1978). Breeding resistant varieties is one of the effective and cheap methods to control this disease. In any breeding programme for disease resistance, the first step is to screen all available materials against disease pathogen. A sound screening technique forms the basis of identification of sources of resistance for disease resistance breeding programme (Anahosur and Hegde, 1979). It is also necessary to investigate the inheritance and genetics of resistance (Jeger *et al.*, 1998).

Screening for resistance to the SDM has been carried out in the field (Anahosur and Hegde (1979), Cardwell *et al.*, 1997, Setty *et al.*, 2001, George *et al.*, 2003, Yen *et al.*, 2004, Nair *et al.*, 2004 and Nair *et al.*, 2005) and in the green house (Jones, 1970, Schmitt and Freytag, 1974, Craig, 1976, and Narayana *et al.*, 1995).

Field screening

Anahosur and Hegde (1979) compared the five different techniques for screening sorghum genotypes against SDM in the field and revealed that 'Infector row' planting was the most reliable technique for assured screening. In this technique, oospore inoculum was incorporated into the furrows and the highly susceptible genotype was sown two weeks early. In continuation to these infector rows, the test lines were sown where the oospore inoculum was previously incorporated. So the test lines were exposed to both oospores from soil and conidia from infector rows thus minimizing escapes.

Cardwell *et al.*, (1997) have developed 'Direct seed inoculation' method for screening maize genotypes against SDM. They followed the same steps as in the case of infector row technique. But the difference is they used conidial infected pre-germinated seeds for infector row sowing instead of direct sowing. By adopting this technique they obtained consistent and high incidence of downy mildew infected plants. This method required substantially less labour and inoculum than the spray inoculation of spreader row. In this method planting of the pre-germinated seeds required more care than planting unimbibed seed. So planting of the spreader rows is slightly slower in this direct seed inoculation method.

Disease nurseries planted in sick plot where SDM is endemic have been widely used.

Several such SDM sick plots are maintained in South Texas by Commercial seed companies as well as by the Texas Agricultural Experiment Station (Frederiksen, 1980). In India, SDM nursery has been maintained at the University of Agricultural Sciences – Regional Research Station (UAS-RRS), Mandya in Karnataka state (Setty *et al.*, 2001, George *et al.*, 2003, Yen *et al.*, 2004, Nair *et al.*, 2004 and 2005). It is considered to be the National Centre for investigations on maize downy mildew in India (Krishnappa *et al.*, 1995). However, use of these nurseries is limited to the growing season and the erratic occurrence of the disease requires repeated test to ensure reliable identification of resistant lines (Craig, 1980). To overcome such difficulty glass house techniques should be developed. So that maize genotypes can be screened throughout the year.

Glasshouse screening

Craig (1976) has developed seedling inoculation technique in which the maize seedlings were inoculated with conidia of *P. sorghi* in green house when the second leaf of the plant had unrolled enough for the leaf tip to flatten. A classification system was devised for leaf reactions based on the systems exhibited by the second leaf seven days after inoculation as resistant, intermediate and susceptible (Craig, 1982a). Many research workers have reported corn to be more susceptible to conidia than to oospores of *P. sorghi* (Frederiksen and Renfro, 1977; Frederiksen and Ullstrup, 1975; Kenneth and Shahor, 1973 and Saffeulla, 1976). So, conidial inoculation (Craig, 1976) of corn plants in the green house is a quick and relatively inexpensive method of screening for resistance to SDM. Craig (1980) noted that corn plants become less susceptible to conidial inoculum with increased age. This suggested that conidial inoculum at early stage of plant growth would be more efficient.

Narayana and his coworkers (1995) compared six inoculation techniques for artificial promotion of SDM in green house for screening sorghum genotypes. Among the six inoculation method evaluated in the green house they obtained maximum downy mildew incidence of 100 percent when seedlings at the first leaf stage were spray-inoculated. This system is generally used under a controlled environment. Seedlings of the test materials are spray inoculated with a suspension of mature conidia. The advantage of this system is that optimal conditions can be maintained and the amount of inoculum is regulated. It can also provide a rapid technique for screening large quantities of materials in short time. Schmitt and Freytag (1974) also reported that conidial spray inoculation at seedling stage was most efficient in inducing severe downy mildew infection in corn and sorghum. No reliable method has been devised for green house tests of resistance with oospore inoculum (Craig, 1980).

For comparing host reactions it is necessary to develop an accurate and precise assessment method. Normally disease assessment has been carried out as per standard procedure given by Lal and Singh (1984) worldwide in case of systemic downy mildew diseases by recording the per cent downy mildew diseased plants. Based on per cent downy mildew incidence, a rating scale has been developed as 0 – 10 per cent: resistant (R); >10 – 30 per cent: moderately resistant (MR); >30 – 50 per cent: moderately susceptible (MS); >50 per cent: susceptible (S) (Yen *et al.*, 2004, Nair *et al.*, 2004 and 2005).

Genetics of resistance to Sorghum Downy Mildew

Although effective chemical measures (Anahosur and Patil, 1980; Odvody and Frederiksen, 1984a and 1984b; Anaso *et al.*, 1989 and Sharma and Lal, 1998) are available

for SDM, breeding resistant varieties and their cultivation has been a widely accepted phenomena in most of the crop improvement programmes (Shivanna and Anahosure, 1990). Genetic information relating to host resistance would provide more relevant basis for making breeding decisions. Considerable data have been reported concerning sources of resistance (Craig *et al.*, 1977; Schmitt *et al.*, 1977; Lima *et al.*, 1982; De Leon *et al.*, 1993; Setty *et al.*, 2001; Ajala *et al.*, 2003 and Yen *et al.*, 2004). Information on the mode of inheritance of resistance to downy mildew, however, is limited in maize.

Combining ability and gene action for resistance to Sorghum Downy Mildew

Effective breeding methods for producing downy mildew resistant inbreds and hybrids would depend primarily on the mode of inheritance of resistance or susceptibility to the disease (Nair *et al.*, 2004). In countries where downy mildews are a major constraint to maize production, independent research programmes had been initiated to determine the genetic basis of resistance to the prevalent downy mildews only within their respective germplasms. Sudha *et al.*, (2004) identified NAI116 maize inbred having resistance both against Sorghum Downy Mildew and Rajasthan Downy mildew. In reports of genetic interpretation of resistance, there seems to be considerable variation according to the germplasm studied in various maize growing areas (Raymundo, 2000). Bockhold and Frederiksen (1972) and Frederiksen *et al.*, (1973) investigated the inheritance of resistance to SDM in two diallel sets of inbred maize lines in the USA. Resistance was either fully or partially dominant, depending on the particular parents involved although in one parental line which carried an additional gene, susceptibility was dominant. They concluded that two or possible three genes control the reaction to SDM.

Table.1 Pathogens causing downy mildew in maize

Sl. No.	Common name	Scientific name	Geographic distribution
1.	Java DM	<i>Peronosclerospora maydis</i>	Indonesia
2.	Leaf splitting DM	<i>P. miscanthi</i>	Philippines, Taiwan
3.	Philippine DM	<i>P. philippine</i>	India, Indonesia, Nepal, Philippines, Thailand
4.	Sugar cane DM	<i>P. sacchari</i>	Australia, Fiji Islands, India, Japan, Nepal, New Guinea, Philippines, Taiwan, Thailand
5.	Sorghum DM	<i>P. sorghi</i>	Asia, Central America, Europe, North America, South America, Africa
6.	Spontaneum DM	<i>P. spontaneae</i>	Philippines
7.	Rajasthan DM	<i>P. heteropogoni</i>	Rajasthan (India)
8.	Green ear DM	<i>Sclerospora graminicola</i>	World wide
9.	Crazy top	<i>Sclerophthora macrospora</i>	World wide
10.	Brown strip DM	<i>S. rayssiae</i> var <i>zeae</i>	India, Nepal, Pakistan, Sikkim, Thailand

Jinahyon (1973) reported that the resistance to SDM was a quantitative character indicating polygenic inheritance. Frederiksen and Ullstrup (1975) studied the resistance to SDM and indicated that resistance was dominant in some crosses and recessive in others.

Schmitt *et al.*, (1977) screened maize hybrids both in green house and in field and they compared their response to green house and field and suggested that resistance to conidial and oospore infection was under similar genetic systems.

Singburadom and Renfro (1982) in a diallel crosses of ten maize inbred lines, found that susceptibility to *P. sorghi* was expressed as a dominant character. Few crosses showed intermediate levels of resistance. Their results indicated that resistance was controlled by many genes and that both additive and non-additive gene effects were present. Genetic analysis of host resistance to SDM in India has proven that resistance to *P. sorghi* is under polygenic control.

Borges (1987) in a diallel cross of six maize inbred lines including reciprocals found intermediate disease reaction for the crosses between the resistant and susceptible lines. Their findings suggested a polygenic system for resistance to SDM in maize and additivity was clearly more important in determining disease reaction. They also found maternal inheritance for reaction to *P. sorghi*. However cytoplasmic inheritance was not detected in sorghum for resistance to SDM (Sifuentes and Frederiksen, 1988).

Predominance of additive gene action was concluded by Muthiah (1989), Geetha (1997) and Premlatha (2004) in the inheritance of resistance to SDM in maize. Additive effects are the predominant contributors to resistance (Nair *et al.*, 2004). In addition, the recent observations that genotypes resistant to SDM are invariably resistant to Rajasthan downy mildew (*P. heteropogoni*), while the Rajasthan downy mildew resistant genotypes might show differential response to SDM (Nair *et al.*, 2004 and Yen *et al.*, 2004).

Inheritance of resistance to Sorghum Downy Mildew

Breeders should attempt to diversify their sources of SDM resistance as quickly as possible to reduce the vulnerability of maize (Craig and Frederiksen, 1980). Knowledge of the mode of inheritance of SDM resistance is necessary in choosing breeding populations (Singburdom and Renfro, 1982). Studies on the inheritance of resistance provided information on the number of genetic factors involved in resistance to SDM. The identification of different genes for resistance is needed for development of cultivars with durable resistance to SDM (Sifuentes and Frederiksen, 1988). Most of the inheritance studies for resistance to SDM in maize have used generation mean analysis. It is popular method of analysis for quantitative characters.

Singburdom and Renfro (1982) obtained intermediate levels of resistance in crosses between resistance and susceptible to SDM and indicated that several genes were involved and that their effects were additive. Progenies of crosses between resistance lines were more resistant than their parents, probably because of interaction between different resistance genes (epistasis). They indicated that the reactions of maize to SDM were governed by several genes (polygenically) and that the inheritance of resistance was complex. Craig (1982b) studied the inheritance of resistance to SDM in maize using F₁, F₂ and backcross progenies and indicated that susceptibility was partially dominant and that resistance to *P. sorghi* was conditioned by two linked genes. Dominance type of gene effect was significant for resistance to SDM in maize suggested the importance of dominance in the control of SDM. Nevertheless in two crosses additive gene effect was also significant in addition to dominant gene effect (Krishnappa *et al.*, 1995). Susceptibility to SDM infection was

found to be partially dominant over resistance and resistance to SDM controlled by polygenes (Yen and Prasanna, 2001 and Yen *et al.*, 2001) where as in sorghum resistance to SDM was found to be dominant (Reddy *et al.*, 1992). No attempt has been reported in maize regarding number of genes involved for resistance to SDM using a chi-square test so far. However, reports are available in sorghum.

Studies (Rana *et al.*, 1982) suggest that in sorghum there were three genes with major effects influencing SDM resistance in the parental material. When 296 was involved in crossing, only two of the three genes segregated giving a 15: 1 duplicate dihybrid F₂ ratio; when 303 was involved, a 57: 7 F₂ ratio was obtained indicating that two of the genes segregated in a 9: 7 ratio and the third gene in a 3: 1 ratio. Hence, SDM resistance in the crosses studied was controlled genetically by three pairs of genes with both complementary and duplicatory types of interaction involved in their genetics.

Craig and Schertz (1985) used F₁, F₂ and F₃ generations of the cross of sorghum between resistant line (QL-3) and universally susceptible line (Tx412) to determine inheritance of resistance to *P. sorghi*. They obtained the F₂ phenotypic ratio as 3 resistant: 1 susceptible and its genotypic ratio as one homozygous resistant: 2 heterozygous: 1 homozygous susceptible using F₃ phenotypes. Based on the reactions of the F₂ population they indicated that resistance to each pathotype (1, 2 and 3) of *P. sorghi* was conditioned by a single dominant genetic factor.

The relationship between the genes for resistance to *P. sorghi* pathotypes 1, 2, and 3 in the sorghum lines QL3-India and SC414-12 were investigated by Sifuentes and Frederiksen (1988) with reciprocal crosses

between them and susceptible lines. The results supported the hypothesis that QL3-India has two genes conditioning resistance to each of the three pathotypes. Whereas SC414-12 has one gene for resistance to the same three pathotypes.

Since the observed segregation in the F₂ of the cross between the resistant parents was in a agreement with a 63: 1 ratio, there appeared to be no linkage between these genes.

A further study by Reddy *et al.*, (1992) also found resistance in QL-3 was dominant over susceptibility; a two-locus model with independent segregation and a combination of complementary and inhibitory interallelic interaction explained the inheritance pattern they observed. However, most resistance in sorghum is presumed to be monogenic, some possibly oligogenic, which implies a threat of resistance breaking down (Craig and Odvody, 1992).

The inheritance and mechanisms of resistance remain poorly understood and further work is needed to characterize these aspects of resistance in maize.

Molecular marker analysis for resistance to Sorghum Downy Mildew

Progress made in mapping agriculturally important genes with molecular markers forms the foundation for marker-aided selection (MAS). The use of MAS can expedite such difficult screening procedures such as the test for disease resistance. However when several resistance genes are initially present in a donor parent, some of them may be lost during the breeding programs. The chance of losing resistance genes can be reduced if they are detected early (Agrama *et al.*, 1999). The mapping of quantitative trait loci (QTLs) makes feasible the detection, location and characterization of

genetic factors contributing to the variation of polygenically inherited traits (Young, 1996).

The QTL mapping analysis for SDM resistance in Egyptian maize germplasm (Agrama *et al.*, 1999) using single-factor analysis revealed three QTLs on two chromosomes (Chromosome 1 and 9) cumulatively explaining 53.6 per cent of the phenotypic variance. All three QTLs were contributed by the resistant parent G62 and have an additive gene action. Agrama *et al.*, (2002) applied a combination of AFLP (Amplified Fragment Length Polymorphism) technique with bulked segregant analysis (BSA) to map the genes involved in the resistance to SDM in a recombinant inbred population.

Under the Asian Maize Biotechnology Network (AMBIONET) facilitated by CIMMYT (International maize and wheat Improvement Center), Mexico, the Indian team in collaboration with partners in Indonesia, Philippines and Thailand has carried out a study on QTL mapping of resistance against diverse DMs in the Asian region, including SDM (George *et al.*, 2003). The study, utilizing recombinant inbred lines (RILs) derived from Ki3 (resistant) and CML139 (susceptible) led to the identification of five QTLs with significant effects on resistance to the five important DM diseases in Asia. Most significantly, a QTL on chromosome 6 at bin 6.05 was found to confer resistance to all five DM pathogens studied. Nair *et al.*, (2001 and 2005) identified SDM resistance loci on maize Chromosomes 2, 3 and 6 in the Indian maize line NAI116 and verified that the locus on chromosome 6 also contributed resistance to diverse downy mildews.

Sabry *et al.*, (2006) identified three QTLs appeared to have additive effects on resistance, identifying one major gene on

chromosome 2 and two minor genes on chromosome 3 and 9 that contributed to downy mildew resistance. Identification of simple and accurately scored molecular markers for genes that contribute to downy mildew resistance of maize could greatly benefit future efforts to prevent disease losses.

Maize is mainly utilized for direct human consumption in developing countries and for livestock feed in developed countries. However, in recent years its utilization for diversified value-added products has made it an important crop. Corn probably yields more industrial products than any other cereal crops. Sorghum downy mildew (SDM) of maize caused by *Peronosclerospora sorghi* is a disease of great destructive potential because systemically infected plants seldom produce an ear. Breeding resistant varieties is one of the effective and cheap methods to control this disease. Genetic information relating to host resistance would provide more relevant basis for making breeding decisions. In maize, polygenic inheritance is observed for reaction against SDM infection. However, the inheritance and mechanism of resistance in maize are to be studied further in detail.

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