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USE OF IPM IN THE CONTROL OF MULTIPLE DISEASES IN MAIZE: STRATEGIES FOR SELECTION OF HOST RESISTANCE

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ABSTRACT

Maize *(Zea mays)* production in sub-Saharan Africa is constantly threatened by the potential outbreak of multiple foliar diseases such as maize streak, northern leaf blight, gray leaf spot, and rust. Improvement of host resistance to these diseases can provide an important component of integrated pest management (IPM). In this paper, conventional and molecular marker-assisted breeding approaches are reviewed and strategies for improvement of host resistance are presented. Pyramiding of quantitative resistance factors using molecular breeding techniques will be facilitated through cooperative research efforts and adoption of appropriate experimental designs.

Key Words: *Cercospora zeae-maydis, Exserohilum turcicum,* gene pyramiding, molecular breeding, resistance breeding, *Setosphaeria turcica*

RÉSUMÉ

La production du maïs *(Zea mays)* en Afrique au sud du sahara est constamment menacée par l'apparution de multiple infections des feuilles par le streak, le mildiou, le brunissement des feuilles et moisissures. L'augmentation de la résistance a cette maladie peut apporter une importante composante de gestion intégrée de la peste (IPM). Dans cet article, l'approche de croisement assiste des marqueurs conventionels et moléculaires sont présentés et les stratégies d'amélioration de la résistance de la plante hote sont présentées. Les facteurs de résistances pyramidiques et quantitatives utilisant la technique de croisement moléculaires seront facilités à travers les efforts de recherches coopératives et l'adoption d'une approache expérimentale appropriée.

Mots Clés: *Cercospora zeae-maydis, Exserohilum turcicum*, gène pyramidique, croisement moléculaire, croisement résistant, *Setosphaeria turcica*

INTRODUCTION

Demand for maize *(Zea mays)* in sub-Saharan Africa is projected to increase nearly twofold levels by the year 2020 (Rosegrant *et al.*, 2001). In addition to strong demand for maize as a staple, it also has the potential to become an increasingly important non-traditional agricultural export crop (NTAE). The vast majority of maize in Africa is produced by resource limited small-scale farmers. Crops in these diverse cropping systems face many biotic and abiotic threats. Maize production is already spreading into marginal areas so expanding the area of cultivation will likely engender increased risk and minimal increases in production. Fulfilling the growing need for

increased and more dependable maize harvests in Africa will depend as much on preventing yield losses as on maximizing yield potential through additional resource inputs.

Widespread or regionally occurring diseases may threaten tropical maize. Primary diseases include seed rots and seedling blights, root and stalk rots, foliar diseases and ear rots (Paliwal, 2000). Simmonds and Smartt (1999), commenting on the overall severity of attack and economic loss to pathogens, suggested diseases incited by air-borne fungi probably account for the greatest crop losses. A summary of various reports estimating the severity and distribution of disease constraints in Africa (DeVries and Toenniessen, 2001) shows the extreme impact of maize streak virus (MSV) in lowland tropical, and of northern (or *E. turcicum*) leaf blight (NLB) in highland, agroecologies. Research conducted in the Republic of South Africa (RSA) has demonstrated yield reductions of 30 to 60% attributable to gray leaf spot (GLS), depending on the hybrid and environmental conditions (Ward *et al.*, 1997). Recent surveys conducted in Uganda (Bigirwa *et al.*, 2001) and Kenya [KARI maize breeding team, as cited by DeVries and Toenniessen (2001)] documented the wide distribution of persistent foliar diseases: chiefly, GLS, MSV and NLB in both the mid-altitude and highland regions of eastern Africa.

Numerous options have been recommended for the control and management of maize diseases. The availability, feasibility, and cost-effectiveness of each method will differ among production regions and settings (i.e., commercial vs. subsistence). Recommended practices for the control of fungal diseases include conventional tillage that buries crop residues, crop rotation, and fungicide application. Effective management of diseases such as NLB and GLS requires management tactics that focus on protection by limiting sources of primary inoculum through crop rotation and residue management, and by reducing the rate of disease development. Planting of resistant cultivars can effectively reduce the rate of disease development, and that practice is now widely recommended (Ward *et al.*, 1997). Management of MSV relies on inter-cropping, crop rotation, pesticide application, and the planting of resistant cultivars. Agronomic practices impact the population dynamics of the insect vector, whereas resistance breeding has focused on resistance to infection by the pathogen. Disease resistant germplasm is available, but its use is not yet widespread among small-scale farmers. Development of germplasm with good agronomic characteristics and resistance to multiple foliar diseases is particularly challenging, and this goal has been identified as one of the top priorities for research and development of maize in sub-Saharan Africa (DeVries and Toenniessen, 2001).

With limited options for crop protection in many sub-Saharan African farming systems, the challenge will be to increase the productivity and sustainability of maize and maize farming systems while concomitantly protecting the natural resources on which future productivity depends. Thus, IPM strategies, including host resistance, can serve a vital role in fulfilling these objectives. Effective management of diseases will be greatly strengthened through planting maize cultivars with durable host resistance that minimises yield losses.

IMPROVING HOST RESISTANCE

Conventional breeding. Breeding for resistance is a practical, cost-effective strategy for management of diseases (Fehr, 1987). On-going efforts to breed for resistance in sub-Saharan Africa primarily have been undertaken by national programs, private sector, and international agricultural research centers. Maize breeding programs at the International Institute for Tropical Agriculture (IITA) and the Centro Internacional de Mehoriamento de Mais y Trigo (CIMMYT, the International Center for the Improvement of Maize and Wheat) have resulted in the improvement of numerous populations and inbred lines (Kim *et al.*, 1989; Ceballos *et al.*, 1991). Much progress has been made in the development of genetic resistance for many maize diseases: however, large areas are still planted with farmers' varieties that lack resistance to economic pathogens (Paliwal, 2000; Renfro, 1985). Morris (1998) estimated 37% of farmers in Africa regularly plant unimproved varieties. Farmer interviews in three agroecological zones of Uganda revealed that slightly over half the small-scale fields were planted with hybrids or improved

open-pollinated varieties in 1998 (Bigirwa *et al.*, 2001). Limitations to infrastructure, most notably in the seed sector, may inhibit adoption of hybrid maize in some areas. Nonetheless, improved open-pollinated varieties can also make an impact on improving food security. DeVries and Toenniessen (2001) summarized several studies conducted in southern Africa and Ghana that showed improved open-pollinated varieties out-yielded local varieties by 13 to 34%.

In most maize breeding programs, disease resistance is an essential trait for improvement, but resistance must be improved while retaining many other desirable traits as well. The development of new germplasm must address the need for resistance and adaptation across a variety of geographic regions and agro-climatological zones (broad adaptation) or within more narrowly defined agroecosytems (specific adaptation). A specifically targeted breeding program may achieve a measure of success in spite of limited resources and technological inputs, as long as continuity can be maintained. If the need to address the requirements of diverse production environments arises, additional resources and technologies will be needed to assure success. With additional inputs, and through cooperative efforts, breeding programs can be strengthened to address challenging goals. The continued support of national crop improvement programs for essential food crops is a critical component of ensuring food security in sub-Saharan Africa.

Quantitative trait locus discovery and marker assisted selection. Considerable research has been undertaken in genetic mapping of quantitative trait loci (QTL) to assist the development of varieties with improved host resistance. Genetic mapping can detect QTL in discrete regions of plant genomes (Stuber *et al.*, 1987; Paterson *et al.*, 1998). Single-gene resistance is more easily mapped and utilized, but resistance to many important pathogens is polygenic. Hypothetically, resistance controlled by multiple genes should be effective and durable, but resistance of this type is also more difficult to manipulate due to the unknown effects of the environment and the barriers to combining many loci into one genotype.

Resistance QTL associated with NLB, GLS, and MSV have been mapped in maize, thus presenting molecular marker assisted selection (MAS) as a potentially viable strategy to improve resistance to these diseases. Much progress through research has been accomplished, and there are challenges that remain for achieving effective resistance to multiple foliar pathogens. Given past efforts, MAS can play an important role in improving host resistance.

Maize steak. The incidence of maize streak is estimated at 60% across all African agroecosystems where maize is grown (DeVries and Toenniessen, 2001). The disease is caused by a geminivirus that is transmitted by viruliferous leafhoppers of the genus *Cicadulina.* Its effects on grain yield are severe when infection occurs at an early stage of plant growth. Research and breeding efforts, targeted to control MSV have been challenged by problems with insect rearing and inoculation, and concerns about the potential influence of variation among MSV strains (Pixley, 1994).

Reports have ascribed MSV resistance to a single gene lacking dominance (Storey and Howland, 1967), simple inheritance with an apparently strong dominance component (Fourie and Piennar, 1983), five dominant genes (Engelbrecht, 1975) or quantitative inheritance (Rose 1938; Gorter, 1959). The MSV resistance in inbred TZ-Y was considered to be simply inherited (Soto *et al.*, 1982), but Kim *et al.* (1989) reported that resistance in inbred IB32 (Ibadan 32 – developed from TZ-Y) was quantitatively inherited through additive action of up to several genes. More recent studies of genetic control of MSV resistance in population CVR3-C3 have shown that multiple genetic systems for resistance to MSV may indeed exist (Rodier *et al.*, 1995). Both unimodal and bi-modal frequency distributions of symptom ratings were observed when progeny developed by self-pollination within resistant, partially inbred lines were inoculated with MSV. This result suggested the possible existence of two different systems for genetic control of resistance; one with major genes controlling complete resistance, the other with minor genes controlling partial resistance.

Several research groups in cooperation with CIMMYT-Harare have performed QTL mapping of resistance to MSV. Kyetere *et al.* (1995, 1999)

designated a resistance locus (*msv1*) on the short arm of chromosome 1 (1S - bin 1.04) for the resistance it confers to MSV. The locus was discovered in a population of recombinant inbred (RI) lines using restriction fragment length polymorphism (RFLP) probes. The RI population was developed at the University of Hawaii from a cross between susceptible inbred Hi34 and Tzi4, an MSV resistant inbred developed at IITA. Studies by Welz *et al.* (1998) also found a QTL for resistance on chromosome 1S (bin 1.05) in a population of F_2 derived lines genotyped using RFLPs. The population was derived by crossing CML202, an MSV resistant inbred developed at CIMMYT-Harare, and Lo951, a susceptible inbred. Pernet and co-workers (1999a and 1999b) identified a major QTL in the same genomic location on chromosome 1S, and proposed MSV resistance was under the control of two genetic systems, one arising from a major gene on the short arm of chromosome 1 with dominance effects (*msv1*), and with other minor genes on chromosomes 2, 3 and 10, that confer quantitative resistance. Based on the consistent results of these studies, we conclude different genotypes contain what is very likely to be the same resistance factor, *msv1*, that accounts for over half of the phenotypic variance associated with resistance. Clearly, *msv1* is a highly suitable candidate for conversion of susceptible germplasm to resistance using MAS. It is also important to note that total reliance on *msv1* in not envisioned, and MAS should not exclude cycles of phenotypic selection. Combined selection approaches will enhance the probability that minor resistance factors also will be retained, thus favoring durability of resistance genes.

Gray leaf spot. Gray leaf spot has increased in incidence in many commercial production regions over the past two decades, due primarily to an increase in conservation tillage practices (Ward *et al.*, 1999) and in sub-Saharan Africa small-scale systems because of widespread maize cultivation and favorable agroecological conditions (Bigirwa *et al.*, 2001). A survey of maize production and disease severity revealed GLS is endemic throughout diverse agroecological zones of Uganda (Bigirwa *et al.*, 2001). In light of this, the most recent emphasis in our cooperative IPM CRSP research has been the discovery of resistance QTL associated with GLS.

Genetic mapping experiments have identified QTL for resistance to *C. zeae-maydis* (Bubeck *et al.*, 1993; Saghai-Maroof *et al.*, 1996; Clements *et al.*, 2000; Lehmensiek *et al.*, 2001; Gordon *et al.*, 2003, unpubl.). In contrast to the consistent identification of *msv1* (bin 1.04-1.06) identified in all MSV mapping experiments, studies to map loci responsible for resistance to *C. zeae-maydis* have reported resistance loci on all ten chromosomes (Gordon *et al.*, 2003). It is not surprising that researchers identified different genomic regions responsible for *C. zeae-maydis* resistance since the studies utilized different sources of resistance, employed different population structures, generations, disease assessments, and statistical analysis procedures. The studies also were conducted in different environments using either artificial or natural inoculation. All of these factors could have contributed to differences in results because the interactions of genotypes expressing quantitative resistance with environmental conditions are unknown.

The previous studies collectively utilized five resistant inbreds, and a resistance QTL on chromosome 1S (bin 1.05-1.06) is the nearest to a consensus QTL, with three of the five inbreds contributing resistance at this locus. It is interesting to note that QTL is in the same region identified for resistance to MSV. The MSV resistant inbreds CML202 and Tzi04 are known to display intermediate levels of resistance to GLS, but any possible relationship between resistance factors on 1S would be purely speculative at this time.

The results of our studies, using resistance derived from inbred line VO613Y from the RSA, have mapped *C. zeae-maydis* resistance QTLs to the long arms of chromosomes 2 and 4 via selective genotyping of $F_{3:4}$ lines (Gordon *et al.*, 2003). Disease assessments were conducted across several environments (year/location combinations) in the US and RSA. These loci explained approximately half of the phenotypic variation and displayed additive (chromosome 4) and recessive (chromosome 2) gene actions.

Since most sources of resistance to *C. zeae-maydis* are considered to be quantitative, and no races of the pathogen are known at this time, the

only difference among isolates is considered to be aggressiveness (Bair and Ayers, 1986; Dunkle and Carson, 1998; Carson *et al.*, 2002). Based on current knowledge, it is likely that resistance to this pathogen will remain durable (Brown, 1995). We believe that considerable potential exists for transfer of resistance QTL identified by Gordon *et al.* (2003) to elite inbreds such as CML202 through the application of MAS and alternating cycles of phenotypic selection.

Northern corn leaf blight. Resistance to northern leaf blight (NLB) of maize, incited by *Exserohilum turcicum*, may be inherited monogenically (Hooker, 1961, 1963; Hilu and Hooker, 1964) or polygenically (Leonard, 1993, Pataky *et al.*, 1986). Five dominant genes, *Ht*, *Ht2*, *Ht3*, *Htm1* and *Htn1*, control resistance to specific races of *E. turcicum* (Lipps *et al.*, 1997; Welz and Geiger, 2000). Sporulation is suppressed or delayed in plants carrying these genes singly or in combination. Minimum estimates of resistance genes associated with partial resistance ranged from 3 to 6 (Hughes and Hooker, 1971; Jenkins and Robert (1961). Brewster *et al.* (1992) studied the genetics of components of resistance to NLB and concluded that incubation period and lesion number appeared most important in expression of partial resistance. Races of *E. turcicum* capable of overcoming *Ht* genes have been discovered (Smith and Kinsey, 1993; Welz and Geiger, 2000), highlighting the need for deployment of quantitative resistance, alone or in combination with *Ht* genes, to manage NLB. In addition, qualitative resistance genes have shown climatic sensitivity, especially in the tropics (Welz and Geiger, 2000).

Several groups mapped QTL responsible for quantitative resistance to *E. turcicum* (Welz *et al.*, 1998; Freymark *et al.*, 1993; Schechert *et al.*, 1999; Dingerdissen *et al.*, 1996). The consistency of results among these studies was intermediate between the MSV and GLS studies. In addition to the differences in experimental design inherent to all QTL experiments, this may have been due to the fact that both qualitative (major gene) and quantitative (polygene) modes of resistance to *E. turcicum* have been identified (Raymundo and Hooker, 1982).

Freymark *et al.*, (1993) utilized Mo17, an inbred with moderate levels of partial resistance, and B52, a highly susceptible inbred, in their mapping study. A population of $F_{2:3}$ lines was genotyped and resistance QTL on chromosomes 1, 3, 5, 7 and 8 that were common across two environments were found. Dingerdissen *et al.* (1996) utilized the same Mo17 x B52 population and genetic linkage map developed by Freymark *et al.* (1993) to evaluate *E. turcicum* resistance in replicated field trials in Kenya. QTL on chromosomes 3, 5, 7 and 8 for disease severity were significant across environments. Schechert *et al.* (1998) employed a more complete source of resistance, CML202, and found resistance QTL on chromosomes 3, 4, 5, 8 and 9. Other QTL were also detected, some contributed by the susceptible parent, but none was significant across all environments. The consensus QTL on chromosomes 3 (bin 3.06/07), 5 (bin 5.04), and 8 (bin 8.05/06) should be suitable candidates for MAS.

A collaborative strategy for pyramiding resistance to multiple foliar pathogens. We have presented a large amount of information concerning resistance QTL that could be exploited to improve resistance through gene pyramiding. MAS could accelerate development of genotypes with broader and more durable resistance to foliar pathogens. Genetic engineering approaches might also be proposed, but it is not within the scope of this paper to offer review of that subject area.

An objective of a MAS program might be to pyramid resistance loci for NLB and MSV in maize inbred CML202 with those for GLS from a prospective resistance donor such as VO613Y (Gordon *et al.*, 2003). Development of a genotype with resistance to all three diseases would necessitate the assemblage of the six most promising resistance loci (MSV, 1; GLS, 2; NLB, 3) in a desirable genetic background. Genotypic data would need to be obtained in a laboratory equipped for molecular marker analysis and supporting field studies would be required. Ensuring adequate statistical power relative to the costs for obtaining phenotypic and genotypic information on the experimental units (e.g., progeny lines) should be considered carefully.

For example, the probability of recovering a plant in a segregating F_2 population with all six loci in the homozygous condition (at the 0.95 level of probability) would require a population size of over 12,000! A rule of thumb to calculate the necessary population size is 3 x (1/p) where p = the probability of the rare desired event (Simmonds and Smartt, 1999). For a more detailed elaboration on calculation of population sizes necessary for recovery of a trait, one may wish to consult Sedcole (1997). If MAS could be performed in a cost effective manner, then pre-screening large populations could greatly reduce the number of plants that would have to be examined in the field. Less costly marker systems are increasingly available, but it is wise to remember that published demonstrations of successful MAS programs, are rare.

Tests of agronomic performance and verification of disease reactions of selected progenies may encounter additional challenges. The presence of multiple diseases at a particular location may make estimates of the host's true reaction to one disease difficult. In addition, variable host responses to multiple pathogens, due to possible synergism or competition among pathogens (Okori *et al*, 1997), may influence reproducibility of QTL detection. For this reason, we feel all efforts should be made to ensure high levels of disease in nurseries and multiple sites be devoted to evaluation of each disease individually. Phenotypic classification of host responses should be performed at all sites using a well-defined disease assessment scale that relates to the overall damage to the plant. Because the heritability of the resistance traits is considered to be moderate, it also would be advisable to utilize replicated plots whenever possible. It can be readily seen that screening of large populations in replicated trials for multiple diseases over locations, combined with the laborious nature of disease severity assessments and inoculation protocols, can quickly exceed the logistical capacity of most programs.

A logical approach to enhance success of such an endeavor would be to develop a collaborative program that maximizes experimental rigor with optimal allocation of resources, while still retaining sufficient flexibility to accommodate the needs of individual collaborators. Replication of entries at individual locations will be hampered by 1) space requirements of the large number of progenies 2) the need to extend the trials to several sites 3) limited seed supplies that can be obtained from, for example, single $F_{2:3}$ ears. This limitation could be addressed by alternative plot designs utilizing incomplete block and augmented designs. Federer *et al.* (2001) recently published methods for combining results from augmented designs over sites. It would seem prudent to designate each location as a primary experimental site for assessment of one disease, and make optimal expression of that disease a priority.

The power of QTL mapping and MAS can be increased by using large population sizes, replication, and multiple environments – factors addressed earlier. A critical constraint arises when one wishes to genotype many lines, because of the needed laboratory resources and costs involved. Selective genotyping (genotyping only individuals that occur within the extremes or "tails" of the phenotypic trait distribution) can reduce costs of genotyping and focuses on the most informative genotypes (Lander and Botstein, 1989). Selective genotyping is most useful for mapping single traits because it enables the researcher to discard less informative intermediate progeny lines, thus dramatically reducing the resources needed for genotyping. A problem arises when one wishes to use selective genotyping for multiple traits because as the number of traits increases, the power of selective genotyping decreases due to the need to genotype an increasing number of lines. Even if it is necessary to map several traits, a savings could still be realized. It should also be noted that when stringent selective genotyping is practiced (e.g., 10% of population at extremes), the procedure is most effective when a large initial population has been phenotyped prior to marker genotyping (Darvasi and Soller, 1992). Detection of QTL tends to be enhanced by selective genotyping, but researchers should also be aware that bias might be introduced into the estimation of QTL effects under certain conditions (Ronin *et al.*, 1998).

Methods available for analysis of data from QTL mapping studies include single factor analysis (Haley and Knott, 1992), interval analysis (Lander and Botstein, 1989), composite interval analysis (Zeng, 1993) and various others depending upon

the experimental design one chooses. Composite interval analysis can be especially useful when investigating the effects of quantitative and qualitative loci in the same genotype since this procedure allows one to account for major QTLs (like a qualitative locus) and detect other QTL whose effects may be masked by a major locus (Van Ooijen *et al.*, 2002).

It has been proposed that multi-disciplinary approaches toward understanding host-resistance will be the most effective strategy leading to more effective management of foliar diseases (Pratt *et al.*, 1997). Clearly, MAS does not provide a "silver bullet" but it can provide a cost-effective strategy for many types of projects (Dreher *et al.*, 2000). Through cooperative research we hope to enhance our ability to improve host-resistance of germplasm in a more gainful manner.

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