MAPPING QTLS AND DETERMINING RELATIONSHIPS AMONG RESISTANCES TO MULTIPLE FOLIAR PATHOGENS OF MAIZE (*Zea mays* L.)

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Affectionately

Dedicated to

My beloved Parents, My

Sister and

My guide

DEPARTMENT OF GENETICS AND PLANT BREEDING
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CERTIFICATE

This is to certify that the thesis entitled "**MAPPING QTLS AND DETERMINING RELATIONSHIPS AMONG RESISTANCES TO MULTIPLE FOLIAR PATHOGENS OF MAIZE (*Zea mays* L.)**" submitted by Mr. RANGANATHA, H. M., **ID No. PALB 1062,** in partial fulfillment of the requirements for the award of degree of **DOCTOR OF PHILOSOPHY (AGRICULTURE)** in GENETICS AND PLANT BREEDING to the University of Agricultural Sciences, Bangalore, is a record of research work done by him during the period of his study in this university under my guidance and supervision, and that no part of the thesis has been submitted for the award of any degree, diploma, associateship, fellowship or other similar titles.

Bangalore

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With regardful memories . . . . . .

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Mapping QTLs and Determining Relationships among Resistances to Multiple Foliar Pathogens of Maize (*Zea mays* L.)

ABSTRACT

Foliar diseases of Maize *viz.*, northern corn leaf blight (NCLB), sorghum downy mildew (SDM) and southern corn rust (SCR) are the most persistent and destructive biotic constraints in India and worldwide. An investigation was carried out to unravel the genetics of resistance to NCLB and SCR through six generation mean analysis in two crosses of maize *viz.*, CM212 × SKV50 and HKI162 × SKV50 during *Kharif* 2013 and to identify QTLs conferring resistance to foliar diseasesand co-localization of QTLs for resistance to three foliar diseases using194 polymorphic SNPs in 344 $F_{2:3}$progenies derived from the cross CML153 (susceptible) × SKV50 (resistant)during *Kharif* 2012 and *Kharif* 2013 at Mandya.Generation mean analysis revealed significance of additive, dominance and additive × additive gene effects, duplicate gene interaction and partial dominance in the inheritance of NCLB and SCR in both the crosses.Using GMendel 2.0 linkage analysis computer program, 194 polymorphic markers were assigned to ten chromosomes of maizewith thresholdLOD of 3 spanning 2143.02cM with average distance interval length between markers of 10.77 cM.A total of nineteen QTLswere detected for resistance to NCLB, SDM and SCR using composite interval mapping. Two QTLs on chromosome bins 5.04-5.05 and 8.06 explained the total phenotypic variation of 26.58%for NCLB resistance; three QTLs on bin locations 3.04 and 8.06 collectively explained 44.13% variation for SDM resistance and a major QTL on bin location 10.03 with phenotypic variation of 18.59% was detectedfor SCR resistance. Significant pairwise association observed between NCLB, SDM and SCR indicated the presence of multiple disease resistant loci in the $F_{2:3}$population studied. Co-localization of QTLs for resistance to three foliar diseases *viz.,* NCLB (one QTL), SDM (two QTLs) and SCR (two QTLs) were located on bin location 8.03 with common adjacent marker MZA2487-6 which can be used to transfer multiple resistance alleles to susceptible lines through Marker Assisted Selection.

Signature of the Student Signature of the Major Advisor

CONTENTS

LIST OF TABLES

LIST OF FIGURES

LIST OF PLATES

LIST OF APPENDICES

Introduction

I. INTRODUCTION

Maize (*Zea mays* L.) is one of the most important food crops worldwide along with rice and wheat, serving as staple food, livestock feed, and industrial raw material (Troyer, 2006). Maize diversified first in the highlands of Mexico where it was domesticated from the wild progenitor teosinte, *Zea mays* spp. parviglumis (Matsuoka *et al.*, 2002). Globally maize (*Zea mays* L.) is grown under diversity of environments unmatched by any other crop. It is grown from 58°N to 40°S, below average sea level (ASL) to altitudes higher than 3000m and in areas with 250 mm to more than 5000 mm of rainfall per year (Dowswell *et al.*, 1996). Most of the crop is however, grown in the warmer parts of temperate regions and in humid-subtropical climate and the greatest production is in areas having the warmest month isotherms from 21° C to 27° C and a frost-free season of 120 to 180 days duration.

The world area under maize crop is about 177.38 million hectares with the production of 872 million tonnes and productivity is 4.92 t/ha (FAOSTAT, 2013). About 70% of the world maize production area is found in developing countries contributing only 47% of the world's maize production (FAOSTAT, 2013). Asia and Africa together account for about 54% of total maize area, but contribute only about 38% of the total maize production of 883 million tonnes (FAOSTAT, 2013). In Asia, the seven major maize producing countries are China, India, Indonesia, Nepal, Philippines, Thailand and Vietnam together known as "Asia-7", which have progressed from being net importers of maize in the mid-1990s to net exporters in the mid-2000s (FAO and USDA, as cited by Gerpacio and Pingali, 2007; Wada *et al.*, 2008). The average annual growth of maize in the Asia-7 is 3% which exceeded the 2% growth rate in the USA

during 1960s to mid-2000s (Edgerton, 2009; Gerpacio and Pingali, 2007; Phillips 2009).

In India, the crop occupies an area of 8.67 million hectares with the production of 22.50 million tonnes and the average productivity of 25.66 q/ha during 2012-2013 (Anon., 2013). India being the second largest maize growing country in Asia-7, offers innumerable challenges and opportunities to increase regional maize production (Prasanna *et al.*, 2010). Maize is cultivated in the states of Gujarat, Bihar, Andhra Pradesh, Madhya Pradesh, Rajasthan, Chhattisgarh, Karnataka, Maharashtra, Tamil Nadu and Uttar Pradesh. It is also widely believed that, in the very near future maize may become a staple food for human consumption in India, if the demand for rice and wheat is not fulfilled through increased production. Some estimates indicate that we may have to produce 20 million tonnes of maize to meet our requirement for human consumption, poultry, piggery, pharma industry and fodder by 2020.

In Karnataka, before 1960, the area under maize cultivation was only 11,000 hectares with the production of 12,000 tonnes and the average productivity of 12 q/ha. During 2012-2013, a total of 13.22 lakh hectares was covered by maize with the production of 34.55 lakh tonnes and productivity of 28.34 q/ha, which is highest when compared to other states in the country (Anon., 2013). The major maize growing districts of the state are Davanagere, Haveri, Belgaum, Bagalkot, Shimoga, Bangalore Rural, Bellary, Bijapur, Chamarajnagar, Chitradurga, Gulbarga, Dharwad, Gadag, Kolar and Mysore. Area under maize is increasing rapidly in the state because of congenial environment, high yield and ease with which the crop is cultivated. Thus, there is scope to increase maize productivity in Karnataka to a global level of 49.20 q/ha (FAOSTAT, 2013).

Foliar diseases of maize are arguably the primary biotic constraints to maize yields worldwide and the prevalence of these foliar diseases varies depending on the region or season (Smith, 1999). In India, about 61 diseases have been reported to affect the crop (Payak *et al.*, 1973 and Payak and Sharma, 1985). Pratt and Gordon (2006) recently reviewed a number of most important foliar diseases affecting maize production in both tropical and temperate environments. Among various foliar diseases, northern corn leaf blight incited by *Exserohilum turcicum* (Pass) Leonard and Suggs (Teliomorph = *Setosphaeria turcica* (Luttrell), sorghum downy mildew incited by *Peronosclerospora sorghi* (Westen and Uppal) and southern corn rust caused by *Puccinia polysora* (Underwood) are regarded as the most persistent and destructive diseases of field maize. Other maize foliar diseases that can cause significant grain loss include southern corn leaf blight (*Bipolaris maydis*), common rust (*Puccinia sorghi*), Stewart's bacterial wilt (*Erwinia stewartii*) and a number of viral diseases.

Northern corn leaf blight incited by *Exserohilum turcicum* (Pass) Leonard and Suggs is a serious threat to maize cultivation worldwide, reportedly causing yield losses of more than 50 *per cent* (Raymundo and Hooker 1981; Perkins and Pederson 1987). Disease occurs throughout maize producing regions wherever moderate temperatures and high humidity prevail (Carson, 1999; Smith, 1999). Northern corn leaf blight disease was first reported in India by Butler (1907) and is the most important foliar disease causing severe reduction in grain and fodder yield to the tune of 16 -98% (Kachapur and Hegde, 1988). The disease is prevalent in Andhra Pradesh, Karnataka, Bihar, Himachal Pradesh and Maharashtra. Northern corn leaf blight is considered to be one of the most devastating foliar disease in Karnataka resulting in reduction of grain yield of maize by 28 to 91 *per cent* (Pandurangegowda *et al.*, 1991 and Harlapur *et al.*, 2000).

Downy mildews are the most important foliar diseases among the various diseases in South and Southeast Asia. Worldwide, downy mildew has emerged as a destructive systemic disease of major economic importance to maize and the percentage of area with reported economic losses to downy mildew is 30 *per cent*, both in tropical lowland maize and in subtropical, mid-altitude, transition zone and highland maize (Jeffers *et al.*, 2000). In the Asian region, downy mildews are considered as the top priority biotic constraint, where yield losses of 50 *per cent* or more are common (Pingali, 2001). Sorghum downy mildew in maize, caused by *Peronosclerospora sorghi* (Westen and Uppal) is one of the most important foliar diseases among the downy mildews prevalent in India. Sorghum downy mildew causes considerable yield losses in several maize growing states, particularly Karnataka, Tamil Nadu and Rajasthan. A survey conducted in Karnataka revealed that the incidence of disease ranged from 10 to 90% and yield loss up to 30 to 40% (Krishnappa *et al.*, 1995).

Southern corn rust incited by *Puccinia polysora* (Underwood) is another major disease of maize in tropical and subtropical regions worldwide causing yield losses of up to 45-50 *per cent* (Futrell, 1975; Rodrigues *et al.* 1980 and Liu and Wang, 1999). In India, southern corn rust was first noticed in 1991 in Byelkuppa of Mysore district and Arabhavi of Dharwad district in Karnataka (Payak, 1992; Payak, 1994). Southern corn rust is considered most emerging disease in severe form with incidence of 45 *per cent* in the North Karnataka (Harlapur *et al.*, 2000) and yield loss of up to 50 to 70 *per cent* (Agarwal *et al.*, 2001). *Puccinia polysora* rust is becoming a major threat to maize crop in recent years, especially in southern Karnataka and adjoining states like Andhra Pradesh and Tamil Nadu (Chandrashekar, 2009).

Developing strategies to successfully manage these three foliar diseases simultaneously presents a formidable challenge and requires caution when extrapolating arguments from one pathosystem to another. Several disease management options have been recommended to reduce the impact of maize foliar diseases including conventional tillage that buries crop residues, crop rotation, fungicide application and planting of resistant hybrids. Among these practices, planting of resistant cultivars can effectively reduce the rate of disease development and is widely recommended (Ward *et al.*, 1997).

Breeding for resistance is a practical, cost-effective means available to manage these diseases (Fehr, 1987). To breed a genotype with high level of resistance to northern corn leaf blight, sorghum downy mildew and southern corn rust, the knowledge of gene action involved in the expression of resistant reaction in the material being handled, is a pre-requisite. The basic inheritance pattern of resistance reaction should be understood in depth to plan for appropriate breeding programme for developing resistant genotypes. Various biometrical approaches have been developed to decipher the genetic architecture and mode of inheritance of different characters related to yield. Generation mean analysis (Hayman, 1958; Jinks and Jones, 1958 and Mather and Jinks, 1971) is one such approach, which elucidates information about nature and magnitude of different gene action *viz.*, additive and dominance with an unambiguous test for epistasis. The information obtained from this analysis is highly reliable as it is based on first order statistics. It also provides information about the type of epistasis *viz.,* additive × additive, additive × dominance and dominance × dominance operating in the inheritance of a character. This information cannot be elicited by diallel or line × tester analysis. The genetic architecture of disease resistance is very important to understand in order to develop better breeding efforts. Earlier studies on the genetics of resistance to northern corn leaf blight

(Jenkins *et al.* 1952, Hughes and Hooker, 1971, Hettiarachchi *et al.*, 2009, Chaudhary and Mani, 2010), sorghum downy mildew (Geetha and Jayaraman, 2002, Yen *et al.*, 2004, Nair *et al.*, 2004a and 2005) and southern corn rust (Paterniani *et al.*, 2000, So *et al.*, 2003, Brewbaker, 2005, Ji, 2006, Brewbaker *et al.*, 2011) suggests that resistance is complex and polygenic in nature.

Resistance to foliar diseases is effectively obtained through conventional breeding, where susceptible genotypes under disease pressure can be eliminated before harvest (Ali and Yan, 2012). However, conventional breeding is time consuming and less feasible due to complex nature of resistance reaction to foliar diseases. This favoured the development of molecular tools to assist the conventional breeding efforts to breed the resistant cultivars. Among these, identification of quantitative trait loci (QTL) makes feasible for the detection, localization and characterization of genetic factors contributing to the variation of polygenically inherited traits, which in turn helps in selection through marker assisted selection (MAS).

In maize, a large amount of valuable information exists with reference to QTLs conditioning resistance to foliar diseases. A number of quantitative trait loci conditioning resistance to northern corn leaf blight (Welz *et al.*, 1999, Welz and Geiger, 2000, Brown *et al.*, 2001, Ping *et al.*, 2007, Asea *et al.*, 2009, Balint-Kurti *et al.*, 2010, Chung *et al.*, 2010, Zwonitzer *et al.*, 2010, Chung *et al.*, 2011, Poland *et al.*, 2011, Schaefer and Bernardo, 2013), sorghum downy mildew (Agrama *et al.*, 1999; George *et al.*, 2003; Nair *et al.*, 2005; Sabry *et al.*, 2006; Phumichai *et al.*, 2012 and Jampatong *et al.*, 2013) and southern corn rust (Jiang *et al.*, 1999, Jines *et al.*, 2007 and Wanlayaporn *et al.*, 2013) have been identified in maize. Multiple disease resistance in which the same locus conditions resistance to multiple pathogens is both practically and

conceptually important for breeding durable resistant genotypes. The information regarding the studies on application of markers linked to QTL for pyramiding quantitative resistance to multiple foliar pathogens in maize is sparse. This ensures an opportunity for identification of QTLs for resistance to multiple foliar pathogens and co-locating the multiple disease resistance QTLs in our breeding material for practicing effective Marker Assisted Breeding programme.

Keeping all these points in view in the present study, an attempt was made to locate the genomic regions or QTLs conferring resistance to three foliar diseases *viz.*, northern corn leaf blight, sorghum downy mildew and southern corn rust and to localize the multiple disease resistant loci for future use in Marker Assisted Selection with the following objectives:

1. Unravelling the genetics of resistance to northern corn leaf blight and southern corn rust.

2. Identification of QTLs for resistance to three foliar pathogens of Maize *viz.*, northern corn leaf blight, sorghum downy mildew and southern corn rust.

3. Determining relationships among resistances to multiple foliar pathogens of Maize.

Review of literature

II. REVIEW OF LITERATURE

In the present investigation, an attempt has been made to map and identify the QTLs conferring resistance to multiple foliar pathogens of maize (*Zea mays* L.). The research carried out in the past on multiple foliar disease resistance, relevant to the present study in maize is reviewed in this chapter.

The review of literature is presented under the following headings.

2.1 Maize crop

2.2 Maize diseases

2.2.1 Northern corn leaf blight (*Exserohilum turcicum*)

2.2.2 Sorghum downy mildew (*Peronosclerospora sorghi*)

2.2.3 Southern corn rust (*Puccinia polysora*)

2.3 Genetic analysis of resistance to multiple foliar diseases

2.4 Molecular markers and their utility in genetic dissection and breeding for resistance

2.5 Mapping quantitative trait loci (QTL) conferring resistance to multiple foliar pathogens in maize

2.6 Co-localization of QTLs conferring resistance to multiple foliar diseases

2.1 Maize crop

The genus *Zea* belongs to the grass family Poaceae and the tribe Maydeae. The tribe Maydeae consists of seven genera: two of American origin, *Zea* and *Tripsacum*; and five of eastern origin which extend from India through Southern Asia to Australia and include *Coix, Sclerachne,*

Polytoca, *Chionachne* and *Trilobachne* (Watson and Dallwitz, 1992). Broadly, the genus *Zea* includes the wild taxa, collectively known as teosinte and domesticated corn or maize (*Zea mays* L. spp. *mays*). Based on the morphological characteristics and geographic delineations, the genus *Zea* has been classified into four species; *diploperennis*, a perennial diploid teosinte, *perennis,* a perennial tetraploid teosinte, *luxurians*, an annual Guatemalan teosinte, and *mays*, a highly polymorphic, diploid annual species (Doebley and Iltis, 1980). Iltis and Benz (2000) classified *Zea luxurians* from Nicaragua as a new species called *Zea nicaraguensis*.

Doebley (1990) recognized four subspecies within *Zea mays*: (i) spp. *huehuetenangensis*, an annual teosinte found in few highlands of northwestern Guatemala; (ii) spp. *mexicana*, an annual teosinte, from highlands of central and northern mexico; (iii) spp. *parviglumis*, an annual teosinte, common in the middle and low elevations of southwestern mexico, and (iv) spp. *mays* or maize probably first domesticated in the Balsa river valley of southern mexico.

2.2 Maize diseases

Diseases of maize (*Zea mays* L.) are the primary biotic constraints to maize yields worldwide. Parasitic diseases caused by bacteria, fungi, viruses and mycoplasma provide severe constraints on tropical maize production frequently causing 30-40% yield loss (Ullstrup, 1977).

Several foliar pathogens infect maize and considerably reduce the yield. According to Effron (1985), maize foliar diseases are categorized as follows:

i. Local-spot foliar diseases: affect leaves primarily and destroy the leaves and result in significant yield reduction. Notable examples are *Puccinia rust* (*Puccinia* spp.), *Helminthosporium* leaf blights

(*Helminthosporium* spp), Curvularia leaf spot (*Curvularia pallenscens*) and brown leaf spot (*Physoderma maydis*). Amongst these, *Helminthosporium turcicum* blight and *Puccinia polysora* rust are most destructive diseases that routinely limit maize productivity in sub-Saharan Africa, India and Corn Belt (Ullstrup, 1950; Hooker, 1969; Melching, 1975; Payak, 1994; Okori *et al.*, 1999; Ward *et al.*, 1999; Bigirwa *et al.*, 2001; DeVries and Toenniessin, 2001; Agarwal *et al.*, 2001 and Chen *et al.*, 2004).

ii. Systemic foliar diseases: these include maize streak and maize mottle viruses and downy mildew. Downy mildews caused by fungus *Peronosclerospora sp*. is a threat to maize production in some countries of tropical Africa including Nigeria, Ivory Coast and Sudan (Effron, 1985), tropical lowlands including India (Frederiksen *et al.*, 1969; Frederiksen and Renfro, 1977; Williams, 1984; Dhillon and Prasanna, 2001).

iii. Stalk and ear rots: fungal stalk rots are incited by *Botriodiploidia* and *Rhizoctonia* spp. are most important in low land rainforest and moist Savanna. All the stalk rot pathogens cause kernel rot, resulting in grain deterioration and loss of quality especially in soft endosperm genotypes (Effron, 1985).

Several foliar diseases cause economic damage to maize, fortunately, the prevalence of these diseases varies depending on the region or season (Smith, 1999). Literature with regard to occurrence, distribution, mode of infection, symptomology and economic importance of the three important maize foliar diseases *viz.*, Northern corn leaf blight caused by *Exserohilum turcicum*, Southern corn rust caused by *Puccinia polysora* and Sorghum downy mildew incited by *Perenosclerospora sorghi* are presented below.

2.2.1 Northern corn leaf blight (*Exserohilum turcicum*)

2.2.1.1 Occurrence and distribution of the disease

Northern corn leaf blight of maize caused by *Helminthosporium turcicum* Pass. was first reported by Passerini (1876) in Perma, Italy. Later Pammel *et al.* (1910) and Drechsler (1923) regarded it to be *Trichometasphaeria turcica* Luttrell (Luttrell, 1958). Further, Leonard and Suggs (1974) renamed it as *Setosphaeria turcica* and described the conidial stage as *Exserohilum turcicum* (Leonard and Suggs, 1974) is strongly protuberant. *Exserohilum turcicum* incidence is higher under conditions of high moisture and temperature within the range of 18^0 C to 27^0 C (Ullstrup, 1970). Northern blight Severity reported in Central-Western, Southwestern and Southern Brazil (Fernandes and Balmer, 1990; Pereira, 1995) while it imposing significant problem in the north eastern United States, sub-Saharan Africa and areas of China, Latin America and India (Adipala *et al.*, 1995 and Dingerdissen *et al.*, 1996).

In India, northern leaf blight was first reported in 1907 from Bihar (Butler, 1907). Later the disease was reported from different parts, *viz.*, Lalmardi, Srinagar (Kaul, 1957), Punjab (Mitra, 1981), Himachal Pradesh (Chenulu and Hora, 1962) and Kashmir valley (Payak and Renfro, 1968). Northern leaf blight severity was seen in mid-altitude tropical regions where high humidity, low temperature, and cloudy weather prevail during the maize growing season (Singh *et al.*, 2004). *Exserohilum turcicum* incidence was prevalent in the plains of India and in the Himalayan region *viz.*, Karnataka, Jammu and Kashmir, Himachal Pradesh, Uttarakhand, Orissa, Andhra Pradesh and North Eastern Hill states.

2.2.1.2 Mode of infection and disease symptoms

Northern corn leaf blight lesions appear first on lower leaves, then protrude towards top and other parts of the plant (Elliott and Jenkins, 1946). *Exserohilum turcicum* pathogen symptoms start as small elliptical spots on the leaves, greyish green in colour and water soaked lesions, then spots turn greenish with age and get bigger in size, finally attaining a spindle shape. Spores of the *Exserohilum turcicum* develop abundantly on both sides of the lesions. Heavily infected field present a scorched appearance (Chenulu and Hora, 1962).

Ullstrup (1966) described the symptoms of the disease in United States. The disease is recognised by long elliptical greyish or tan lesions. When fully expanded, the spots may be 1½" by 6" in size. These lesions appear first on the lower leaves and as the season progresses, the lesion number increases and all the leaves are covered. Severe reduction in yield is noticed when maize is associated with severe necrosis or chlorosis of leaves in the upper two-thirds of the canopy (Fisher *et al.*, 1976; Bowen and Pedersen, 1988).

2.2.1.3 Economic importance

Northern corn leaf blight caused by *Exserohilum turcicum* can cause extensive defoliation before tasseling stage, grain-filling stage, resulting in yield losses of more than fifty *per cent* in several parts of world and India.

Ullstrup (1951) reported yield of susceptible single cross hybrids reduced from 40 to 68 *per cent* and 1.8 to 18 *per cent* in resistant single cross hybrid due to northern corn leaf blight infection. Robert (1953) reported that the disease severity varies from trace to 50 *per cent* depending on the various maize cultivars.

Chenulu and Hora (1962) observed that the loss in grain yield varied from 27.6 to 90.7 *per cent* and green weight of plant from 16.6 to 68.8 *per cent* depending upon the intensity of northern corn leaf blight infection and also indicated that loss in yield is directly proportional to the intensity of infection.

Sharma and Aujla (1968) claimed yield loss of 39.7 *per cent* in India depending on severity of disease while Hirose and Toda (1970) stated that *Exserohilum turcicum* affected the maize ear length, number of kernels, 1000 kernel weight and grain yield.

Raymundo and Hooker (1981) measured the relationship between *Exserohilum turcicum* of maize and yield loss using susceptible, moderately resistant and resistant hybrids and concluded that loss in grain yield ranging from 16.7 to 62.3 *per cent* depending on disease severity levels.

Lipps (1982) reported that the loss in grain yield was as high as 50 *per cent* on susceptible hybrids in mid-western states. Leonard *et al.* (1985) reported that *Exserohilum turcicum* blight was epidemic in North Carolina in USA destroying up to 75 *per cent* of leaf area.

Survey conducted by Pandurangegowda (1987) in southern parts of Karnataka revealed that the severity of *Exserohilum turcicum* leaf blight of maize varied from 10 to 100 *per cent* in all hybrids and composites in the surveyed areas.

Perkins and Pederson (1987) reported significant reduction in grain yield which ranged from 8 to 18 *per cent* due to *Exserohilum turcicum* leaf blight while Rai (1987) reported the loss in grain yield of maize from 27.6 to 90.7 *per cent* and this loss was directly proportional to the intensity of the disease.

Shankaralingam *et al.* (1987) observed 28 to 91 *per cent* loss in grain yield depending on the severity of disease while Kachapur (1988) revealed the loss in grain yield is maximum when disease developed before tasseling stage and the yield losses ranged from 78.44 to 98.97 *per cent* in Northern Karnataka and losses in fodder was 35.26 to 66.86 *per cent* in susceptible cultivars.

Pandurangegowda (1991) observed grain yield loss of 46.70 *per cent* in maize cultivars due to infestation of *Exserohilum turcicum*. Pandurangegowda *et al.* (1993a) assessed grain yield losses due to turcicum leaf blight in two hybrids, Deccan and Ganga 5 by two methods *i.e.,* on the basis of whole rows and on the basis of varying disease levels. They indicated that the method based on the average grain yield at different levels of infection is more reliable and authentic than the other based on whole rows basis and the maximum grain yield loss observed was 45.7 *per cent* on the rating of 5 on a scale of 1 to 5.

Shivankar and Shivankar (2000) reported that total incidence of *Exserohilum turcicum* was 39.23, 35.46, 33.6, 31.42 *per cent* and loss in grain yield was 18.77, 15.45, 13.81 and 10.50 *per cent* on the maize cultivars AMC1, AMC2, Manjari and Kargill 633, respectively under artificial inoculated conditions.

Levy and Leonard (2008) studied the effects of defoliation at specific leaf positions on yield of sweet corn plants and the yield loss caused by infection of *E. turcicum* by approximately 22 *per cent*.

2.2.2 Sorghum downy mildew in maize

2.2.2.1 Occurrence and distribution of the disease

The sorghum downy mildew of maize is widely distributed all over the world and attacks all kinds of maize. The disease was first observed and reported by Butler (1907) in Maharashtra and Tamil Nadu states of

India as *Sclerospora graminicola*. Later, Melechers (1931) reported the downy mildew of maize and sorghum in Egypt. He gave the evidence that it might have entered Egypt through the packing material from India.

2.2.2.2 Mode of infection and disease symptoms

Renfro (1970) gave detailed account of the disease cycle and symptoms. Oospores are formed in or adjacent to vascular bundles in chlorotic area of leaves or ear bracts. The stripes later turn brownish due to the resting of dark spores within. The oospores either fall to the soil or windblown often within the host tissue. They remain viable in soil for five to ten years. Root exudates and other processes in the soil stimulates the growth of coenocytic germ tubes and later infection occurs through the hypocotyl or mesocotyl with upward growth to the shoot apex. Conidia are formed during night in large numbers. Seven to eight hours of dark period coupled with 20-30°C temperature is optimum for the production of conidia in the presence of free water during the last four and a half hours. Conidia are wind borne and short lived and they require about 45 minutes to germinate under favourable conditions and about four to six days are necessary for mycelia to ramify to the shoot apex. Thereafter all subsequently emerging tissues are systematically infected.

Frederiksen *et al.* (1970) reported systematically infected maize plants are chlorotic, stunted and occasionally have stripped leaves. However, Shivakumar and Shankar-Bhat (1982) found that oospores in intervenal areas which appear as narrow, yellowish streaks running all along the infected leaf. Oospores production followed by leaf shedding was observed by them.

Warren *et al.* (1974) described three reasons for increased incidence of sorghum downy mildew. They are (1) monoculture of susceptible corn hybrids, (2) adoption of minimum tillage practices and (3) persistence of oospores in the soil. The pith of all infected plants

showed a mottled brown discolouration; excessive brace root formation extended up to the sixth node; portion of first diseased leaf often narrow at the base while remaining leaves are normal in size and shape showing partial disease symptoms. Plants are typically barren, when an ear did form, few or no seed was produced and the shanks of the infected ears twisted and elongated with 6-8 nodes.

Li (1983) described the mode of infection of the pathogen. He stated that the primary inoculum of the disease comes from oospores of *Perenosclerospora sorghi* in the soil. A few seedlings of maize systematically infected during their emergence in the fields under suitable conditions develop sporangia on the diseased plants, forming foci from which the disease spreads to the whole field. But there are distinct varietal variation in their reaction to the disease.

2.2.2.3 Economic importance

Sorghum downy mildew is particularly destructive because systemic infection of the host generally results in a barren inflorescence. The effect of infection on yield is best illustrated by reference to several reports in the literature. Payak (1975) reported that in parts of India annual yield loss due to sorghum downy mildew was at least 105 tonnes. In Venezuela, crop loss was so severe in the early 1970's that a national emergency was declared (Frederiksen and Renfro 1977). In Israel both forage sorghum and maize were severely infected with incidences of up to 50% (Kenneth 1976), and in the USA incidences of 90% have been reported (Frederiksen *et al.*, 1969). The effect of systemic sorghum downy mildew is now more clearly understood, since models have been developed that show a linear relationship between incidence of systemic SDM and yield loss at normal sowing densities (Frederiksen and Ullstrup, 1975; Tuleen and Frederiksen, 1981).

Renfro (1970) estimated loss due to sorghum downy mildew from different countries in South East Asia which could be as high as 40 to 60 *per cent* in certain parts during this century. In India, significant yield loss in maize is usually seen in late sown areas and in those nearer to infected Kans grass. A survey conducted in Karnataka revealed that the incidence of disease ranged from 10 to 90 *per cent* and yield loss from 30-40 *per cent* (Krishnappa *et al.*, 1995).

2.2.3 Southern corn rust (*Puccinia polysora*)

2.2.3.1 Occurrence and distribution of the disease

The polysora rust or southern corn rust of maize (*Zea mays* L.) was first reported by Underwood in 1897 on eastern gamma grass (*Tripsacum dactyloides* L.) in Alabama which was later observed in maize specimens collected in 1879 (Bailey *et al.*, 1987). *Puccinia polysora* was reported in Florida and New Jersey in 1920, Indiana in 1949-1950, Illinois in 1958, Wisconsin in 1959 and Mississippi river valley (eastern Texas to western Alabama) in 1972-1974 (Hollier, 2010). *Puccinia polysora* fungus occurred and shown enormous destructive potential in Corn Belt in 1949 (Ullstrup, 1950; Hooker, 1969; Melching, 1975; Chen *et al.*, 2004).

In India, southern corn rust was first noticed in 1991 in Byelkuppa of Mysore district and Arabhavi of Dharwad district in Karnataka (Payak, 1992; Payak, 1994). Subsequently, uredinospores of *P. polysora* was reported in October, 1999 in infected samples during post-entry quarantine inspection of maize crop raised from pesticide treated seeds at Bangalore (Agarwal *et al.*, 2001). The incidence of *P. polysora* rust is seen in majority of the cultivars grown in Southern districts of Karnataka *viz.,* Bangalore, Mysore, Mandya, Chamarajnagar, Hassan, parts of Coorg, Kolar, Davanagere, Shimoga, Tumkur and Chitradurga (Anon., 2002).

2.2.3.2 Mode of infection and disease symptoms

Puccinia polysora generally infects the exposed leaf of the plant and the disease becomes progressively more severe as the plant develops. Rust pustules appear on stem and leaf sheaths and are smaller in shape and more circular. Pustules are deep orange red during uredial stage and chocolate brown to black and appear as a circle around uredosori during telial stage. Epidermis remains intact for longer periods giving a slimy appearance. Pustules become so numerous that the leaf and sheath tissues get killed and they turn dark brown as the plants approach maturity and the plants are prematurely desiccated (Pandurangegowda *et al.*, 2005).

2.2.3.3 Economic importance

The effect of polysora rust infection on yield is best illustrated by reference to several reports in the literature. Melching (1975) rated *P. polysora* as the most destructive of the rusts of maize. Southern Corn Rust, since its observation in Alabama in 1891 was regarded as minor pathogen of corn until it was found in the Corn Belt in 1949, 1958 and North Carolina in 1972 and 1973 causing epiphytotics (Futrell, 1975). Polysora rust was quite destructive in Africa, Mexico, Central and South America in the fifties (Woods and Lipscomb, 1956) and in North California in 1972, 1973 and 1974 (Rodrigues *et al.*, 1980).

Southern corn rust is a disease that can cause serious yield loss primarily on late planted corn and the yield loss ranging from 45-50 *per cent* has been described (Futrell, 1975). Losses of up to 50-70% were reported in West Africa (Wood and Lipscomb, 1956, Rodrigues *et al.*, 1980), up to 60-80% in Pennysylvania and Maryland in America (Raid *et al.*, 1988) and 42-53% loss in northern China reaching epiphytotic levels (Liu and Wang, 1999).

Rodrigues *et al.* (1980) assessed the yield losses caused by *Puccinia polysora* ranging from 4.23 to 45 *per cent* in three successive biweekly plantings in 1976 and 1978. Raid *et al.* (1988) reported the loss in grain yield due to *P. polysora* (17.7 to 39.1 %). Several southern rust epidemics occurred in the southern United States during the 1970s (Bailey *et al.*, 1987) and in Brazil (Goday *et al.*, 2003).

In India, significant economic yield losses due to *Puccinia polysora* incidence in maize was assessed by Gupta (1981) of up to 11.2 to 33.6 *per cent* in Diara region in Bihar and Sharma *et al.* (1982) of up to 32.18 *per cent*.

Harlapur *et al.* (2000) reported that polysora rust disease as the most emerging disease in severe form with incidence of 45 *per cent* in the North Karnataka. Agarwal *et al.* (2001) estimated 50 to 70 *per cent* yield loss due to moderate to severe infection of *Puccinia polysora* in Bangalore.

Other than the grain yield loss, the rust infection reduced forage yield and quality. Infected plants may die prematurely from stalk rot and may lodge more (Futrell, 1975; Melching, 1975; Sim, 1980). Severe southern rust outbreaks that occur every 3-4 years cause upto 50% reductions of anticipated yields (Hagan, 2010).

2.3 Genetic analysis of resistance to multiple foliar diseases

Many genetic models given by various workers have been employed to describe the action of genes controlling quantitative characters. Fisher (1918) was the first to attempt for the contribution of a gene model, which includes dominance at a single locus. He also indicated that there may be a deviation from simple additive effects between loci when more than one locus affects a given character. Later, Fisher *et al.* (1932) used the gene model to describe gene action of any numbers of genes on a

given character. Mather (1949) also developed gene models to study and assess the relative importance of additive and dominance gene effects assuming epistatic effects to be negligible.

Anderson and Kempthorne (1954) in their gene model partitioned gene effects into additive, dominance and epistasis. Mather (1949) and Hayman and Mather (1957) proposed a gene model to assess the contribution of gene interaction to continuous variation.

Hayman (1958) and Jinks and Jones (1958) described a general procedure to estimate parameters referring to additive [$\hat{d}$], dominance [$\hat{h}$], additive x additive [$\hat{i}$], additive x dominance [$\hat{j}$] and dominance x dominance [$\hat{l}$] effects, based on the theory developed by Fisher *et al.* (1932).

2.3.1 Northern corn leaf blight (*Exserohilum turcicum*)

2.3.1.1 Nature and mechanisms of resistance

Genetics of resistance to Northern leaf blight is generally classified as; (1) major gene resistance or race specific resistance conferred by several dominant or partially dominant qualitative genes Ht1 (Hooker, 1963), Ht2 (Hooker, 1977), Ht3 (Hooker, 1981), HtN (Gevers, 1975) and HtP (Ogliari *et al.*, 2005). The Ht1, Ht2, and Ht3 genes confer a “chlorotic lesion” type of reaction to the pathogen, whereas the HtN gene results in a delay of symptoms until after anthesis. (2) Quantitative or partial resistance inherited polygenically with additive gene action (Jenkins *et al.* 1952; Hughes and Hooker, 1971). Partial resistance expressed as a reduction in intensity of disease development *via* reducing *per cent* leaf area infected, lesion size, lesion numbers, and lesion expansion rate. Combination of qualitative and quantitative resistance genes are required in breeding for durable resistance to northern leaf blight, with more emphasis on quantitative genes (Pratt and Gordon, 2006).

2.3.1.2 Genetics of resistance to northern corn leaf blight in maize

The most appropriate and economical strategy to manage turcicum leaf blight of maize is through exploitation of host resistance, which is not only environmentally friendly but convenient to adapt at farmers level. Host plant resistance plays a significant role in integrated disease management approach. Thus, identifying resistant genes and genotypes to this important disease and combining them with yield traits is a priority of Indian maize breeding programme (Sharma *et al.*, 2005).

Jenkins and Robert (1952) studied the inheritance of resistance in crosses of maize involving three susceptible and five resistant inbred lines. They concluded that the resistance is under polygenic control and extreme susceptibility of the inbred line R_4 was due to a recessive gene.

Jenkins *et al.* (1952) studied the inheritance of 12 groups of F_3 progenies. The data indicated that the resistance was controlled by many genes but some genes have major effects. Some genes showed their effects only at low severity of disease, others showed their major effects at a high severity of disease and still others showed their effects over a wider range of disease severity.

Jenkins and Robert (1959) evaluated 30 *Exserohilum turcicum* resistant inbred lines, which differed significantly in their blight reaction when these lines were crossed with three susceptible tester lines. They observed significant differences in blight reaction among the three testers and for interaction of lines x testers, both for the F_1 and F_2 progenies. Later in 1961, they observed that resistance was polygenic which expressed primarily in a reduced number of lesions, smaller in size and amount of sporulation compared to those found in susceptible genotypes. They also noted that resistance was partially dominant and controlled by many genes, some of which produced major effects.

Hooker (1963) reported monogenic resistance in corn to *H. turcicum*. He reported two lesions types, (a) greyish green wilted spots that soon elongated into tan necrotic lesions supporting abundant fungus sporulation, and (b) smaller chloratic type, with delayed necrosis and inhibited fungus sporulation.

Hooker (1962, 1963) found a single dominant gene in the dent corn inbred GE 440 and in the popcorn variety Lady's finger to condition the chlorotic lesion type of resistance to *H. turcicum*. Discovery of single dominant gene resistance to northern corn leaf blight has resulted in making breeding easier. This resistance was more complete than multiple gene resistance and resistant plants were easy to identify (Hooker, 1961, 1963a).

Jha and Dhawan (1970) have studied the nature of gene action for resistance to *H. turcicum* through generation mean analysis of all possible combinations of four resistant and four susceptible lines in two locations viz., Bajaura (Kashmir) and Dholi (Bihar) which revealed that (1) the GCA accounted for 94 and 97 *per cent* of the total genetic variance respectively, even though both GCA and SCA were significant at both locations, (2) there were contrasting differences in the reaction of resistant and susceptible lines of *H. turcicum*, (3) the GCA effects were similar for each inbred at both locations, (4) the genetic variability was mostly additive and partial dominance was effective at both locations.

Hirose and Toda (1970) indicated that the resistance is controlled by three to twelve additive genes with high heritability values for resistance. Ullstrup (1970) pointed out that genotypes carrying monogenic resistance may have debilitating effect which is reflected in lower grain yield.

Hughes and Hooker (1971) studied the nature of gene action in maize to *E. turcicum* and concluded that the resistance was conditioned

by relatively less number of genes, primarily additive in effect and breeding for resistance to this disease should be effectively accomplished by phenotypic recurrent selection. Later, Hooker and Kim (1973) reported that greater kernel weights and lower leaf blight scores were associated with the presence of dominant Ht gene conditioning chlorotic lesion resistance.

The progeny from a cross between isolates of *E. turcicum* avirulent or virulent to monogenic Ht1 resistant maize line segregated in a 3:1 ratio indicating that the virulence in the pathogen is monogenic in nature (Lim *et al.*, 1974).

Gevers (1975) reported a new major gene 'Htn' which is reasonably stable compared to other sources of monogenic resistance. He observed that homozygous resistant plants were more resistant than heterozygous ones and those with dominant genes at two loci appeared to be more resistant than plants with dominant genes at a single locus.

Hooker (1977) reported that the gene 'Ht2' conditioned a lower level of resistance than Ht1 but the two genes interact together to condition a higher level of resistance than either gene alone. Ramamurthy *et al.* (1980) in a diallel study involving four *E. turcicum* resistant and susceptible inbreds and their crosses indicated that the gene action was primarily additive with partial dominance with no inter allelic interaction.

Lipps (1982) observed that monogenic resistance was conditioned by the Ht gene and polygenic resistance can be incorporated in a single cross hybrid. He also pointed out that monogenic resistance is easy to incorporate in a single cross maize hybrid and the utilization of polygenic resistance has been limited by the difficulty of transferring multiple genes for resistance into inbred lines.

Patil *et al.* (1982) carried out the genetic analysis of three crosses by utilizing a monogenic and a multigenic resistant source in late and early maturing highly susceptible lines. They observed that the nature of epistasis was duplicate in presence of both monogenic and multigenic resistance. It was complementary in the presence of any one of them with early maturing susceptible lines.

In a study on the inheritance of resistance to *H. maydis* race 'o' in a diallel cross of eleven inbred lines, general and specific combining ability effects were significant for disease score, number and area of regions and spore production per lesion. Additive and non-additive effects were also important and resistance to disease was partly dominant (Zhang *et al.*, 1983).

Renfro (1984) reported that polygenic resistance controls the number of lesions and partially dominant. He noted that additive gene action was more important than dominance or epistatic effects.

In a diallel crosses among eight open cultivars, including parents but excluding reciprocals for resistance to *H. turcicum* tested under natural epiphytotic condition at Kalimpong indicated that resistance was mainly conferred by additive genes (Das and Chaudhuri, 1986).

Li and Liu, (1984) studied three lines differing in their resistance to *H. turcicum*, in to which the Ht gene had been introduced in different combination of recessive and dominant alleles. It was seen that lesion number, lesion size and amount of sporulation differed according to the particular association of Ht alleles with genetic background *i.e.,* in a highly resistant background, the susceptibility conferred by the dominant allele was marked.

Sigulus *et al.* (1988) studied the genetics of polygenic resistance to *E. turcicum* through diallel analysis using four maize inbred lines and

they revealed that the GCA effects were much larger than the SCA effects indicating the importance of additive genes in conferring resistance.

Shankarlingam *et al.* (1989) revealed that the nature of inheritance was not governed by major genes but it was quantitative in nature. The significant differences among parental lines for their SCA was also noted. The GCA component of variance was higher than that of SCA showing the preponderance of additive gene action. All types of gene effects namely additive, dominance and epistasis were operating in the control of resistance but additive gene action and dominance x dominance type of epistasis with duplicate nature were operating in controlling resistance.

Sharma and Payak (1990) studied durable resistance to two leaf blights, *Setasphaeria turcica* and *Cochilobolus heterostropus* in two maize inbred lines, CM 104 and CM 115 for 16 and 14 years, respectively and found that the resistant inbred lines have the potential to transmit this resistance to progenies in hybrid combination that are governed by additive gene action.

Pandurangegowda *et al.* (1993b) found the importance of additive and non-additive gene action for resistance to *E. turcicum* while studying the combining ability and six generation mean analysis using two resistant, two moderately resistant and two susceptible parental lines. The generation mean analysis confirmed that all types of gene effects i.e., additive, dominance and epistasis were operating in one or the other crosses in controlling resistance. Dominance gene action and dominance x dominance type of epistasis were present in most of the crosses and the lines used under this study could be utilized for developing double cross/double top cross hybrids.

Mahajan *et al.* (1995) studied the genetics of resistance to northern leaf blight in maize using eight maize inbred lines which were crossed in

a half diallel method and found that for resistance to disease, non-additive gene action was more important at mid-altitude during both years and was influenced by environment while, additive gene action was important for low and medium altitudes and was less influenced by environment.

Carson (1995) reported on inheritance of latent period length in maize against *Exserohilum turcicum* through generation mean analysis, revealed that over 92 *per cent* of variation among generation means could be explained by additive gene action while dominance and epistatic effects were negligible.

Saindass *et al.* (2000) conducted a study on the gene effect for maydis leaf blight (*Dreschlera maydis*) disease resistance though line x tester analysis under artificial inoculation condition. The combining ability analysis revealed that non-additive gene variances were more important in the expression of disease resistance.

Six generation mean analysis was undertaken to analyse the genetic basis of resistance to turcicum leaf blight in four crosses of resistant and susceptible inbreds. Importance of additive, additive x additive, additive x dominance and dominance x dominance genetic effects in the cross CM139 x NAI147; additive, dominance, additive x additive, dominance x dominance effects best explained the genetic effects in cross CM139 x SKV18; additive, additive x dominance and dominance x dominance effects were important for leaf blight expression in cross CM139 x SKV21 and the analysis revealed that nature of inheritance of turcicum leaf blight resistance is population-specific (Hettiarachchi *et al.*, 2009).

Chaudhary and Mani (2010) studied the inheritance of the turcicum leaf blight using six generations derived from four susceptible (CM 128, V 327, V 128 and V 17) and two resistant inbred lines (V 335

and V 13), which revealed that additive, dominance and epistasis were important in the inheritance of turcicum leaf blight. The magnitude of additive component being relatively higher than non-additive component. They indicated that reciprocal recurrent selection be followed for the development of turcicum leaf blight resistant cultivars of maize for the Himalayan hilly region. Whereas Opio *et al.* (2010) also noted the significance of GCA and SCA effects, indicating the preponderance of both additive and non-additive gene action for resistance to northern corn leaf blight.

A half diallel study of 12 inbred lines was conducted to assess the types of gene action involved in turcicum leaf blight resistance across five locations in central and western Kenya, concluded that both additive and non-additive gene effects were important and highly dependent on test ecologies and proposed multi-locational breeding nurseries to identify turcicum resistant lines (Njoroge and Gichuru, 2013).

2.3.2 Sorghum downy mildew in maize

2.3.2.1 Nature and mechanisms of resistance

Renfro (1970) reported that the infection centres remain localized in resistant plants. The plants of less than four weeks old are more susceptible to infection by the pathogen and the infection become systemic. Craig (1985) compared ten inbreds for disease reaction by inoculating with conidia of *Perenosclerospora sorghi.* The positive correlation between the severity of the symptoms of inoculated leaves and degree of susceptibility to systemic infection in the field indicated that reaction of leaves to inoculation can be used to identify corn genotypes resistant to downy mildew.

Several types of resistance to *Perenosclerospora sorghi* exist in maize. The major symptoms associated with the diseases are (1) stripping

or chlorosis (systemic infection) and (2) local lesions (secondary infection) as reported by Lal and Singh (1984).

Craig and Schertz (1985) studied the inheritance of resistance and ascertained that resistance to *Perenosclerospora sorghi* was expressed as an incompatibility of host-pathogen relationship in which pathogen was unable to produce sporulation in the host tissue. However, histological studies revealed that inability of pathogen *Perenosclerospora sorghi* to grow in the leaf tissue of resistance genotypes was one of mechanisms of resistance to sorghum downy mildew in the field.

2.3.2.2 Genetics of resistance to sorghum downy mildew in maize

Sorghum downy mildew, or SDM, (*Peronosclerospora sorghi* (Weston and Uppal (Shaw)) infects maize (*Zea mays* L.) and causes serious economic yield losses. A major investment was made during the 1960's-1980 in an attempt to control this disease. Genetic resistance is a cost-effective and environmentally-safe alternative in controlling the downy mildews.

The most efficient, effective and economical means of controlling downy mildew disease is through the use of resistant genotypes. Immunity is unknown and resistance is relative to inoculum potential. Resistance breeding has become complex due to lack of stability of downy mildew resistance and physiological specialization of downy mildew fungus changing its virulence to overcome these host genes (Renfro, 1970).

Carangol *et al.* (1970) made a vital observation while breeding for resistance to *Sclerospora philippinensis* that resistance has to be incorporated before selection could be made. The frequency distribution of the S1 lines indicated that resistance is a quantitative trait followed an additive nature of inheritance.

Mochizuki *et al.* (1974); Yamada and Aday (1977) and Singhburandom and Renfro (1982) studied the mode of inheritance of resistance to Philippine downy mildew by means of diallel analyses. They all concluded that resistance was controlled by dominant gene(s) and that level of dominance was in the range of partial to over-dominance.

Asnani and Bhushan (1970) reported the importance of both additive and dominance type of gene action for disease under artificial epiphytotic conditions, however, they found partial to complete dominance of resistance for the disease.

Hardoo *et al.* (1970) also reported importance of both additive and non-additive types of gene action for resistance to brown stripe downy mildew in maize. On the contrary, if substantial additive genetic variance exists, host resistance can be built up by accumulation of genes; such an approach could go *via* mass selection, S_1 selection, full sib family selection or recurrent selection under optimum epiphytotic conditions. Conversely, in cases where resistance is dominant then recurrent selection for specific combining ability could be practiced. Even simple back cross method may be useful.

Bockholt and Frederiksen (1973) studied resistance to sorghum downy mildew in corn and reported that resistance is controlled either by two or three genes in a review on inheritance of resistance to downy mildew in maize. Frederiksen and Ullstrup (1975) studied the resistance to *Sorghum* downy mildew. Their results indicated resistance to be dominant in some crosses and recessive in others.

Singh and Asnani (1975) found that additive, dominance and epistatic effects were all important in majority of crosses, however, additive effects were more in magnitude than dominance and epistatic effects. They suggested that breeding for resistance involves the simultaneous study of genetic variability both in the host and the

pathogen to select suitable genotypes of the host which combines resistance to disease, high yielding potential and other favourable agronomic traits.

Bhat *et al.* (1982) indicated from their study on *Perenosclerospora sorghi* on sorghum that resistance was dominant and controlled by six genes. Craig (1982) indicated resistance to *Peronosclerospora sorghi* was conditioned by two linked genes with susceptibility being partially dominant.

Susceptibility to SDM is generally inherited as a dominant characteristic (Narong and Renfro, 1982). Both additive and non-additive gene actions were detected by portioning gene effects by generation means. They found that reaction of maize to downy mildew was governed by several genes and that the inheritance of resistance is complex. They identified an inbred line Mexico, Gpo.48 var.168-1 as a promising general source of resistance to downy mildew.

A diallel cross of six maize inbred lines was analysed for reaction to sorghum downy mildew. The crosses between resistant and susceptible lines showed intermediate disease reaction, suggesting a polygenic system for resistance to sorghum downy mildew in maize (Orangel and Borges 1987). De Leon (1990) suggested that variation in inheritance pattern many a times due to variable epiphytotic conditions present during the disease screenings.

DeMilliano (1992) reported that significant differences were found between entries for both downy mildew incidence (DMI) and downy mildew severity (DMS). Inoculation significantly increased mean DMI and about 40 *per cent* of sorghums had DMI above 10 *per cent*. Some 20 *per cent* showed a susceptible reaction to DMS in the field. Resistance differed between both sorghum races and groups according to country of

origin. Dura bicolor and Guinea bicolor races had more resistant genotypes than the other races.

De Leon *et al.* (1993) studied the genetics of resistance to Philippine downy mildew in three maize populations under artificial inoculations. The study revealed that the resistance inherited polygenically and controlled mainly by additive gene action. Susceptibility being largely governed by dominant effects together with epistasis, resulted in slow progress in the enhancement of downy mildew resistance using cyclical improvements procedure.

Studies on the inheritance of resistance to downy mildew in maize by Krishnappa *et al.* (1995) revealed significant dominant type of gene effect in all the four crosses suggesting its importance in the genetic control of resistance.

Geetha and Jayaraman (2002) stated that crosses between resistant lines and the susceptible lines showed intermediate disease reaction suggesting polygenic system for resistance to sorghum downy mildew in maize.

The resistance to both sorghum downy mildew and Rajasthan downy mildew was polygenically based, with the dominance of resistance over susceptibility (Nair *et al.*, 2004a). The mode of inheritance of resistance to Rajasthan downy mildew was less complex compared to sorghum downy mildew. The study also revealed the potential utility of inbred lines such as NAI 116 in devising a breeding strategy for integrated resistance to the two downy mildews.

Premalatha *et al.* (2010) conducted genetic studies for downy mildew resistant to reveal the occurrence of monogenic, digenic and also the possible role of polygenic inheritance.

2.3.3 Southern corn rust (*Puccinia polysora*)

2.3.3.1 Nature and mechanisms of resistance

Southern corn rust occur predominantly on the upper leaf surface, pustules (uredinia) are organish in colour, circular, raised and most prominent on the leaf but also occur on the stems and sheaths. Two types of resistance to *Puccinia polysora* exists in maize are (1) Monogenic sources of immunity or fleck-type resistance or race specific resistance to southern rust (UIIstrup, 1965) and (2) General resistance or adult plant resistance or as slow rusting to southern rust (Bailey, 1987; Zummo, 1988; Scott and Zummo, 1989).

General resistance is expressed in terms of amount of tissue not infected (uredia number) and ranges from nearly rust-free tissue to that heavily rusted and is desirable to rate rust infection as percentage infected tissue at different positions within the canopy as it occurs mainly on the upper portion of the leaf canopy (Hooker, 1985) while monogenic resistance which is seen when seedlings of various corn genotypes are inoculated with different isolates of *Puccinia polysora* (Robert, 1962; Ryland and Storey, 1955; Storey and Howland, 1959).

2.3.3.2 Genetics of resistance to southern corn rust

Southern corn rust is incited by *Puccinia polysora* (Underwood) infects maize (*Zea mays* L.) and causes enormous economic yield losses. A major investment was made during the 1950's in an attempt to understand the disease resistance and control disease. Genetic resistance is a cost-effective and environmentally-safe alternative in controlling the polysora rust.

Studies on the inheritance of resistance in several lines by using four corresponding segregating populations by Scott *et al.* (1984) revealed that one-gene or two-gene models with varying degrees of dominance

were used to explain the resistance found in different populations. The type of gene action involved in controlling rust included the complete, partial, or no dominance.

Scott and Zummo (1989) conducted genetic studies for resistance to *Puccinia polysora* to reveal single dominant gene for resistance response and also identified eight experimental maize inbreds having slow rusting resistance to *Puccinia polysora* on the basis of pustule number, smaller pustules and delayed pustule rupture.

The durable resistance to *Puccinia polysora* was polygenically based, with the dominance of resistance over susceptibility (Pandurangegowda *et al.*, 1993b). The study also revealed the potential utility of inbred lines such as CM-104 and CM-105 in devising a breeding strategy for durable resistance to southern rust.

Gingera *et al.* (1995) conducted studies on six generation mean analysis in five crosses of maize and reported the importance of additive effects in three crosses, dominance effects in two crosses and di-genic interactions were not significant in any of the five crosses. A large proportion of the genetic variation was explained by additive effects in controlling the first pustule appearance of common rust fungus (*Puccinia sorghi* L.).

Castellanos *et al.* (1998) revealed a vital observation while breeding for resistance to *Puccinia polysora* and *Exserohilum turcicum* that importance of emphasizing selection for disease resistance during line development.

Investigation on inheritance of resistance to southern rust (*Puccinia polysora* Underw.) in two $F_{2:3}$ populations was conducted by Holland *et al.* (1998) and indicated resistance is under simple genetic control, with a major gene or genes on chromosome 10S and minor genes

on chromosomes 3 and 4, together with their epistatic interactions, explaining much of the phenotypic variation for resistance with the dominance of resistance over susceptibility.

Pinho *et al.* (1999) conducted generation mean analysis for dissecting the genetic control of resistance to *Puccinia polysora* and *Physopella zeae* rusts in two maize crosses over two locations and indicated the significance of additive genetic effects without the epistatic effects was enough to explain the segregation for both pathogens. Genetic control of rust resistance was oligogenic and an average number of seven resistance genes to *Puccinia zeae* and three to *Puccinia polysora* were estimated.

Paterniani *et al.* (2000); Brewbaker, (2005) and Josue, (2007) studied the mode of inheritance of resistance to southern corn rust by means of diallel analyses and they concluded the importance of both additive and non-additive type of gene action for disease resistance, however, they indicated that additive effects were prevalent in controlling disease.

Silva *et al.* (2001) and Vieira *et al.* (2011) studied the mode of inheritance of resistance to southern corn rust by means of partial diallel analyses, which indicated the preponderance of additive gene effects for rust resistance based on GCA and SCA variances.

Chen *et al.* (2004) conducted genetic analysis of southern corn rust in five F_2 populations and BC_1F_1 population derived from resistant parent Qi319 clearly indicated that the resistance to southern corn rust is controlled by single dominant resistant gene, which was named RppQ.

Predominance of additive [$\hat{d}$] and additive x additive [$\hat{i}$] gene effects for southern corn rust score was observed in a study of three maize crosses using five generation mean analysis by So *et al.* (2003).

The additive [$\hat{d}$] gene effect was significant but negative, while dominance [$\hat{h}$] gene effect as well as non-allelic interactions ($\hat{i}$, and $\hat{l}$) were significant for rust resistance using six generation mean analysis by Ji, (2006) who suggested the presence of duplicate epistasis in controlling the disease.

Resistance appeared to be a slow-rusting or incomplete resistance and that was effective in adult plants but not in young seedlings. Severity of southern rust was less than 10 % on resistant progeny from crosses with Va59 compared with severity exceeding 70 *per cent* on susceptible progeny which was reported by Medina *et al.* (2007).

Predominance of non-additive gene action for rust rating score was reported by Agbaje *et al.* (2008) through line × tester analysis involving 84 hybrids produced by crossing 42 lines with two testers in maize while Woyengo *et al.* (2006) concluded the preponderance of additive genetic effects for controlling rust through line x tester analysis involving 144 top cross hybrids produced by crossing 72 lines with two synthetic testers.

Genetic studies for general resistance to southern rust through generation mean analyses of six crosses was conducted by Authrapun *et al.* (2009) and they revealed significant additive and dominant effects in three crosses and significant epistasis effect in the two crosses. General resistance to southern rust is governed by polygenic system in the inbred lines.

The inheritance of resistance to southern rust (*Puccinia polysora* Underw.) was studied in nine crosses of three resistant inbreds and three

susceptible inbreds through six generation mean analysis by Thaitad *et al.* (2008). The results revealed that predominance of additive gene effects for rust resistance in most of the crosses as well as epistatic effects in some crosses. Southern rust resistance was controlled by dominant genes with complete, partial and absence of dominance depending on the level of resistance in parents. Both duplicate and complementary interaction was found in crosses for resistance to southern rust.

Brewbaker *et al.* (2011) in their studies on six generation mean analysis reported importance of additive, dominance as well as epistatic effects in the genetic control of rust resistance. However, additive gene effect had major contribution and among epistatic effects, additive x additive was predominant.

2.4 Molecular markers and their utility in genetic dissection and breeding for resistance

Aim of any conventional/classical breeding methods is to develop resistant maize cultivars to the prevalent diseases in the locality through screening of germplasm for diseases and then selective crossing of selected promising resistant inbred lines, but it requires a long time to develop a resistant cultivars. Conventional strategy needs complementary information in order to accelerate the process and increase its accuracy, this is achieved through use of molecular tools to complement the classical breeding methods.

Genetic or DNA markers represent genetic differences between individual organisms or species. Genetic markers that are located in close proximity to genes (i.e. tightly linked) may be referred to as gene 'tags'. DNA markers may be broadly divided into three classes based on the method of their detection: (1) hybridization-based; (2) polymerase chain reaction (PCR)-based and (3) DNA sequence-based (Joshi and

Nguyen, 1993; Winter and Kahl, 1995; Jones *et al.*, 1997; Gupta *et al.*, 1999).

Restriction fragment length polymorphism (RFLPs), was the first marker system used to locate resistance genes and linkage map construction on maize. In this marker system, digestion of plant genomic DNA using restriction enzymes and the resultant fragments are then run on electrophoresis for their separation with bands formed later are blotted and hybridized with a probe. The main disadvantage of RFLP system is that it requires large amount of DNA and restriction enzymes are expensive (William *et al.* 2006). Amplified Fragment Length Polymorphism (AFLPs), which is a new marker system that combines both PCR amplification and restriction enzymes used for detection of polymorphisms more accurately. However, AFLPs are too slow to be produced as they need extensive marker screening, long capillary electrophoresis, and adaptor ligation (Dieguez *et al.*, 2006). The random amplified polymorphic DNA (RAPD) was the simplest marker system developed in which random primers were used to amplify regions; however, the method was very sensitive to PCR conditions, requiring repeating to meet the desired differential amplification (Gupta *et al.*, 2006).

Advances in genomics led to the identification of numerous DNA markers in maize during the last few decades, including thousands of mapped microsatellite or simple sequence repeat (SSRs) markers, and more recently, single nucleotide polymorphism (SNPs) markers. SSR has the advantage of ease in detecting polymorphisms between individuals, rather than population samples, of study. Until recently, SSRs have been the most widely used markers by maize researchers due to their availability in large numbers in the public domain (MaizeGDB, 2011; *http://www.maizegdb.org*), simplicity and effectiveness.

Single nucleotide polymorphisms (SNPs) are intraspecific differences in individual nucleotides, commonly used to asses genetic diversity due to their high frequency on both animal and plant chromosomes. As SNPs are amenable to automation, high throughput genotyping in maize using the SNP markers is possible; hence this approach saves time and manpower in detecting novel polymorphism that can be used in gene identification and QTL analysis of desirable plant traits (Mammadov *et al.*, 2012).

Next generation sequencing allows for high-throughput screening of thousands of SNPs. Notably, Illumina Bead Array assays, which are based on bead chips, have become popular as they allow the detection of SNPs of whole genomes in parallel, while overcoming sample preparation problems of some other methods (Fan *et al.*, 2006). Discovered SNPs are hybridized to the beads on the microscopic bead chip, which is divided into sections. SNPs are flanked with universal primers, and whenever a hit of an SNP occurs on the screened sample, a laser beam signal is emitted and detected. The reaction is PCR based and can accommodate 24 to 96-well plates. The reaction is also temperature controlled, robotic assisted, and automated (Gupta *et al.*, 2008).

Compared with the genomes of other cultivated plant species, SNP frequency in maize is high, with one SNP being found every 28–124 bp (Vroh-Bi *et al.*, 2006). A database and resource for SNP discovery and trait dissection have been established for maize in which genotype, phenotype and polymorphism data can be accessed for diverse maize inbred lines and populations (*http://www.panzea.org*). Nearly one million maize SNPs are available in public databases (*http://www.panzea.org*), and several high throughput genotyping platforms have been developed for commercial use (Prasanna, 2011).

2.5 Mapping quantitative trait loci (QTL) conferring resistance to multiple foliar pathogens in maize

A 'quantitative trait loci' is a region of the genome that is associated with an effect on a quantitative trait (Liu and Saint, 2002). 'QTL analysis' is the phrase used currently to study the quantitative genetic variation to locate the genes responsible for them and to explain their effects and interactions.

2.5.1 QTL mapping for northern corn leaf blight resistance

Brewster *et al.* (1992) identified regions on chromosome 3, the short arm of chromosome 4, and the long arm of chromosome 6 were consistently associated with northern leaf blight resistance in crosses of the resistant maize inbred Mo17 and susceptible translocation stocks.

In the $F_{2:3}$ population derived from the cross between Mo17 (moderately resistant) and B52 (susceptible), Freymark *et al.* (1994) revealed significant QTLs affecting disease severity on chromosomes 1S, 3L, 7L and 8L with additive gene action and partial dominance or overdominance. Later, Dingerdissen *et al.* (1996) located QTLs conferring resistance to northern leaf blight on chromosome arms 3L, 5S, 7L and 8L with partially dominant gene action and they together explained 48.8 *per cent* of the phenotypic variance.

Schechert *et al.* (1999) using CML 202 as the resistance source found 12 QTLs conferring resistance to northern leaf blight (AUDPC and Incubation period) in $F_{2:3}$ lines using RFLP markers on chromosomes 3, 4, 5, 8 and 9 over three environments in Kenya. The identified QTLs explained 52 and 56 *per cent* of the phenotypic variance of incubation period and AUDPC, respectively. The magnitude of the individual QTL effect was homogeneous and their gene action was additive.

Welz *et al.* (1999) detected 13 QTLs on chromosome bins 1.06/07, 3.06, 4.03, 5.04, 6.05/06 and 8.06 for resistance to *Setosphaeria turcica* in an early maturing Dent × Flint $F_{2:3}$ maize by composite interval mapping for two disease ratings (0 and 3 weeks after flowering) and these QTLs explained 48 *per cent* and 62 *per cent* of the phenotypic variation, respectively for disease ratings while Welz and Geiger (2000) indicated three QTL regions on chromosomes 3 (bin 3.06), 5 (bin 5.04), and 8 (8.06) were consistently significant in different populations and are suitable candidates for Marker Assisted Selection for resistance to northern corn leaf blight.

Brown *et al.* (2001) identified genomic regions associated with partial resistance to northern corn leaf blight using RFLP markers over two seasons. Fourteen QTLs distributed over eight chromosomes significantly controlling leaf blight severity were identified, of which seven QTLs accounts for 30.1 *per cent* of phenotypic variability in 1994 and seven QTLs accounted for 46.0 *per cent* variability in 1996. A common QTL located on 9.05 was detected over both years which explained 10.70 *per cent* of phenotypic variability.

Ping *et al.* (2007) identified three northern leaf blight resistance QTLs on chromosome 2 explaining 13.89, 19.33 and 14.36 *per cent* of the phenotypic variance, respectively and two QTLs conferring resistance on chromosome 8, which explained 9.33 and 7.62 *per cent* of the phenotypic variance, respectively.

Asea *et al.* (2009) mapped six QTLs (two each on chromosome bins 3.06, 5.04 and 8.06) conferring resistance to northern corn leaf blight (two leaf blight component traits namely *per cent* leaf area affected and SAUDPC) in both $F_{2:3}$ and $F_{3:4}$ families.

Advanced intercross recombinant inbred line population (IBM population) was used for locating the QTLs for weighted mean disease and incubation period related to northern leaf blight resistance over three environments (BalintKurti *et al.*, 2010). Several environment specific QTLs were detected; QTLs on 4.08 and 2.02 for weighted mean disease and incubation period were consistent over three environments.

Chung *et al.* (2010) characterized and fine-mapped a northern leaf blight QTL on chromosome bin 8.06 between markers *ctg358-18* – *ctg358-44* (10.20–11.20 cM on the S11 x DK888 genetic map, and 143.92–144.38 Mb on B73 physical map) which is likely to be identical, allelic, or closely linked to the known major gene Ht_2 explaining 60 *per cent* of phenotypic variability for disease leaf area affected.

In a study utilizing 109 recombinant inbred lines (RILs), mapping of QTLs conferring resistance to northern leaf blight was achieved (Zwonitzer *et al.*, 2010). Three QTLs that were located in chromosome bins 1.06, 8.02 and 8.05 accounted for 18.7, 12.8, and 8.3 *per cent* of phenotypic variation, respectively.

Chung *et al.* (2011) identified QTLs conferring resistance to northern leaf blight using heterogeneous inbred family (HIF) based targeted QTL analysis and classical RIL-based QTL mapping. Four QTLs (on chromosome bins 1.07–1.08, 5.03, 6.05, and 8.02–8.03) for northern leaf blight were identified using HIFs whereas four QTLs (on chromosome bins 1.06-1.07, 5.03, 6.05, and 8.05–8.06) mapped for Northern leaf blight resistance using RILs and these corresponded to QTLs found using the HIFs.

Poland *et al.* (2011) evaluated nested association mapping RILs population for Northern corn leaf blight over three seasons and identified 29 QTLs for resistance, each with a small effect using SNP markers. Later, Schaefer and Bernardo (2013) conducted Genome-wide

Association Mapping for identifying major QTL in a panel of 284 historical maize inbreds and identified 13 QTLs conferring resistance to northern corn leaf blight which collectively explained the total phenotypic variability of 55 *per cent*.

2.5.2 QTL mapping for sorghum downy mildew resistance

The first comprehensive analysis of QTLs determining the response of maize plant to sorghum downy mildew was carried out by Agrama *et al.* (1999). This study led to identification and mapping of three QTLs that significantly contributed to resistance to sorghum downy mildew (SDM). Two of these mapped loci were closely together on chromosome 1, while the third one was on chromosome 9. The percentage of phenotypic variances explained by each QTL ranged from 12.4 *per cent* to 23.8 *per cent*. Collectively, three QTLs identified in this study explained 53.6 *per cent* of the phenotypic variation in susceptibility to the infection.

QTL analysis of Krutto (2002) revealed that QTL-marker linkages were found on chromosome 3, 5, 6 and 9 for *per cent* infected plants, while the QTLs for *per cent* disease severity were linked to markers on chromosome 1, 2 and 4. The phenotypic variance explained were 35.34 and 47.14 % for *per cent* infected plants and *per cent* disease severity, respectively.

Canama *et al.* (2002) constructed a genetic linkage map using 142 BC_1F_1 mapping population derived from the cross between the susceptible parent, Pi 23 and the resistant source P345C4S2B46-2-2-1-2-B-B-B using 52 restriction fragment length polymorphism (RFLP), 33 simple sequence repeats (SSR) and 15 resistance gene analog (RGA) marker loci distributed into 10 linkage groups. The total map length was 1,822 cM with an average distance of 18.22 cM between markers. This represents 90% of the total map length (1,980 cM distributed in 99 bins) published for corn using RFLP-based markers.

George *et al.* (2003) located six genomic regions on chromosomes 1, 2, 6, 7 and 10 involved in the resistance to the downy mildews, explaining 26–57% of the phenotypic variance for disease response. A strong QTL on chromosome 6 was stable across environments, significantly affecting disease resistance at five locations in four Asian countries. Simple sequence repeat markers tightly linked to this QTL were identified for potential use in marker assisted selection.

Three QTLs (one each on chromosomes 2, 3 and 6) for sorghum downy mildew (SDM) resistance and two QTLs (one each on chromosomes 3 and 6) for Rajasthan downy mildew (RDM) resistance, all of which were contributed by resistant parent (NAI 116) were identified and mapped by Nair *et al.* (2005). Significance of the major QTL on chromosome 6 (bin 6.05) that confers resistance to diverse DMs in tropical Asia, including SDM and RDM in India, was also verified. The results confirmed that some common QTLs contribute to both SDM and RDM resistance, while additional loci might specifically govern resistance to SDM.

Sabry *et al.* (2006) identified three putative QTLs conferring resistance to downy mildew in different environments using composite interval mapping (CIM). Despite environmental and symptom differences, one locus on chromosome 2 had a major effect and explained up to 70 *per cent* of the phenotypic variation and other two QTLs on chromosome 3 and chromosome 9 had minor effects; each of which explained not more than 4 *per cent* of the phenotypic variation. The three QTLs appeared to have additive effects on resistance, identifying one major gene and two minor genes that contributed to downy mildew resistance.

Nine QTLs were identified by Jampatong *et al.* (2013) for resistance to downy mildew, one QTL each on chromosome 2, 3, 4 and 6, three QTLs on chromosome 5, and two QTLs on chromosome 9. All of the

resistant alleles on each QTL came from the resistant parent, Nei9008, except the QTL on chromosome 5.07 for which the resistance alleles came from the susceptible parent, CML289.

Hayde *et al.* (2012) successfully applied DNA marker technology to breed for DM resistant yellow corn variety for the Philippines. Corn microsatellite (SSR and EST-SSR) and resistance gene analog (RGA) markers were successfully used to (a) purify parent lines, (b) re-establish genetic structure of recombinant inbred line (RIL) population for genetic mapping, (c) fine-map DM resistance loci (QTL), (d) develop DM resistance gene-specific marker, (e) approximate combining ability based on genetic distance, and (f) in combined marker-assisted selection breeding (MAB) schemes. They validated resistance QTLs in multi-location DM screening involving the major corn growing regions in the Philippines.

Although the information generated with respect to QTL mapping for downy mildew resistance in maize has been impressive and encouraging, very few successful experiments on marker assisted selection (MAS) for improvement of downy mildew resistance in maize have been published. This may be attributed to the limited number of major QTLs identified for the trait, the reduced amount of phenotypic variance that each QTL expresses individually, their interaction with the environment, plus complexities associated with epistasis and GxE (Prasanna *et al.*, 2010). Some of these constraints are now overcome with the development of improved MAS protocols and availability of reliable PCR- based markers, which make screening of a substantial segregating population possible in a reasonable time period (Prasanna *et al.*, 2010).

2.5.3 QTL mapping for southern corn rust resistance

Holland *et al.* (1998) evaluated two $F_{2:3}$ populations for southern corn rust resistance in multiple environments and reported the significant QTL-marker (RFLP marker) linkages on chromosome 3, 4, and 10 for southern rust scores. The phenotypic variance ranged from 9 % to 83%. Despite environmental and symptom differences, loci on chromosome 10 had a major effect and explained up to 83 *per cent* of the phenotypic variation and other loci on chromosome 3 and chromosome 4 had minor effects.

Three putative QTLs (one each on chromosome 3, 4 and 9) conferring resistance to *Puccinia polysora* identified by Jiang *et al.* (1999) in different environments using composite interval mapping (CIM) explaining the phenotypic variance of 6.90%, 8.20% and 5.60%, respectively.

Brunelli *et al.* (2002) identified two microsatellite markers located on chromosome nine linked to QRL (quantitative resistance loci) to *Puccinia polysora*, which explained 12.90% and 5.10% of the phenotypic variance in resistance, respectively.

The first comprehensive analysis of QTLs determining the responses of maize plant to SCR was carried out by Jines *et al.* (2007). This study led to identification and mapping of four QTLs with resistance to southern corn rust in RILs that significantly contributed to resistance to southern corn rust. A major QTL was located on the short arm of chromosome 10 and 3 minor QTLs on chromosomes 4, 8 and 9. The percentage of phenotypic variance explained by each QTL ranged from 1.50 *per cent* to 82.70 *per cent*. Collectively, four QTLs identified in this study explained 87.80 *per cent* of the phenotypic variation in susceptibility to the infection.

Wanlayaporn *et al.* (2013) located six genomic regions on chromosomes 1, 2, 5, 6, 9 and 10 involved in the partial resistance or non-race specific resistance to the southern corn rust, explaining 75.50 *per cent* of the total phenotypic variance for disease response. A major QTL on chromosome 10 was consistent across two environments significantly affecting disease resistance. Simple Sequence Repeat markers tightly linked to QTL on chromosome 10 were identified for potential use in marker assisted selection.

2.6 Co-localization of QTLs conferring resistance to multiple foliar diseases

In maize, enormous amount of valuable information exists in the literature concerning QTL conditioning partial resistance to diseases that may be exploited to improve durable multiple disease resistance (MDR). Multiple disease resistance (MDR), in which the same chromosome locus conditioning resistance to multiple pathogens, is both practically and conceptually important. The detection of clusters of QTL conferring resistance to multiple diseases is consistent with but does not prove the hypothesis that MDR genes are present in plants (Wisser *et al.* 2006).

Many common chromosomal segments were associated with resistance to multiple diseases on the dQTL consensus map. Every maize chromosome had co-localizing dQTL for at least two different diseases and pointed out tight clusters of resistance factors in bins 3.04 and 6.01 (McMullen and Simcox, 1995). Later, Wisser *et al.* (2006) using a synthesized consensus map of disease QTL confirmed that these chromosomal bins 3.04-3.05 and 6.01 were associated with clusters of resistance factors.

Brown *et al.* (2001) made an effort to map QTLs for partial resistance to Stewart's wilt, northern corn leaf blight and common rust an $F_{2:3}$ population. They revealed six chromosomal regions, three were

associated with partial resistance to Stewart's wilt (chromosomes 4:07, 5:03, and 6:04), one was associated with NCLB (chromosome 9:05), and two were associated with common rust (chromosomes 2:04 and 3:04) and they were not able to find a common chromosomal region for these three diseases.

Wisser *et al.* (2008) evaluated the high resolution IBM mapping population over several environments for resistance to southern leaf blight, gray leaf spot and northern leaf blight and identified a co-localized QTL in bin 2.04 for SLB and GLS resistance; in bin 2.07, NLB QTL co-localized with GLS QTL and revealed that resistance effects for all three diseases were co-localized on chromosome 4, though the SLB effect was non-significant.

Asea *et al.* (2009) validated consensus QTLs conferring resistance to multiple foliar pathogens *viz.,* northern corn leaf blight to chromosomal bin 5.04, gray leaf spot to bin 4.08 and maize streak virus to bin 1.04 using 410 $F_{2:3}$ families and selected $F_{3:4}$ families derived from a cross between maize inbred CML202 and VP31.

Balint-Kurti *et al.* (2010) observed significant correlation between resistance to northern corn leaf blight, southern corn leaf blight and gray leaf spot and also detected chromosomal bins 2.00-2.01 and 4.08 controlling northern corn leaf blight resistance in an RIL population and also identified QTL for southern leaf blight and gray leaf spot. They found no co-localizing QTLs for northern corn leaf blight, southern corn leaf blight and gray leaf spot resistance.

Zwonitzer *et al.* (2010) located QTLs for resistance to southern leaf blight, gray leaf spot and northern leaf blight of maize in a RIL population and also evaluated for co-localizing QTLs conferring multiple disease resistance (MDR). Nine, eight, and six QTLs were identified for SLB, GLS, and NLB resistance, respectively. The QTLs for all three

diseases co-localized in bin 1.06; SLB and GLS resistance QTL co-localized in bins 1.08-1.09, 3.04/3.05; SLB and GLS resistance co-localized in bin 10.05; QTLs co-localized in bin 2.02/2.03 for NLB and GLS resistance and co-localized QTL in bin 8.05 for NLB and SLB resistance. This shows the evidence for presence of QTLs for multiple disease resistance in maize.

Chung *et al.* (2011) identified QTLs deliberating resistance to NLB and Stewart's wilt co-localized at bin 1.06–1.07 and QTL at bin 6.05 was associated with NLB and anthracnose stalk rot resistance, based on HIF- and/or RIL population. These identified QTLs confirmed existence of same chromosomal segments conditioning multiple disease resistance.

Belcher *et al.* (2012) worked out association between SLB weighted mean disease, NLB weighted mean disease, NLB incubation period and GLS weighted mean disease ratings in NILs. GLS-WMD was significantly associated with NLB disease ratings (WMD and IP) and SLB-WMD was moderately correlated with only NLB-WMD and concluded that introgressions 6A and 9B conferred resistance to multiple diseases.

Review of literature has revealed that several studies independently identified quantitative trait loci (QTL) conditioning resistance to foliar diseases *viz.*, northern corn leaf blight (*Exserohilum turcicum*), sorghum downy mildew (*Perenosclerospora sorghi*) and southern corn rust (*Puccinia polysora*) in different maize inbreds and populations. However, there are no reports regarding the co-localization of QTLs for resistance to these three foliar pathogens of maize.

Material and methods

III. MATERIAL AND METHODS

The present investigation was undertaken at the Zonal Agricultural Research Station, V. C. Farm, Mandya. The plant material used and methods followed for the present study are outlined in this chapter under the following headings.

3.1 General view of the experimental site

3.2 Genetic analysis of disease (northern corn leaf blight and southern corn rust) resistance through six generation mean analysis

3.3 Mapping of QTLs conferring resistance to three foliar pathogens of maize *viz.,* sorghum downy mildew, northern corn leaf blight and southern corn rust

3.4 Co – localization of QTLs for resistance to three foliar diseases in maize

3.1 General view of the experimental site

The study was conducted during *Kharif* 2011, *Rabi* 2011-12, summer 2012, *Kharif* 2012, *Rabi* 2012-13 and *Kharif* 2013 at the Zonal Agricultural Research Station, V. C. Farm, Mandya which is situated at an altitude of 695 meters above mean sea level. Geographically, it lies at latitude of 12°14' to 13°57'N and longitude of 76°24'E. The soil of the experimental site is light red sandy loam in nature and contains 83.2 *per cent* sand and, 5.9 *per cent* silt and 10.3 *per cent* clay. The average annual rainfall is about 710 mm and mean relative humidity is 82 *per cent* and the average maximum and minimum temperature is 26-35^{o} C and 14-22^{o} C, respectively. For the development of three foliar diseases, the conditions were congenial as rains were well distributed providing required humidity and temperature during the cropping period.

The Zonal Agricultural Research Station, V. C. Farm, Mandya is hot spot for screening majority of foliar diseases of maize and national disease screening nurseries are maintained for northern corn leaf blight, sorghum downy mildew and southern corn rust.

3.2 Genetic analysis of disease resistance (northern corn leaf blight and southern corn rust) through six generation mean analysis

3.2.1 Plant material

The material for the experiment comprised of six generations *viz.*, P_1, P_2, F_1, F_2, B_1 and B_2 for each of the two maize crosses *viz.*, CM212 × SKV50 and HKI162 × SKV50 developed at the Zonal Agricultural Research Station, V. C. Farm, Mandya. During *Kharif* 2012, the two F_1s were developed by crossing two inbreds with susceptibility to northern corn leaf blight and southern corn rust *viz.*, CM212 and HKI162 with resistant inbred SKV50 through hand pollination (Table 1 and Plate 1). Later in *Rabi* 2012-13, the F_1 plants of both crosses were selfed to produce F_2 generation as well as backcrossed to corresponding parents of each crosses to produce B_1 [(CM212 × SKV50) x CM212 and (HKI162 × SKV50) × HKI162] and B_2 [(CM212 × SKV50) x SKV50 and (HKI162 × SKV50) × SKV50] generations. During *Kharif* 2013, developed experimental material including all the generations was evaluated in the disease screening nurseries for northern corn leaf blight and southern corn rust at ZARS, V. C. Farm, Mandya.

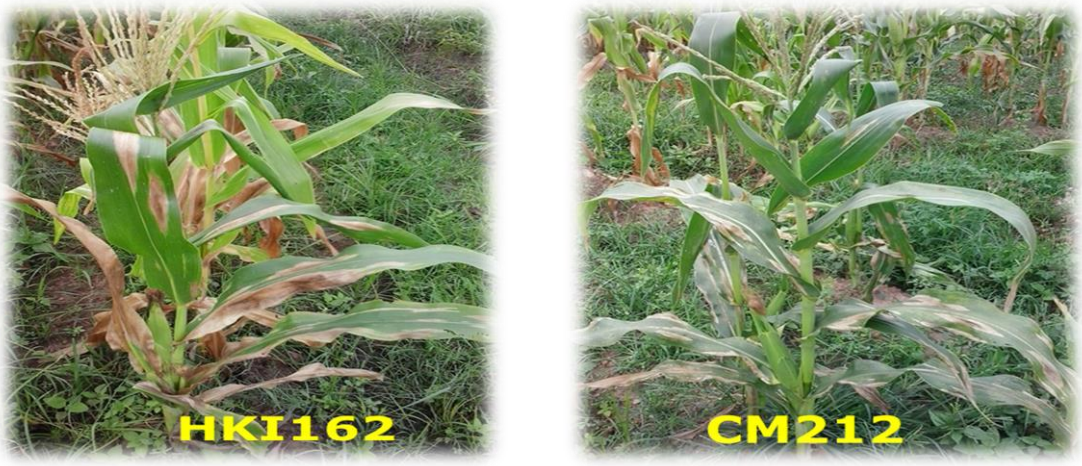
3.2.2 Screening for resistance to northern corn leaf blight

3.2.2.1 Field layout

The evaluation of six generations of the crosses *viz.*, CM212 × SKV50 and HKI162 × SKV50 was conducted in two replications during *Kharif* 2013 in the national disease screening nursery for northern corn leaf blight at ZARS, V. C. Farm, Mandya. The non-segregating

Table 1: Maize inbred lines used for understanding the genetics of resistance to northern corn leaf blight and southern corn rust through generation mean analysis

Inbred Line	Pedigree of inbred line	Source	Status of disease reaction		Use in crossing program as
			Northern corn leaf blight	Southern corn rust	
SKV50	Pop-147-F2#64-1-1-B-2-B	AICRP on Maize, VC Farm, Mandya	Resistant	Resistant	Male
CM212	EVPOP30-SRBC2/SR52#b-1-2sr-1-3-3-b-b	AICRP on Maize, VC Farm, Mandya	Susceptible	Susceptible	Female
HKI162	-	AICRP on Maize, VC Farm, Mandya	Susceptible	Susceptible	Female



Northern corn leaf blight susceptible inbred lines

Southern corn rust susceptible inbred lines

Northern corn leaf blight and Southern corn rust resistant inbred

Plate1. Resistant (SKV50) and susceptible (CM212 and HKI162) parents used for genetic analysis of resistance to northern corn leaf blight and southern corn rust

generations (Parents and F_1s) were grown in four row plots of 2 meter length, while the segregating generations *viz.*, B_1 and B_2 were grown in ten row plots, F_2s were grown in twenty row plots of 2 meter length. The entries were sown in rows spaced 75 cm apart and with an intra row spacing of 20 cm. Susceptible checks for northern leaf blight *viz.,* CM202 and NAI219J were sown after every 20th row to assess the disease pressure as well as to serve as spreader rows.

3.2.2.2 Creation of artificial epiphytotic condition

To ensure uniform disease infestation, artificial inoculation was done by following the procedure given by Shekhar and Kumar (2012). The initial inoculum for artificial inoculation of *Exserohilum turcicum* was grown in artificial media under laboratory condition. The infected leaf tissues were collected from the diseased field, and washed thrice with sterile water, cultured on potato dextrose agar medium, and then multiplied on sorghum seeds. For this, the sorghum seeds were soaked overnight, then transferred to sterilized conical flasks next day, and the pathogen inoculum was added. The flasks were shaken once in two days, and equal amount of fresh sorghum seeds were mixed after one week. The infected sorghum with pathogen inoculum was ground to fine powder, and 1 to 1.5 gram of the ground inoculum was added to each leaf whorl, followed by a light spray of water to moisten the tissue and initiate infection. Artificial inoculation for northern corn leaf bight made 20 to 30 days after sowing between 3.00 to 6.00 PM and inoculation was repeated twice after one week of first inoculation.

3.2.2.3 Disease scoring methodology

In non-segregating generation's *viz.*, parents and F_1s, the disease severity was recorded on 40 plants, whereas in the case of segregating generations, the data were recorded on 200 plants in F_2 generation and 100 competitive plants each in B_1 and B_2 generations of both crosses

viz., CM212 × SKV50 and HKI162 × SKV50. The northern corn blight severity was recorded at flowering stage *i.e.,* 60th day and also at dough stage *i.e.,* 80th day after sowing by visualizing the leaf area using a standard scale consisting of five broad categories designated by numerals from 1 to 5 (Payak and Sharma, 1983). Intermediate ratings between two numerals (1.5, 2.5, 3.5 and 4.5) were given, thereby providing for a total of nine classes or categories. Data were recorded at 60th day after sowing on a modified rating scale (based on the CIMMYT northern leaf blight rating system) as presented below.

Grade (or) Scale	Infection type	Reaction category
1	Very slight to slight infection, one or two to few scattered lesions on lower leaves	Highly resistant
1.5-2.0	Light infection, moderate number of lesions on lower leaves only	Resistant
2.5-3.0	Moderate infection, abundant lesions on lower leaves, few on middle leaves	Moderately resistant
3.5-4.0	Heavy infection, lesions abundant on lower and middle leaves, extending to upper leaves	Susceptible
4.5-5.0	Very heavy infection, lesions abundant on almost all leaves, plants prematurely dry or killed by the disease	Highly susceptible

3.2.3 Screening for resistance to southern corn rust

3.2.3.1 Field layout

The six generations of the crosses *viz.*, CM212 × SKV50 and HKI162 × SKV50 were evaluated for southern corn rust incidence during *Kharif* 2013. Parents and F_1s were grown in four row plots of 2m length,

while the segregating populations *viz.*, B_1 and B_2 were grown in ten row plots, F_2s in twenty row plots of 2 meter length. The spacing of 75 cm between rows and 20 cm between plants was provided. The inbred NAI219J which is susceptible to southern corn rust was sown as border rows around screening plots and also sown after every 20th row to assess the disease pressure as well as to serve as spreader rows.

3.2.3.2 Artificial inoculation

Artificial inoculation was done to ensure uniform establishment of disease by following the procedure of Shekhar and Kumar (2012). The uredospores of *Puccinia polysora* were collected on a butter paper by tapping severely infected leaves with fingers and then stored in glass tubes and were sealed. The uredospores thus obtained were kept in a freezer at lower temperature *i.e.*, 5 – 7^0 C and were used for inoculation purpose. The infected leaves were collected from severely diseased maize plots and were macerated thoroughly in between two palms of the hand dipped in the bucket of water, until the water got sufficiently colored. Inoculation for southern corn rust was made at 6 to 8 leaf stage and inoculation was repeated within 2 weeks. The spore (urediniospore) suspension @ 60, 000 spores/ml was applied in the leaf whorl using a syringe, atomizer or sprayer in the early morning hours.

3.2.3.3 Disease scoring methodology

The southern corn rust severity was assessed at dough stage *i.e.,* 80th day after sowing by visualizing the percentage leaf area covered by rust pustules. In parents and F_1s, the disease severity was recorded on 40 plants and on 200 plants in F_2 generation and 100 plants in B_1 and B_2 generations of both crosses *viz.,* CM212 × SKV50 and HKI162 × SKV50. The disease reactions were recorded using a rating scale of 1 to 5 (Cramer, 1967) and intermediate ratings between two numerals was

recorded (1.5, 2.5, 3.5 and 4.5) as per Shekhar and Kumar (2012). Southern corn rust rating was done at 80 days after sowing as below.

Grade (or) Scale	Infection type	Reaction category
1	Very slight-to-slight infection, one or two to few scattered pustules on lower leaves only	Highly resistant
1.5-2.0	Moderate number of pustules on lower leaves only (light infection)	Resistant
2.5-3.0	Abundant pustules on lower leaves; few on middle leaves	Moderately resistant
3.5-4.0	Abundant pustules on lower and middle leaves, extending to upper Leaves	Susceptible
4.5-5.0	Abundant pustules on all leaves, plant may dry prematurely or killed by the disease.	Highly susceptible

3.2.4 Statistical Analysis

3.2.4.1 Population means and variances

Means and variances in respect of disease severity for northern corn leaf blight and southern corn rust were calculated for each population using data recorded on individual plants as detailed below.

$$\text{Mean} = \Sigma x_i / n$$

$$\text{Variance} = \frac{\Sigma x_i^2 - (\Sigma x)^2/n}{n-1}$$

$$\text{Variance of sample mean} = \text{Variance}/n$$

$$\text{Standard error (SE)} = \sqrt{\text{Variance of population}} / n$$

Where,

x_i = i^{th} observation of a population; n = Number of observations

3.2.4.2 Mather's scaling tests

The three quantities of scaling tests *viz.*, A, B, C and D were calculated following the methods of Mather (1949) to detect the presence or absence of epistasis as detailed below.

$$\mathbf{A = 2\bar{B}_1 - \bar{P}_1 - \bar{F}_1} \qquad \mathbf{B = 2\bar{B}_2 - \bar{P}_2 - \bar{F}_1}$$

$$\mathbf{C = 4\bar{F}_2 - 2\bar{F}_1 - \bar{P}_1 - \bar{P}_2} \qquad \mathbf{D = 2\bar{F}_2 - \bar{B}_1 - \bar{B}_2}$$

Where, $\bar{P}_1$, $\bar{P}_2$, $\bar{F}_1$, $\bar{F}_2$, $\bar{B}_1$ and $\bar{B}_2$ are the mean values of the respective generations. Significant deviation of A, B and C values from zero indicate presence of epistasis justifying the use of six parameter model.

Variance of mean of the corresponding generation was used to test the significance of these three quantities. The variance of A, B, C and D were calculated as follows.

$$\mathbf{V_A = 4\ V(\bar{B}_1) + V(\bar{P}_1) + V(\bar{F}_1)} \qquad \mathbf{V_B = 4\ V(\bar{B}_2) + V(\bar{P}_2) + V(\bar{F}_1)}$$

$$\mathbf{V_C = 16\ V(\bar{F}_2) + 4\ V(\bar{F}_1) + V(\bar{P}_1) + V(\bar{P}_2)} \qquad \mathbf{V_D = 4V(\bar{F}_2) + V(\bar{B}_1) + V(\bar{B}_2)}$$

Where,

V_A, V_B, V_C and V_D are the variances of A, B, C and D, respectively while V_{P1}, V_{P2}, etc., are the variances of means of the respective generations.

The 't' values for each of these three quantities were calculated as follows,

$$\mathbf{t_{(A)} = A/SE(A)\ ;} \quad \mathbf{t_{(B)} = B/SE(B);} \quad \mathbf{t_{(c)} = C/SE(C);} \quad \mathbf{t_{(D)} = D/SE(D)}$$

Where,

Standard error (SE) is the square root of respective variance**.**

The calculated values of 't' were then compared with 1.96 which is the tabulated value of t at 5 % level of significance.

The value of A, B, C and D should be equal to zero within the limits of their standard error. The significance among any one of these scales is taken to indicate presence of non-allelic interaction.

3.2.4.3 Joint scaling test

The parameters such as m, the mean of all possible genotypes arising out of selfing of a cross, [$\hat{d}$] additive gene effect and [$\hat{h}$] dominance deviation effects were estimated from the observed means of six generations (P_1, P_2, F_1, F_2, B_1, and B_2) using Cavalli's (1952) joint scaling test as described by Mather and Jinks (1982). The estimates of these parameters were then used to calculate expected means which were then compared with observed means assuming the adequacy of additive-dominance model. Since six generation means were used to estimate three parameters, weighted least square analysis was employed as it enables precise estimation of m, [$\hat{d}$] and [$\hat{h}$] by reducing the error mean square associated with the segregating generations that contain a greater number of individuals than the non-segregating generations. In this approach, reciprocal of variance of the means of each generation was used as weight. Because the means of various generations may not be known with equal precision, the weights were used to estimate m, [$\hat{d}$] and [$\hat{h}$] components.

Three normal equations were developed to estimate m, [$\hat{d}$] and [$\hat{h}$]. First normal equation was obtained by multiplying each element of all the six rows comprising of co-efficients and means by the product of weight and co-efficient of 'm' and adding over six generations.

In the similar manner, second normal equation was obtained by multiplying each element of all the six rows by the product of weight and coefficient of $[\hat{d}]$ and the third normal equation was obtained by multiplying each element of all the six rows by the product of co-efficient of $[\hat{h}]$ and its weight. The three normal equations obtained were as follows

$$a_{11}\, m + a_{12}\, d + a_{13}\, h = A_1$$

$$a_{21}\, m + a_{22}\, d + a_{23}\, h = A_2$$

$$a_{31}\, m + a_{32}\, d + a_{33}\, h = A_3$$

Where,

A_1 = 1 (weight × co-efficient of 'm' × observed mean)

A_2 = 1 (weight × co-efficient of 'd' × observed mean)

A_3 = 1 (weight × co-efficient of 'h' × observed mean)

The three normal equations so derived were arranged in the following matrix form.

$$\begin{bmatrix} a_{11} & a_{12} & a_{13} \\ a_{21} & a_{22} & a_{23} \\ a_{31} & a_{32} & a_{33} \end{bmatrix} \begin{bmatrix} m \\ d \\ h \end{bmatrix} = \begin{bmatrix} A_1 \\ A_2 \\ A_3 \end{bmatrix}$$

[J] [M] = [S]

The elements of the three normal equations were obtained from the co-efficient matrix and termed as "Information matrix" (J). The parameters to be estimated *viz.*, m, $[\hat{d}]$ and $[\hat{h}]$ were arranged in a column matrix (M). The elements of normal equations obtained as a result of multiplication of observed values by co-efficient and weights were arranged in column matrix (S) called "Score matrix".

Estimates of m, [$\hat{d}$] and [$\hat{h}$] were obtained as M = SJ^{-1} where J^{-1} is the inverse of information matrix, which is a variance-covariance matrix in which principal diagonal elements represent variances of the estimates of the parameters m, [$\hat{d}$] and [$\hat{h}$].

The standard error of each of these estimates was obtained as under root of the diagonal elements of the inverse matrix. The significance of these three parameters was tested against their respective standard error as follows.

$$t = \frac{\text{Estimated parameter}}{\text{SE of the parameter}}$$

The calculated 't' value was tested against table 't' value of 1.96 for testing the significance.

Estimated values of m, [$\hat{d}$] and [$\hat{h}$] were then used to calculate expected means of six generations by using the following formula:

$\bar{P}_1 = m + [\hat{d}]$ $\quad$ $\bar{P}_2 = m - [\hat{d}]$

$\bar{F}_1 = m + [\hat{h}]$ $\quad$ $\bar{F}_2 = m + ½ [\hat{h}]$

$\bar{B}_1 = m + ½ [\hat{d}] + ½ [\hat{h}]$ $\quad$ $\bar{B}_2 = m - ½ [\hat{d}] + ½ [\hat{h}]$

The deviations of observed means of six generations from those of expected means were tested using Chi-square test as follows:

$$\text{Chi} - \text{square} = \frac{(\text{Observed generation mean} - \text{Expected generation mean})2}{\text{Expected generation mean}}$$

The chi-square value was compared with table x^2 at (6-3) degrees of freedom. The non-significance of chi-square value indicated the adequacy of additive-dominance model. Significance of chi-square test was indicative of non-adequacy of additive-dominance model. The traits for which additive-dominance model was inadequate to explain their

inheritance, the di-genic interaction effects, *viz.*, additive × additive [$\hat{i}$], additive × dominance [$\hat{j}$] and dominance × dominance [$\hat{l}$] were estimated employing six parameter model using perfect fit solution (Jinks and Jones, 1958).

3.2.4.4 Estimation of gene effects using generation means

The six parameter model (Jinks and Jones, 1958; Mather and Jinks, 1971) was used to estimate gene effects for the traits for which additive-dominance model was inadequate as indicated by joint scaling test of Cavalli (1952)/scaling tests of Mather (1949).

Mean $= \hat{m} = \frac{1}{2}\bar{P}_1 + \frac{1}{2}\bar{P}_2 + 4\bar{F}_2 - 2\bar{B}_1 - 2\bar{B}_2$

Additive effect $= [\hat{d}] = \frac{1}{2}\bar{P}_1 + \frac{1}{2}\bar{P}_2$

Dominance effect $= [\hat{h}] = 6\bar{B}_1 + 6\bar{B}_2 - 8\bar{F}_2 - \bar{F}_1 - \frac{3}{2}\bar{P}_1 - \frac{3}{2}\bar{P}_2$

Add × Add effect $= [\hat{\imath}] = 2\bar{B}_1 + 2\bar{B}_2 - 4\bar{F}_2$

Add × Dom effect $= [\hat{\jmath}] = 2\bar{B}_1 - \bar{P}_1 - 2\bar{B}_2 + \bar{P}_2$

Dom × Dom effect $= [\hat{l}] = \bar{P}_1 + \bar{P}_2 + 2\bar{F}_1 + 4\bar{F}_2 - 4\bar{B}_1 - 4\bar{B}_2$

The variances of gene effects were estimated as:

$$V_m = \frac{1}{4}V_{\bar{p}_1} + \frac{1}{4}V_{\bar{P}_2} + 16\,V_{\bar{F}_2} + 4\,V_{\bar{B}_1} + 4\,V_{\bar{B}_2}$$

$$V_{[d]} = \frac{1}{4}V_{\bar{P}_1} + \frac{1}{4}V_{\bar{P}_2}$$

$$V_{[h]} = 36\,V_{\bar{B}_1} + 36\,V_{\bar{B}_2} + 64\,V_{\bar{F}_2} + V_{\bar{F}_1} + \frac{9}{4}V_{\bar{P}_1} + \frac{9}{4}V_{\bar{P}_2}$$

$$V_{[i]} = 4\,V_{\bar{B}_1} + 4\,V_{\bar{B}_2} + 16\,V_{\bar{F}_2}$$

$$V_{[j]} = 4\,V_{\bar{B}_1} + V_{\bar{P}_1} + 4\,V_{\bar{B}_2} + V_{\bar{P}_1}$$

$$V_{[l]} = V_{\bar{P}_1} + V_{\bar{P}_2} + 4V_{\bar{F}_1} + 16V_{\bar{F}_2} + 16V_{\bar{B}_1} + 16V_{\bar{B}_2}$$

Standard errors of various gene effects were estimated as:

$$SE_m = \sqrt{V_m} \qquad SE_{[d]} = \sqrt{V_{[d]}}$$

$$SE_{[h]} = \sqrt{V_{[h]}} \qquad SE_{[i]} = \sqrt{V_{[i]}}$$

$$SE_{[j]} = \sqrt{V_{[j]}} \qquad SE_{[l]} = \sqrt{V_{[l]}}$$

The 't' value for all the gene effects were calculated as given below

$$t_m = \frac{m}{SE_m} \qquad t_{[d]} = \frac{[d]}{SE_{[d]}}$$

$$t_{[h]} = \frac{[h]}{SE_{[h]}} \qquad t_{[i]} = \frac{[i]}{SE_{[i]}}$$

$$t_{[j]} = \frac{[j]}{SE_{[j]}} \qquad t_{[l]} = \frac{[l]}{SE_{[l]}}$$

These calculated 't' values were compared with 1.96 which is tabulated value of 't' at 5 % level of significance for testing the significance of various gene effects as done for scaling tests.

3.2.4.5 Number of effective factors

The minimum number of effective factors differentiating the parents was worked out using the formula given by Wright (1968).

$$N = \frac{0.25\,(0.75 - h + h^2)\,D^2}{V\bar{F}_2 - V\bar{F}_1}$$

Where,

N = Estimated number of effective factors

$h = (\bar{F}_1 - \bar{P}_1) \,/\, (\bar{P}_2 - \bar{P}_1)$

$D = \bar{P}_2 - \bar{P}_1$

$V\bar{F}_1$ and $V\bar{F}_2$ = Variances of the F_1 and F_2 generations, respectively.

$\bar{P}_1$ and $\bar{P}_2$ = Means of the smaller and greater parent, respectively.

3.2.4.6 Potence ratio (PR)

The Potence Ratio (PR) which indicates the degree of dominance was computed from generation means as per Peter and Frey (1966).

In F_1 generation, $\hat{h}_1 = PR_1 = (\bar{F}_1 - \bar{P}) / (\bar{P}_1 - \bar{P})$

In F_2 generation, $\hat{h}_2 = PR_2 = 2(\bar{F}_2 - \bar{P}) / (\bar{P}_1 - \bar{P})$

Where,

$\bar{P}$ = Average of two parents *i.e.*, $(\bar{P}_1 + \bar{P}_2)/2$

$\bar{P}_1$ = Mean of superior parent involved in the F_1

$\bar{P}_2$ = Mean of inferior parent involved in the F_1

$\bar{F}_1$ = Mean of F_1 population

$\bar{F}_2$ = Mean of F_2 population

Based on the potence ratio, the degree of dominance in respect of different traits were classified as follows,

PR < 0 - No dominance

PR > 1 - Over dominance

PR < 1 - Partial dominance

PR = 1 - Complete dominance

3.3 Mapping of QTLs conferring resistance to multiple foliar diseases *viz.,* northern corn leaf blight, sorghum downy mildew and southern corn rust

3.3.1 Development of mapping population

. Based on the previous year's data on disease reaction at All India Co-ordinated Maize Improvement Project at ZARS, V.C. Farm, Mandya., two contrasting inbred lines *viz.,* CML153 (susceptible inbred, P_1) and

SKV50 (resistant inbred, P_2) for disease reaction against northern corn leaf blight, sorghum downy mildew and southern corn rust were selected for the development of mapping population. The selected contrasting inbreds were crossed during *Kharif* 2011. The F_1 (CML153 × SKV50) was grown during *Rabi* 2011-12 and self-pollinated to form a set of 344 F_2 individuals. These 344 F_2 individuals selfed to derive $F_{2:3}$ families (Appendix 1) during summer 2012 and from each $F_{2:3}$ families, leaves from five randomly selected plants were collected for genotypic analysis. Later, during *Kharif* 2012 and *Kharif* 2013, $F_{2:3}$ families were challenged/screened for disease reaction against northern corn leaf blight, sorghum downy mildew and southern rust in the national disease nurseries maintained at ZARS, V. C. Farm, Mandya.

3.3.2 Genotyping of $F_{2:3}$ mapping population

A total of 765 SNPs (Appendix 2) covering maize whole genome (*www.panzea.com*) was used for genotyping of 344 $F_{2:3}$ population and their parents. Leaf samples were pooled from 5 random plants per $F_{2:3}$ family and per parent, and lyophilized in 96 well plates. Samples were loaded to the Illumina Bead Xpress Vera Code Reader for genotyping, according to Illumina protocols *(http://www.illumina.com/)*.

Scoring of SNP marker generated bands

The polymorphism detected by SNP marker was scored as follows:

A = homozygous maternal genotype (CML153)

B = homozygous paternal genotype (SKV50)

H = heterozygote genotype

- = Missing samples

3.3.3 Phenotyping of $F_{2:3}$ mapping population

Seeds from the self-pollinated 344 F_2 individual plants, along with the two parental lines were screened against the foliar pathogens *viz.,* *Exserohilum turcicum*, causing northern corn leaf blight, *Perenoscelrospora sorghi* causing sorghum downy mildew and *Puccinia polysora* causing southern corn rust of maize. The disease screening was conducted in two seasons during *Kharif,* 2012 and *Kharif,* 2013 in the national disease screening nurseries maintained at Mandya, separately.

3.3.3.1 Northern corn leaf blight (*Exserohilum turcicum*)

The experiment consisted of 344 $F_{2:3}$ progenies along with two parents was challenged against the *Exserohilum turcicum* fungus in the disease screening nursery during *Kharif*, 2012 and *Kharif,* 2013 using Randomized Complete Block Design with two replications. All the progenies were planted in a 2m single row of 10 plants each with a spacing of 75 cm between rows and 20 cm between plants. The artificial inoculation procedure as in 3.2.2.2 was followed for creating disease epiphytotic condition. The northern leaf blight severity was assessed as in 3.2.2.3 at flowering stage *i.e.*, 60th day after sowing by visualizing the leaf area. The disease score data were converted into *per cent* disease severity by using the formula given by Wheeler (1969).

$$\text{Disease Severity} = \frac{\text{Sum of individual disease ratings}}{\text{Total no. of plants observed X Maximum grade}}$$

3.3.3.2 Sorghum downy mildew (*Perenosclerospora sorghi*)

The 344 F_3 progenies derived from the cross CML153 × SKV50 along with two parents were evaluated in the sorghum downy mildew nursery in Mandya during *Kharif* 2013 under Randomized Complete Block Design with two replications. All the progenies were planted in a 3m single row

of 20 plants each with a spacing of 75 cm between rows and 20 cm between plants.

The 'sandwich method and spreader row technique' was used for screening the genotypes against sorghum downy mildew (Craig *et al.* 1977). In the sandwich method, seeds of maize line CM 500 were inoculated with SDM before planting. The seeds were first spread out between layers of DM-infected maize leaves with visible conidial growth and then incubated for 2–3 days at room temperature (100% relative humidity under light) to facilitate entry and establishment of the fungus into the germinating seeds. The infected seeds were planted as spreader rows on all sides of the experimental block 30 days prior to the planting of the test entries. One bed of spreader row was planted for every two beds of test entries. As a susceptible check, uninfected CM 500 seeds were planted after every tenth row of test materials. Spraying of spores of the pathogen was done seven days after sowing using Knapsack sprayer in the early morning at 3AM for about seven to ten days *i e.,* till the disease symptoms appeared on the seedlings of susceptible check (CM500). Severe infection (98–100% DM incidence) in the check rows across the experimental block indicated uniform and strong pathogen pressure, leaving no possibility for 'disease escapes'.

Recording of observations

The disease reaction was assessed at 25 and 35 days after planting by scoring for systemic DM infection in the individual plants. Systemically infected plants were taken into consideration while recording the disease incidence. Observations were recorded in only two classes *viz.,* 1) downy mildew infected and 2) healthy plants at 30 days after sowing of crop. Total number of plants showing systemic infection in a genotype was counted and the *per cent* disease incidence was calculated using following formula:

$$\text{Disease Incidence} = \frac{\text{Number of infected plants}}{\text{Total no. of plants}} \text{ X } 100$$

The reactions of the $F_{2:3}$ progenies against *P. sorghi* were classified into six different classes as per Rao *et al.* (1984).

Class	*Per cent* disease incidence	Reaction category
1	0.00	Highly resistant (HR)
2	0.1 – 10	Resistant (R)
3	10.1 – 15	Moderately resistant (MR)
4	15.1 – 30	Moderately susceptible (MS)
5	30.1 – 50	Susceptible (S)
6	50.1 – 100	Highly susceptible (HS)

3.3.3.2 Southern corn rust / polysora rust (*Puccinia polysora*)

The 344 $F_{2:3}$ individuals along with two parents were challenged against the *Puccinia polysora* fungus in disease nursery maintained at Mandya during *Kharif*, 2013 under Randomized Complete Block Design with two replications. All the progenies were planted in a 2m single row of 10 plants each with a spacing of 75 cm between rows and 20 cm between plants. The artificial inoculation procedure as in 3.2.3.2 was followed for creating disease epiphytotic condition. The Southern corn rust severity was assessed as in 3.2.3.3 at dough stage i.e., 80th day after sowing by visualizing the percentage leaf area covered by rust pustules. The disease score data were converted into *per cent* disease severity by using the formula given by Wheeler (1969).

$$\text{Disease severity} = \frac{\text{Sum of individual disease ratings}}{\text{Total no. of plants observed X Maximum grade}}$$

3.3.4 Statistical analysis

The statistical analysis on the mean values of each $F_{2:3}$ families with two replications was carried out using SAS (version 9.3) statistical package. Different statistical methods employed for analysis are presented below.

3.3.4.1 Phenotypic data analysis

3.3.4.1.1 Transformation of field data

The phenotypic data from field was recorded as *per cent* disease index of northern leaf blight and southern corn rust infection, percentage of plants showing sorghum downy mildew infection. The percentages covered a wide range of values between zero and one hundred. Such data generally have a binomial distribution rather than a normal distribution, which causes the variance to be related to the means but in quite a different way. The arcsine transformation is the appropriate transformation recommended by Little and Hills (1978) for this type of data. This transformation was expected to make the means and variances independent and normally distributed. For field data, analysis of variance was conducted on transformed phenotypic data using PROC GLM, procedure of SAS package version 9.3.

3.3.4.1.2 Analysis of variance (ANOVA)

The treatment mean for each replication was calculated and used for analysis of variance. The ANOVA table was constructed following the procedure of Panse and Sukhatme (1964). Analysis was done using SAS 9.3 statistical package.

Source of variation	Df	SS	MS	'F' ratio
Replication	(r-1)	A	P	P/R
Entries	(t-1)	B	Q	Q/R
error	(r-1)(t-1)	C	R	-

Where,

r = number of replications

t = total number of genotypes

A = replication sum of squares

B = treatment sum of squares

C = Error sum of squares

P, Q and R = mean sum of squares for replication, treatment and error, respectively

The significance was tested by referring to the table given by Fisher (1936). Critical difference (CD) and coefficient of variation (CV) were worked out using appropriate formulae comparing means of the genotypes.

Bartlett's test was used to test for homogeneity of error variances between data obtained from two seasons (*Kharif* 2012 and *Kharif* 2013) before pooling northern corn leaf blight data (Gomez and Gomez, 1984). Components of variance for the $F_{2:3}$ families across seasons were computed considering all effects (seasons, replicates and $F_{2:3}$ families) as random in the statistical model. Transformed entry means were used to compute the combined analyses of variance and covariance across seasons as described by Bohn *et al.* (1996). Estimates of variance components σ_e^2 (error variance), σ_{ge}^2 (genotype-by environment (G x E) interaction variance), and σ_g^2 (genotypic variance) of $F_{2:3}$ families were

calculated as described by Searle (1971). Heritability (H^2) on a $F_{2:3}$ family transformed mean basis was estimated as described by Hallauer and Miranda (1981).

$$H^2 = \frac{\sigma 2\, g}{\sigma 2\, g + \frac{\sigma 2\, g}{r\, e} + \frac{\sigma 2\, ge}{e}}$$

where,

σ_g^2 = denotes the genetic variance

σ_{ge}^2 = is genotype × environment interaction

σ_e^2 = is experimental error

r =number of replications

e= number of test environments

3.3.4.1.3 Two way analysis of variance

The data obtained on sorghum downy mildew, northern leaf blight and southern corn rust incidence on $F_{2:3}$ families across two seasons were subjected to two way analysis of variance as per Sundararaj *et al.* (1972) in order to find out the variation due to genotypes and environments to reveal the existence of genotype × environment interaction.

Source of variation	**d.f.**	**MS**	**Expected value of MS**	**F_{cal}**
Replications	(r-1)	M_1	-	-
Environments	(e-1)	M_2	-	-
Genotypes	(g-1)	M_3	$\sigma^2e + \sigma^2ge + e\sigma^2g$	-
Genotypes × Environment	(g-1) (e-1)	M_4	$\sigma^2e + \sigma^2ge$	-
Pooled error	M*	M_5	σ^2e	

* Degrees of freedom pooled over seasons

3.3.4.1.4 Descriptive statistics

The following descriptive statistics were calculated as per Sundararaj *et al.* (1972).

Mean: Mean is the sum of all observations in a sample divided by number of observations (n).

Where,

X_i = i^{th} observation of a population

n = number of observations

Range: Range is the minimum and maximum values of the observations in a sample.

Test of normality and nature of frequency distribution

The normal distribution (the term first used by Galton, 1989) function was determined by the following formula:

$$f(x) = 1/ [(2*x) ½ *σ]*e**\{-1/2 * (x-μ)/ σ2\}$$

Where,

'μ' is the mean

'σ' is the standard deviation

'e' is Euler's constant (2.71)

'x' is the constant φ (3.14)

Frequency distribution of $F_{2:3}$ families for northern corn leaf blight, sorghum downy mildew and southern corn rust incidence was constructed by plotting trait mean values on X-axis and frequency or counts of $F_{2:3}$ families on Y-axis.

Kolmogorov-Smirnov goodness of fit test

The Kolmogorov-Smirnov test is used to decide if a sample comes from a population with a specific distribution. The Kolmogorov-Smirnov (K-S or D) test purpose is to test for Distributional Adequacy.

If the 'D' statistic is significant, then the hypothesis that the respective distribution is normal should be rejected. Test of normality for all the traits recorded was carried out using 'SAS' software program.

Skewness and kurtosis

Skewness, the third degree statistics and kurtosis, the fourth degree statistics were estimated as per Snedecor and Cochran (1994) to understand the nature of distribution of $F_{2:3}$ families for northern leaf blight, sorghum downy mildew and southern corn rust resistance. The mean values of three traits of the cross were used to estimate coefficients of skewness and kurtosis using 'SPSS version 16' software program. Kurtosis indicates the relative number of genes controlling the traits (Robson, 1956). Three types of kurtosis are recognized based on the kurtosis value which depends on distribution curve.

If kurtosis value = 3 = Normal curve = Mesokurtic

If kurtosis value > 3 = Leaping curve = Leptokurtic

If kurtosis value < 3 = Flat curve = Platykurtic

Similarly, the lack of symmetry *i.e.,* skewness was recognized based on the co-efficient of skewness values which range from -3 to +3. The type of distribution based on the skewness values are as follows.

If skewness value is zero = symmetrical distribution

If skewness value is negative = negatively skewed distribution

If skewness value is positive = positively skewed distribution

3.3.4.1.5 Estimation of genetic variability parameters

The genetic variability parameters for northern corn leaf blight, sorghum downy mildew and southern corn rust were estimated in the F_3 population as detailed below:

Components of variance

Phenotypic and genotypic components of variance in F_3 population were computed as per the method suggested by Lush (1945).

$$\text{Phenotypic variance } (V_p) = V_g + V_e$$

$$\text{Genotypic variance } (Vg) = \frac{GMS - EMS}{r}$$

$$\text{Environmental variance } (V_e) = EMS$$

Where,

r = replication

GMS = Genotypic mean sum of squares from ANOVA

EMS = Error mean sum of squares from ANOVA

Co-efficients of variation

The co-efficient of variability both at phenotypic and genotypic levels for all the characters were computed by applying the formula as suggested by Burton and De Vane (1953).

1. **Genotypic coefficient of variation (GCV)**

$$GCV\ (\%) = \frac{\sigma g}{X} X\ 100$$

2. **Phenotypic coefficient of variation (PCV)**

$$PCV\ (\%) = \frac{\sigma p}{X} X\ 100$$

Where,

$\bar{X}$ = mean of the population

σ_p = Phenotypic standard deviation of F_3 population

σ_g = Genotypic standard deviation of F_3 population

PCV and GCV were classified as suggested by Robinson *et al.* (1949).

Low = 0.1-10%

Moderate = 10.1%-20%

High = >20%

Heritability (H^2)

Heritability in broad sense for all the characters was estimated for all the characters as the ratio of genotypic variance to the total phenotypic variance as suggested by Johnson *et al.* (1955).

$$h^2_{(bs)} = \frac{\sigma 2g}{\sigma 2p} X\ 100$$

Where,

h^2_{bs} = Heritability (broad sense) expressed in *per cent*

σ^2_g = Genotypic variance

σ^2_p = Phenotypic variance

The heritability was classified as suggested by Robinson *et al.* (1949).

Low = < 30%

Moderate = 30-60%

High = >60%

Genetic advance (GA)

The extent of genetic advance expected through selection for each character was estimated by using the following formula given by Johnson *et al.* (1955).

$$\text{Genetic advance (GA)} = K\ h^2\ \sigma_p$$

Where,

K = Selection differential which is equal to 2.06 at 5 *per cent* selection intensity

h^2 = Broad sense heritability estimate

σ_p = Phenotypic standard deviation

Further, the GA as *per cent* of mean (GAM) was estimated by using the following formula

$$\text{GAM} = \frac{\text{GA}}{\bar{X}} \text{ X } 100$$

Where, GA = genetic advance estimated

$\bar{X}$ = mean of the population

The genetic advance as *per cent* of mean was categorized as suggested by Johnson *et al.* (1955) and the same is given below.

Low = (0-10%)

Moderate = 10.1-20%

High = >20%

3.3.4.2 Genotypic data analysis

3.3.4.2.1 Linkage map construction by iMAS (GMendel)

One hundred and ninety nine SNP marker data of 344 $F_{2:3}$ progenies were subjected for linkage map construction. Chi square test was performed on the genotypic data to test the null hypothesis of expected 1:2:1 Mendelian segregation. Of these, five markers showed segregation distortion (SD) and were not used for linkage map

construction. The linkage analysis was performed using GMendel programme of iMAS software. A minimum LOD of 3.0 and maximum recombination fraction of 0.40 were set as threshold values for linkage groups determination. GMendel 2.0 is unique and perform multipoint linkage analysis on populations with complex genetic structures. GMendel 2.0 generates two point maximum likelihood estimates for all pair wise markers. Linkage phases are correctly assigned based on probability rules and gene order is estimated using an advance multipoint mapping algorithm. Multipoint gene order was determined by GMendel 2.0 using a powerful method called the simulated annealing algorithm (SAA). The marker ordering was validated by Monte Carlo and bootstrap methods. Recombination fraction was converted into map distances in centi Morgan (cM) using Haldane mapping function. The intermarker distances calculated from GMendel were used to construct the linkage map.

3.3.4.2.2 QTL location by WinQTL Cartographer version 2.5

The analysis of QTLs controlling the northern corn leaf blight, sorghum downy mildew and southern corn rust resistance was performed on the means of $F_{2:3}$ family replicates for the arcsine transformed data within each season as well as across seasons. The phenotypic data (*Kharif* 2012 and *Kharif* 2013) and genotypic data of 194 SNP markers across 10 chromosomes were subjected to construct linkage map in order to identify the QTLs associated with the trait using WinQTL Cartographer version 2.5 (Wang *et al.*, 2010).

The replicated mean data of 344 F_3 progenies for northern leaf blight, sorghum downy mildew and southern corn rust were used for QTL mapping in each season. To determine the QTLs across the seasons, replicated means of across season means of 344 $F_{2:3}$ progenies were

used. QTL analysis was performed using the composite interval mapping method (CIM) (Zeng, 1994) as in WinQTL Cartographer 2.5.

The presence of putative QTL in an interval was tested by using a critical value for LOD threshold of 2.5 as determined by WQTL Cartographer using the Bonferroni chi-square approximation (Zeng, 1994) corresponding to genome wise type-I error. As the mapping population comprised of $F_{2:3}$, both the additive and dominance model were used for analysis. The point at which the LOD score had the maximum value in the interval was taken as the estimated QTL position. The coefficient of determination also known as coefficient of variance (R^2) explained by the QTL was used as a measure of the magnitude of association and it was estimated as the square of the partial correlation coefficient. Estimates of the additive genetic effect of each detected QTL, the total LOD score, the total proportion of phenotypic variance explained by all the detected QTLs were obtained by fitting a multiple linear regression model that simultaneously included all the detected QTLs for the trait in question. The LOD score was calculated from the F value for the multiple regressions (Haley and Knott, 1992) as

$$LOD=n/n\ (1+p8F/Df)*0.2171$$

Where,

p = number of parameters fitted (Haley and Knott, 1992)

F ratio = SSR (full)-SSR (red)/pMSE (full)

SSR (full) = Sum of square for regression with full model *i.e.* with QTL and cofactors

SS (red) = Sum of square for regression with reduced model *i.e.* without the QTL

MSE (full) = SSE/DEF=Residual mean square (full model)

pMSE = Number of estimated QTL effects

Df = Number of degrees of freedom for residual sum of square in multiple regression

The percentage of phenotypic variance (R^2) explained by a QTL was estimated. This is based on the partial correlation of putative QTL with observed variable, adjusted for cofactors (Kendall and Stuart, 1961). In the simultaneous fit, the cofactors were ignored and only the putative QTLs initially detected and their estimated position were used in multiple regressions to obtain the final estimates of the additive genetic effects and the percentage of phenotypic variation for the particular trait that could be explained by the QTLs. The additive effect was calculated as half the differences between genotypic values of two homozygotes (Falconer, 1981).

Gene action was determined by the ratio of the absolute value of the estimated dominance effect divided by the absolute value of the estimated additive effect |d| / |a| following Stuber *et al.* (1987) (additive = 0 to 0.20; partial dominance = 0.21 to 0.80; dominance = 0.81 to 1.20; and overdominance > 1.20).

3.4 Co – localization of QTLs for multiple foliar diseases of maize

The maize genetic map is divided into 100 segments, called bins, of approximately 20 centiMorgans between two fixed Core Markers (Gardiner *et al.* 1993). The segments are designated with the chromosome number followed by a two-digit decimal (e.g., 1.00, 1.01, 1.02, etc). A bin is the interval that includes all loci from the leftmost or top Core Marker to the next Core Marker (*www.maizegdb.org/bin-*

viewer). The identified QTLs for resistance to three foliar pathogens were co-localized to different bins of the chromosomes.

Pearson correlation coefficients between the means of northern leaf blight, sorghum downy mildew and southern corn rust measured at individual seasons as well as over seasons was calculated to assess degree of association between the foliar diseases of maize.

$$r_p\,(xy) = \frac{Cov_p\,(xy)}{\sqrt{\sigma^2_p\,(x)\,.\,\sigma^2_p\,(y)}}$$

Where,

r_p (xy) = Correlation between 'x' and 'y'

Cov_p (xy) = Covariance between 'x' and 'y'

σ^2_p (x) = Variance of 'x'

σ^2_p (y) = Variance of 'y'

Results

IV. RESULTS

The present investigation was undertaken to know the genetic basis of resistance to northern corn leaf blight and southern corn rust through six generation mean analysis and to map the genomic regions/QTLs conferring resistance to three important foliar diseases *viz.*, northern corn leaf blight, sorghum downy mildew and southern corn rust of maize and the results of the same are presented below.

4.1 Genetic analysis of resistance to northern corn leaf blight and southern corn rust through six generation mean analysis

Higher disease severity of northern corn leaf blight and southern corn rust was achieved through artificial inoculations. The results obtained through six generation mean analysis involving both segregating and non-segregating generations (P_1, P_2, F_1, F_2, B_1 and B_2) of the crosses CM212 × SKV50 and HKI162 × SKV50 of maize are presented under following headings.

4.1.1 Generation means

The mean values along with standard error and variances of six generations (P_1, P_2, F_1, F_2, B_1 and B_2) of the crosses CM212 × SKV50 and HKI162 × SKV50 of maize in respect of northern corn leaf blight and southern corn rust are tabulated in Table 2 and briefly presented below.

Northern corn leaf blight

Wide range of disease severity score was observed among non-segregating populations for northern corn leaf blight and severity score was maximum (3.713) in parent CM212 compared to other parents HKI162 (3.225) and SKV50 (0.888), while the F_1s recorded intermediate northern corn leaf blight score compared to its parents. Among segregating generations, the disease score of B_1 population of both

Table 2: Estimates of means of generations with their standard error, variance and variance of mean for response to northern corn leaf blight and southern corn rust

Parents/Generations		Northern corn leaf blight			Southern corn rust		
		Mean ± SE	Variance	Variance of mean	Mean ± SE	Variance	Variance of mean
Parents (40)	CM212	3.713 ± 0.103	0.422	0.0105	3.288 ± 0.069	0.191	0.0048
	HKI162	3.225 ± 0.074	0.217	0.0054	3.062 ± 0.082	0.272	0.0068
	SKV50	0.888 ± 0.066	0.173	0.0043	0.475 ± 0.069	0.192	0.0048
F_1^s (40)	CM212 × SKV50	2.488 ± 0.110	0.481	0.0120	2.075 ± 0.077	0.238	0.0059
	HKI162 × SKV50	2.425 ± 0.092	0.340	0.0085	1.862 ± 0.064	0.166	0.0042
F_2^s (200)	CM212 × SKV50	2.403 ± 0.070	0.969	0.0048	2.550 ± 0.062	0.759	0.0038
	HKI162 × SKV50	2.715 ± 0.066	0.858	0.0043	2.337 ± 0.059	0.701	0.0035
B1 (100)	CM212 × (CM212 × SKV50)	3.335 ± 0.084	0.702	0.0070	3.030 ± 0.053	0.282	0.0028
	HKI162 × (HKI162 × SKV50)	3.035 ± 0.071	0.501	0.0050	2.510 ± 0.051	0.257	0.0026
B2 (100)	SKV50 × (CM212 × SKV50)	1.975 ± 0.057	0.325	0.0033	1.750 ± 0.057	0.326	0.0033
	SKV50 × (HKI162 × SKV50)	1.690 ± 0.081	0.650	0.0065	1.350 ± 0.070	0.492	0.0049

Figures in parenthesis indicate sample size

crosses CM212 × SKV50 and HKI162 × SKV50 (3.335 and 3.035, respectively) was maximum compared to either B_2 (1.975 and 1.690, respectively) or F_2 (2.403 and 2.715, respectively) populations.

Southern corn rust

Among parents, CM212 and HKI162 recorded the highest disease score of 3.288 and 3.062, respectively compared to the parent SKV50 which recorded the lowest mean disease score of 0.475. Whereas, the mean of F_1s CM212 × SKV50 (2.075) and HKI162 × SKV50 (1.862) was within the limits of parents in respect of this trait. However, the mean disease score of B_1 population of crosses CM212 × SKV50 (3.030) and HKI162 × SKV50 (2.510), and F_2 populations (2.550 and 2.337, respectively) was relatively higher than B_2 population mean (1.750 and 1.350, respectively) of both crosses CM212 × SKV50 and HKI162 × SKV50.

4.1.2 Scaling tests and joint scaling tests

The mean and variance of six generations viz., P_1, P_2, F_1, F_2, B_1 and B_2 of the crosses CM212 × SKV50 and HKI162 × SKV50 in respect of northern corn leaf blight and southern corn rust disease score were subjected to scaling tests as per the method of Mather (1949) and Joint scaling test as per the method of Cavalli (1952) to test the presence or absence of gene interaction in the inheritance of these traits. The significance of 'A' and 'B' scales indicates the presence of all the three types of non-allelic interactions viz., additive × additive [$\hat{i}$], additive × dominance [$\hat{j}$] and dominance × dominance [$\hat{l}$]. The significance of 'C' scale suggest the importance of dominance × dominance [$\hat{l}$] type of interaction. The significance of any one of the scaling tests indicates inadequacy of simple additive-dominance model. The results of scaling tests and joint scaling tests for Northern corn leaf blight and southern

Table 3: Estimates of scaling tests for northern corn leaf blight and southern corn rust scores of different generations

Cross	Scaling Test	Northern corn leaf blight	Southern corn rust
CM212 × SKV50	**A**	0.470* ± 0.225	0.698** ± 0.148
	B	0.275 ± 0.176	0.662** ± 0.161
	C	-0.265 ± 0.377	2.000** ± 0.310
	D	-0.505** ± 0.172	0.320* ± 0.146
HKI162 × SKV50	**A**	0.420* ± 0.184	0.095 ± 0.146
	B	0.068 ± 0.197	0.363* ± 0.169
	C	1.898** ± 0.335	2.088** ± 0.290
	D	0.705** ± 0.169	0.815** ± 0.147

**Significant at 5% level of significance*

***Significant at 1% level of significance*

corn rust disease score are presented in Table 3 and Table 4, respectively and presented below.

Northern corn leaf blight

The scaling tests 'A' and 'D' were significant in the cross CM212 × SKV50, whereas tests 'A', 'C' and 'D' were significant in the cross HKI162 × SKV50, indicating the inadequacy of additive-dominance model, which was further confirmed by significance of additive and dominance components in joint scaling test. This clearly indicated presence of digenic or still higher order epistasis in the inheritance of this trait.

Southern corn rust

Simple additive-dominance model failed to explain the inheritance of southern corn rust as indicated by the significance of all four scaling tests in the cross CM212 × SKV50, whereas in the cross HKI162 × SKV50, the scaling tests 'B', 'C' and 'D' were significant, which was further confirmed by high level of significance of additive and dominance components in the joint scaling test. This necessitated the extension of additive- dominance model to include digenic interaction or epistasis in the inheritance of this trait.

4.1.3 Estimation of gene effects under inadequacy of additive-dominance model

After ascertaining the failure of additive-dominance model in explaining the inheritance of various quantitative characters, the perfect fit six-parameter estimates of digenic interaction model were estimated following the methods of Jinks and Jones (1958) and Mather and Jinks (1971). The six generations of the crosses CM212 × SKV50 and HKI162 × SKV50 were used to estimate the gene effects *viz.*, $[\hat{m}]$, $[\hat{d}]$, $[\hat{h}]$, $[\hat{i}]$, $[\hat{j}]$ and $[\hat{l}]$ for northern corn leaf blight and southern corn rust scores. The

Table 4: Estimates of components of generation means and test for adequacy of additive-dominance model for inheritance of northern leaf blight and southern corn rust

Disease	Cross	[m]	[$\hat{d}$]	[$\hat{h}$]
Northern corn leaf blight	**CM212 × SKV50**	2.486** ± 0.058	1.273** ± 0.054	0.105 ± 0.114
	HKI162 × SKV50	2.132** ± 0.046	1.205** ± 0.045	0.547** ± 0.094
Southern corn rust	**CM212 × SKV50**	2.195** ± 0.048	1.250** ± 0.045	0.230** ± 0.090
	HKI162 × SKV50	1.880** ± 0.046	1.263** ± 0.045	0.165* ± 0.083

**Significant at 5% level of significance* ***Significant at 1% level of significance*

Table 5: Estimates of components of generation means based on perfect fit solution (Joint Scaling Test) for northern corn leaf blight and southern corn rust of maize

Disease	Cross	[m]	[$\hat{d}$]	[$\hat{h}$]	[$\hat{i}$]	[$\hat{j}$]	[$\hat{l}$]	Type of Epistasis
Northern corn leaf blight	**CM212 × SKV50**	1.440** ± 0.350	1.263** ± 0.064	2.803** ± 0.854	1.010** ± 0.344	0.195 ± 0.240	-1.755** ± 0.554	Duplicate
	HKI162 × SKV50	3.466** ± 0.342	1.169** ± 0.049	-1.964** ± 0.848	-1.410** ± 0.339	0.353 ± 0.236	0.922 ± 0.545	Duplicate
Southern corn rust	**CM212 × SKV50**	2.665** ± 0.297	1.262** ± 0.054	0.130 ± 703	-0.640* ± 0.292	0.035 ± 0.190	-0.720 ± 0.440	Duplicate
	HKI162 × SKV50	3.399** ± 0.298	1.294** ± 0.054	-2.709** ± 0.724	-1.630** ± 0.293	-0.268 ± 0.204	1.173** 0.452	Duplicate

**Significant at 5% level of significance* ***Significant at 1% level of significance*

gene effects estimated by using perfect fit model in respect of disease score are tabulated in Table 5 and results are presented below.

Northern corn leaf blight

In the cross CM212 × SKV50, the estimates of additive [$\hat{d}$] and dominance [$\hat{h}$] gene effects were significant. The magnitude of dominance [$\hat{h}$] effect was more compared to additive gene effect. Among interaction components, additive × additive [$\hat{i}$] interaction was significant in positive direction, whereas dominance × dominance [$\hat{l}$] interaction was significant in negative direction. In the cross HKI162 × SKV50, additive gene effect [$\hat{d}$] was significant in positive direction and dominance [$\hat{h}$] gene effect was significant in the negative direction, while among interaction components, only additive × additive gene effect [$\hat{i}$] was significant in negative direction.

Opposite signs of [$\hat{h}$] and [$\hat{l}$] components indicated the presence of duplicate gene interaction in the genetic control of Northern corn leaf blight in both crosses CM212 × SKV50 and HKI162 × SKV50.

Southern corn rust

It is evident from six parameter model that only additive [$\hat{d}$] component was significant and additive × additive gene interaction effect [$\hat{i}$] was significant in the negative direction in the cross CM212 × SKV50. Whereas in cross HKI162 × SKV50, both [$\hat{d}$] and [$\hat{h}$] gene effects were significant in the positive and negative direction, respectively. Among interactions, additive × additive [$\hat{i}$] type of interaction was negative and significant while dominance × dominance [$\hat{l}$] type was negative and significant.

Duplicate type of epistasis was observed to be operating in both crosses CM212 × SKV50 and HKI162 × SKV50 as evidenced from the estimates of [$\hat{h}$] and [$\hat{l}$] which had opposite signs.

4.1.4 Number of effective factors and potence ratio

Minimum number of effective factors influencing a trait in F_2 population and the degree of dominance computed from generation means of F_1 as well as F_2 generations in terms of potence ratio for two crosses CM212 × SKV50 and HKI162 × SKV50 of maize for northern corn leaf blight and southern corn rust was estimated and results of the same are tabulated in Table 6 and briefly presented below.

Northern corn leaf blight

Northern corn leaf blight appeared to be governed by at least two groups (2.06) of effective factors in the cross CM212 × SKV50 whereas in the cross HKI162 × SKV50 it was controlled by one group (1.38) of effective factors.

In F_1 generation of both crosses CM212 × SKV50 and HKI162 × SKV50, the degree of dominance in terms of potence ratio (h_1) was between zero and unity for northern corn leaf blight (0.13 and 0.32, respectively) indicating either no dominance or partial dominance in the genetic control of the trait. Whereas, in F_2 generation of CM212 × SKV50, the potence ratio (h_2) was less than unity for northern corn leaf blight which indicated the presence of partial dominance, while the trait was under over dominance as their potence ratio (h_2) value was more than unity in F_2 of the cross HKI162 × SKV50.

Table 6: Number of effective factors in the genetic control of northern corn leaf blight and southern corn rust in F_2 generation and potence ratio (in F_1 and F_2) of two crosses in Maize

Disease	Number of effective factors (N) in F_2		Potence ratio in F_1		Potence ratio in F_2	
	CM212 × SKV50	HKI162 × SKV50	CM212 × SKV50	HKI162 × SKV50	CM212 × SKV50	HKI162 × SKV50
Northern corn leaf blight	2.06	1.38	0.13	0.32	0.15	1.13
Southern corn rust	1.92	1.57	0.14	0.07	0.95	0.88

Southern corn rust

In both crosses CM212 × SKV50 and HKI162 × SKV50, at least two groups (1.92 and 1.57, respectively) of effective factors appeared to control the inheritance of southern corn rust.

Potence ratio (h_1 and h_2) in F_1 generation (0.14 and 0.07, respectively) and F_2 generation (0.95 and 0.88, respectively) of both crosses CM212 × SKV50 and HKI162 × SKV50 was less than unity indicating the partial dominance for southern corn rust.

4.2 Identification of QTLs for northern corn leaf blight, sorghum downy mildew and southern corn rust diseases

4.2.1 Phenotypic data analysis of $F_{2:3}$ mapping population derived from the cross CML153 × SKV50

Weather conditions at Mandya favoured the development of severe northern corn leaf blight, sorghum downy mildew and southern corn rust epidemics. The 344 $F_{2:3}$ families along with parents, resistant and susceptible checks were evaluated for northern corn leaf blight, sorghum downy mildew and southern corn rust incidence under artificial epiphytotic condition. The phenotypic data was recorded as percentage of leaf area showing disease symptoms as in case of northern corn leaf blight and southern corn rust and expressed as *per cent* disease severity, while sorghum downy mildew data were recorded as percentage of plants showing disease symptoms and expressed as *per cent* disease index. In *Kharif* 2012, the expression of southern corn rust and sorghum downy mildew disease among the $F_{2:3}$ progenies was not uniform, so we excluded data pertaining to *Kharif* 2012 for those two diseases from further analysis.

The *per cent* disease severity and *per cent* disease index values for *Kharif* 2012 and *Kharif* 2013 ranged from zero to hundred *per cent* which

followed binomial distribution. Data were transformed using arcsine transformation as described by Gomez and Gomez (1984). The transformation of data was envisioned to make the means and variances as independent and normally distributed. The arcsine transformed *per cent* disease data of 344 $F_{2:3}$ progenies (Appendix 3) for both seasons *Kharif* 2012 and *Kharif* 2013 in respect of northern corn leaf blight and *Kharif* 2013 in respect of sorghum downy mildew and southern corn rust, were used for statistical analysis and QTL mapping.

4.2.2 Analysis of variance in the $F_{2:3}$ progenies during *kharif*-2012 and *kharif*-2013

Analysis of variance was conducted on arcsine transformed values of the phenotypic data for individual seasons and combined over two seasons using PROC GLM procedure of SAS package version 9.3. The components of variance for the $F_{2:3}$ families in both the seasons and across the seasons were computed considering all effects in the statistical model as random.

Analysis of variance for incidence of three foliar diseases is given in Table 7 for *Kharif* 2012 and *Kharif* 2013. The analysis of variance revealed significant differences among the progenies indicating the presence of genetic variability in the F_3 progenies.

Before pooling Northern corn leaf blight data across seasons, Bartlett's chi square test was performed to test the homogeneity of error mean sum of squares between the two seasons and results are presented in the Table 8. Non-significant Bartlett's chi square indicated the homogeneity of error mean sum of squares between the *Kharif* 2012 and *Kharif* 2013 seasons.

Pooled ANOVA of $F_{2:3}$ families for northern corn leaf blight over two seasons is presented in Table 9. Variance due to genotype and genotype

Table 7: Analysis of variance of $F_{2:3}$ mapping population for foliar pathogen incidence during *Kharif*, 2012 and Kharif, 2013 in Mandya

Source	df	Mean Sum of Squares			
		Northern corn leaf blight		Sorghum downy mildew	Southern corn rust
		Kharif 2012	Kharif 2013	Kharif 2013	Kharif 2013
Replication	1	3168.12	51.27	2125.58	3330.78
Genotypes	343	114.93**	80.75**	323.62**	298.20**
Error	343	29.8	31.66	30.78	10.02

Table 8: Homogeneity test for northern corn leaf blight incidence during *Kharif*, 2012 and *Kharif*, 2013

Combination	Northern corn leaf blight	
	Chi - Square	Pr > ChiSq
***Kharif*, 2012 × *Kharif*, 2013**	15.530	<0.0932

Table 9: Pooled analysis of variance in the $F_{2:3}$ mapping population evaluated for northern corn leaf blight incidence

Source	df	Mean Sum of Squares
		Northern corn leaf blight
Seasons	1	8414.91**
Genotypes	343	59.43**
Genotypes × Seasons	343	38.41**
Error	686	12.30

× season interaction was significant indicating that among F_3 families expression of disease incidence significantly varied and disease incidence depends upon testing season.

4.2.2 Descriptive statistics

The descriptive statistics *viz.*, mean, range, variance, skewness (S) and kurtosis (K), test for normality with respect to multiple foliar pathogen incidence are briefly presented below.

Estimation of mean and range

The mean and range values of northern corn leaf blight, sorghum downy mildew and southern corn rust incidence in $F_{2:3}$ generation and its parents are presented in Table 10. Tests for significance of parental means indicated that parents CML153 and SKV50 differed significantly in their reaction to multiple foliar diseases (Plate 2) as indicated by their *per cent* disease incidence. The parent SKV50 showed resistance to northern corn leaf blight (5 and 7 *per cent* incidence in *Kharif* 2012 and *Kharif* 2013), sorghum downy mildew (1.50 *per cent* incidence in *Kharif* 2013) and southern corn rust (0.00 *per cent* incidence in *Kharif* 2013). The other parent (CML153) was susceptible to northern corn leaf blight (66.0 and 75.0 *per cent* incidence in *Kharif* 2012 and *Kharif* 2013), sorghum downy mildew (91.50 *per cent*) and southern corn rust (73.66 *per cent*) in *Kharif* 2013.

In F_3 progenies, mean northern corn leaf blight disease incidence was 52.38 *per cent* in *Kharif* -2012 and 40.71 *per cent* in *Kharif* 2013, and 46.55 *per cent* when it was pooled over seasons. Maximum range of disease incidence (17.14 to 84.29%) was recorded in *Kharif* -2012 followed by *Kharif* 2013 (16.00 to 74.00 %), while pooled northern corn leaf blight incidence ranged from 26.50 to 69.25 *per cent*. During *Kharif* 2013, mean sorghum downy mildew and southern corn rust incidence

Table 10: Estimates of mean, range of parents and 344 $F_{2:3}$ population of maize for pathogen incidence during *Kharif*, 2012 and *Kharif*, 2013

Parameters		Disease Incidence			
	Sorghum downy mildew	Northern corn leaf blight			Southern corn rust
	Kharif, 2013	*Kharif*, 2012	*Kharif*, 2013	Pooled	*Kharif*, 2013
CML153 (P_1)	91.50 (73.02)	66.00 (54.31)	75.00 (59.98)	70.50 (57.14)	73.66 (59.10)
SKV50 (P_2)	1.50 (7.03)	5.00 (12.92)	7.00 (15.34)	6.00 (14.13)	0.00 (0.00)
P1 v/s. P_2 (Pr<t)	<0.0009	<0.0036	<0.0028	<0.0031	<0.0012
F_3 Grand Mean	55.36	52.38	40.71	46.55	30.54
F_3 Range	0.00 - 91.50	17.14 - 84.29	16.00 - 74.00	26.50 - 69.25	0.00 - 80.00

Note: Values in parenthesis are arcsine transformed values

Resistant inbred (SKV50) **Susceptible inbred (CML153)**

Plate 2. Multiple disease resistant (SKV50) and susceptible (CML153) parents used for the development of $F_{2:3}$ mapping population (NCLB – Northern corn leaf blight, SDM – Sorghum downy mildew and SCR – Southern corn rust)

Table 11: Test for normality, skewness and kurtosis for incidence of three foliar diseases in $F_{2:3}$ population of maize

Character	Disease Incidence				
	Sorghum downy mildew	Northern corn leaf blight			Southern corn rust
	Kharif, 2013	*Kharif*, 2012	*Kharif*, 2013	Pooled	*Kharif*, 2013
Skewness	0.087	0.095	0.188	0.125	0.143
Kurtosis	-0.246	-0.397	-0.101	-0.362	-0.201
KS test (D)	0.061	0.08	0.063	0.072	0.062
Pr>D	<0.01	<0.01	<0.01	<0.01	<0.01

was 55.36 and 30.54 *per cent*, respectively in F_3 progenies. Wide range of sorghum downy mildew incidence (0.00 to 91.50 *per cent*) and southern corn rust incidence (0.00 to 80.00 *per cent*) was recorded in F_3 progenies.

Test for normality, skewness and kurtosis

The frequency distribution pattern of F_3 families for Northern corn leaf blight was positively skewed and platykutic in *Kharif* 2012, *Kharif* 2013 and when pooled over seasons. The frequency distribution for sorghum downy mildew and southern corn rust incidence in F_3 progenies during *Kharif* 2013 was also found to be platykurtic and skewed (tabulated in Table 11 and depicted in Fig. 1, 2 and 3). The expression of northern corn leaf blight, sorghum downy mildew and southern corn rust appeared to be controlled by larger number of genes having decreasing effects and involvement of dominance based complementary interaction as evidenced by platykurtic and positively skewed distribution.

The results of test for normality by Kolmogorov-Smirnov goodness of fit test are presented in Table 11. According to Kolmogorov-Smirnov goodness of fit test, the distribution of phenotypic means, within and across the seasons deviated significantly from normal distribution with the majority of the progenies skewed towards resistance in northern corn leaf blight and southern corn rust and towards susceptibility in sorghum downy mildew.

4.2.3 Genetic variability studies in $F_{2:3}$ population of maize

Genetic variability parameters *viz.*, phenotypic variance, genotypic variance, phenotypic co-efficient of variation (PCV), genotypic co-efficient of variation (GCV), narrow sense heritability (h^2) and genetic advance as *per cent* of mean (GAM) were estimated using *per cent* disease incidence data and are presented in Table 12 and briefly outlined below.

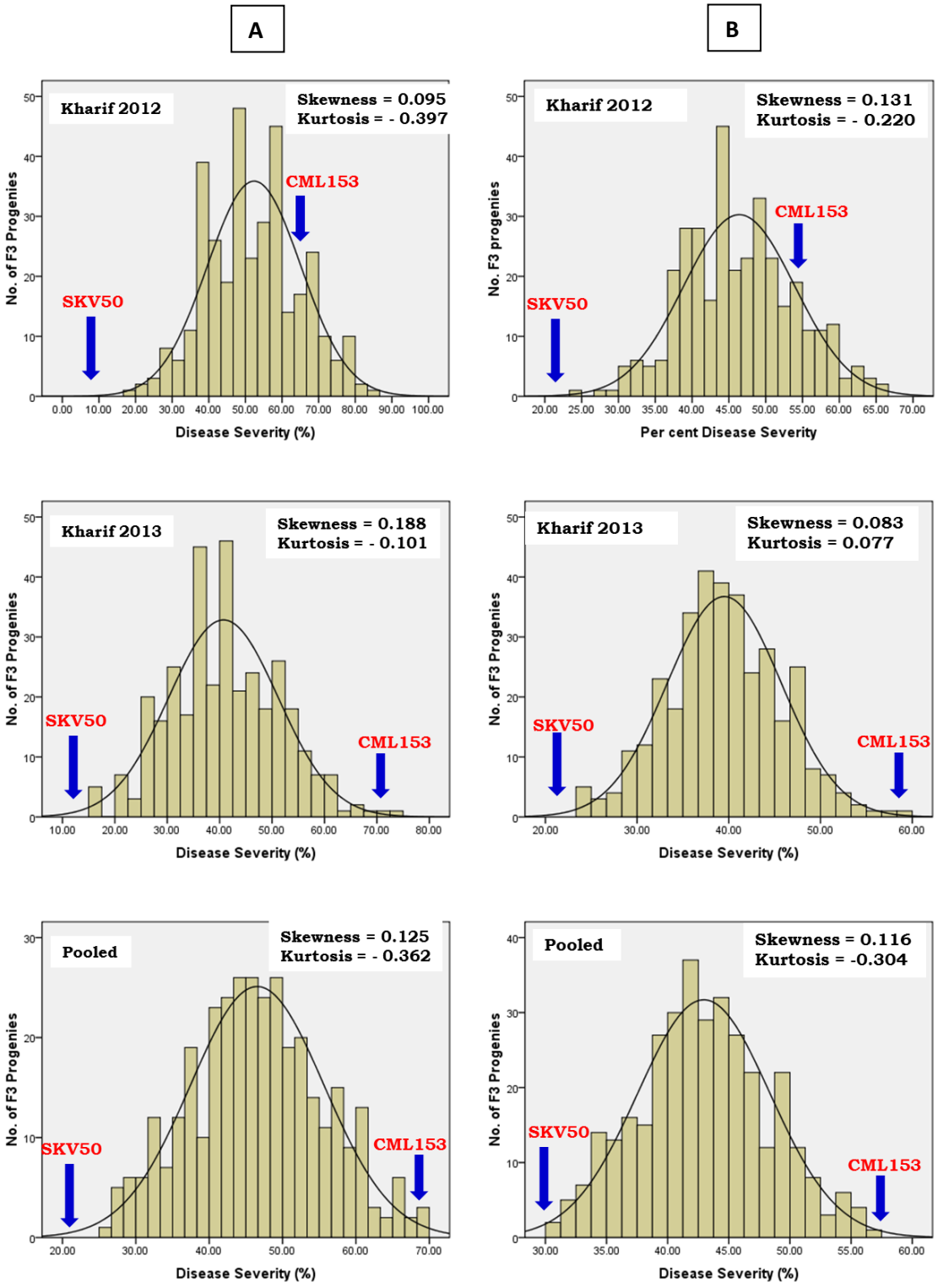


Fig. 1. Frequency distribution of mean *per cent* disease severity of northern corn leaf blight in the $F_{2:3}$ population derived from the cross CML153 × SKV50 (A=original; B= Arcsine transformed)

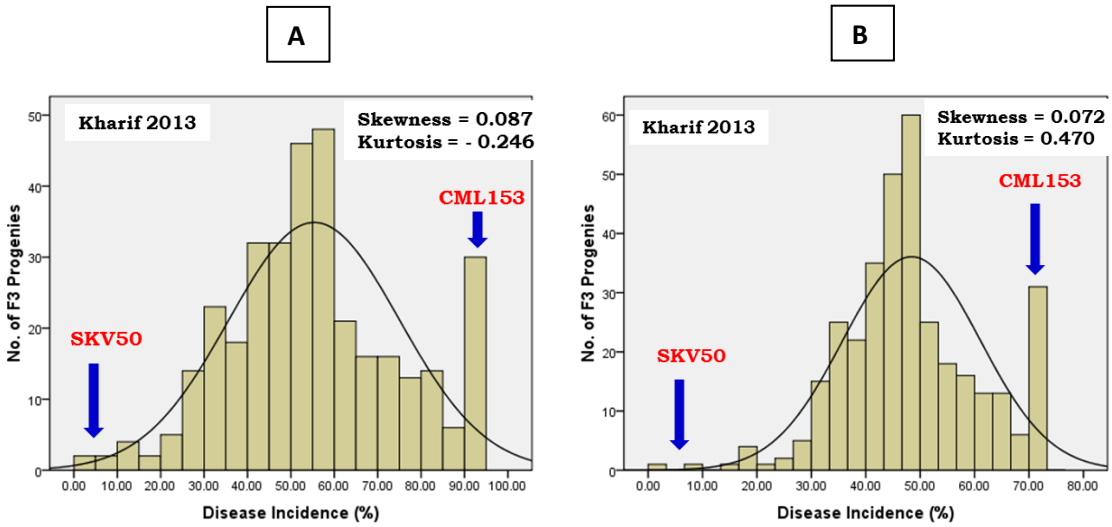


Fig. 2. Frequency distribution of mean *per cent* disease incidence of sorghum downy mildew in the $F_{2:3}$ population from the cross CML153 × SKV50 (A=original; B= Arcsine transformed)

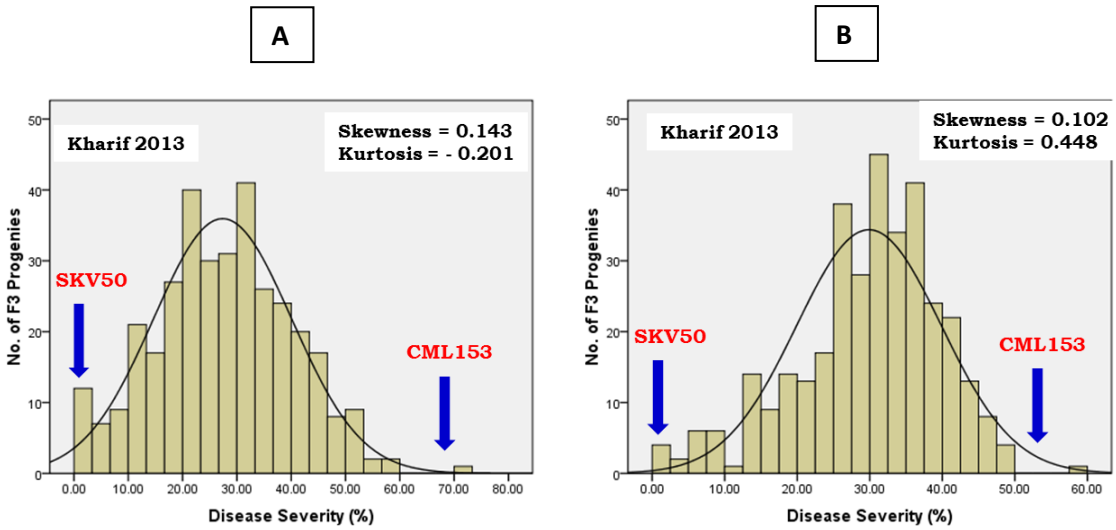


Fig. 3. Frequency distribution of mean *per cent* disease severity of southern corn rust in the $F_{2:3}$ population from the cross CML153 × SKV50 (A=original; B= Arcsine transformed)

Table 12: Estimates of genetic parameters in F2:3 mapping population for incidence of three foliar diseases

Genetic parameters	Multiple foliar disease incidence				
	Sorghum downy mildew	Northern corn leaf blight			Southern corn rust
	Kharif, 2013	*Kharif*, 2012	*Kharif*, 2013	Pooled	*Kharif*, 2013
Genotypic variance (Vg)	146.42	42.56	24.54	23.57	144.09
Phenotypic variance (Vp)	177.20	72.36	56.20	35.86	154.11
Genotypic coefficient of variation (GCV) (%)	21.86	12.46	12.17	10.43	39.30
Phenotypic coefficient of variation (PCV) (%)	24.05	16.24	18.42	12.86	40.65
Heritability (broad sense) (%)	90.90	58.82	43.67	65.72	93.50
Genetic advance as per cent Mean (GAM) (%)	45.03	19.68	16.57	17.42	78.29

The estimates of PCV and GCV values for northern corn leaf blight in *Kharif* 2013 (18.42% and 16.24%) were on par with the PCV and GCV values in *Kharif* 2012 (16.24% and 12.46%, respectively). For pooled data, estimates of PCV and GCV were 12.86 and 10.43 *per cent*, respectively. Moderate heritability and GAM were noticed in *Kharif* 2012 (58.82% and 19.68%, respectively) and *Kharif* 2013 (43.67% and 16.57%, respectively), whereas in pooled data heritability value was 65.72 *per cent* and GAM was 17.42 *per cent*.

During *Kharif* 2013, PCV and GCV values were high for sorghum downy mildew (24.05% and 21.86%, respectively). Broad sense heritability was high (90.90%) coupled with high (45.03%) genetic advance expressed as *per cent* of mean indicating major role of additive gene action in the genetic control of this character.

Southern corn rust registered relatively higher values of PCV (40.65%) and GCV (39.30%) in *Kharif* 2013. However, the broad sense heritability estimate was high (90.91%) coupled with higher (78.29%) genetic advance as *per cent* of mean indicating major role of additive gene action in the genetic control of the trait.

4.3 Construction of linkage map

4.3.1 Parental polymorphism survey using SNPs

SNP genotyping of the resistant inbred SKV50 and susceptible inbred CML153 using 765 SNP markers was done for assaying the parental polymorphism. The *per cent* polymorphism between the two parents used for developing mapping population as revealed by SNP markers was 25.35 *per cent* (Table 13).

A total of 765 SNPs were used for genotyping the $F_{2:3}$ progenies along with parents (CML153 and SKV50). Among these markers, only 194 polymorphic SNP (Table 14) markers showed high polymorphism

Table 13: Number of polymorphic SNP markers assigned to each chromosome and their average distances

Chromosome	Polymorphic SNP markers	Length (cM)	Average distances (cM)	
Ch1	33	308.76	9.36	**Parental Polymorphism (%) = 25.35**
Ch2	19	234.07	12.32	
Ch3	12	248.13	20.68	
Ch4	26	237.51	9.14	
Ch5	28	216.27	7.72	
Ch6	19	179.75	9.46	
Ch7	6	205.29	34.22	
Ch8	24	199.45	8.31	
Ch9	12	152.25	12.69	
Ch10	15	161.54	10.77	
Total	**194**	**2143.02**	**10.77**	

Table 14: List of polymorphic SNP markers used for genotyping $F_{2:3}$ mapping population

Sl No.	SNP Marker Name	Chromosome	Distance	Sl No.	SNP Marker Name	Chromosome	Distance
1	C00365-01	1	39.47	51	C00271-01	2	211.83
2	C00304-01	1	40.01	52	C00214-01	2	234.07
3	MZA1653-31	1	52.70	53	C00278-01	3	42.31
4	MZA4531-46	1	68.63	54	C00327-01	3	77.49
5	MZA18887-12	1	76.93	55	MZA5502-31	3	79.23
6	MZA3951-25	1	84.39	56	MZA7417-21	3	79.28
7	C00260-01	1	88.02	57	MZA1959-26	3	114.44
8	MZA2177-85	1	94.92	58	MZA2919-23	3	172.71
9	C00252-01	1	111.47	59	C00146-01	3	183.90
10	C00288-01	1	113.65	60	MZA8828-7	3	185.88
11	C00186-01	1	120.49	61	MZA2204-96	3	226.71
12	MZA5306-16	1	122.39	62	C00341-01	3	228.00
13	C00159-01	1	134.91	63	MZA2423-33	3	242.86
14	MZA1932-51	1	136.59	64	MZA617-71	3	248.13
15	C00145-01	1	137.70	65	MZA1971-20	4	8.01
16	MZA2187-46	1	139.83	66	MZA3963-33	4	36.57
17	MZA3147-18	1	151.60	67	MZA2518-28	4	58.89
18	MZA12706-14	1	191.11	68	MZA687-25	4	63.56
19	C00121-01	1	198.53	69	MZA259-11	4	63.65
20	MZA4942-12	1	205.62	70	MZA8527-2	4	68.86
21	C00351-01	1	207.01	71	C00213-01	4	72.70
22	C00100-01	1	216.98	72	MZA5572-19	4	91.27
23	MZA4992-10	1	217.73	73	MZA3587-6	4	103.00
24	C00089-01	1	225.25	74	C00218-01	4	103.00
25	MZA3034-3	1	234.76	75	C00170-01	4	105.09
26	C00295-01	1	252.14	76	C00122-01	4	116.62
27	C00088-01	1	252.62	77	MZA3155-14	4	140.61
28	C00190-01	1	273.41	78	C00362-01	4	146.12
29	C00129-01	1	281.80	79	MZA2006-57	4	147.84
30	C00315-01	1	294.42	80	C00205-01	4	177.94
31	C00212-01	1	298.10	81	MZA1684-20	4	183.57
32	MZA673-33	1	307.76	82	MZA4117-14	4	184.88
33	C00322-01	1	308.76	83	MZA5519-25	4	189.03
34	MZA1511-14	2	16.81	84	MZA5137-12	4	189.26
35	MZA13440-11	2	17.05	85	MZA9804-28	4	197.14
36	C00375-01	2	74.84	86	MZA5599-20	4	201.25
37	MZA6111-5	2	93.05	87	MZA5665-26	4	207.05
38	MZA7964-27	2	94.40	88	MZA5665-10	4	207.05
39	C00359-01	2	104.85	89	MZA13084-4	4	225.12
40	MZA13360-13	2	129.91	90	C00199-01	4	237.51
41	MZA3626-3	2	130.96	91	MZA662-27	5	2.82
42	C00255-01	2	131.56	92	C00215-01	5	5.00
43	C00115-01	2	139.75	93	C00116-01	5	5.91
44	MZA635-23	2	141.65	94	MZA5359-10	5	21.43
45	C00324-01	2	142.36	95	MZA3137-17	5	50.05
46	MZA3668-12	2	164.36	96	MZA3103-47	5	53.13
47	MZA7953-11	2	166.63	97	MZA533-46	5	53.98
48	MZA16125-47	2	170.93	98	C00096-01	5	63.97
49	C00175-01	2	171.11	99	C00256-01	5	70.56
50	MZA14412-4	2	173.36	100	MZA565-31	5	81.21

Table 14. Con't...

Sl No.	SNP Marker Name	Chromosome	Distance	Sl No.	SNP Marker Name	Chromosome	Distance
101	MZA9009-13	5	82.53	151	MZA191-18	8	85.40
102	MZA4647-8	5	83.61	152	C00142-01	8	87.12
103	MZA3691-18	5	86.77	153	C00135-01	8	88.70
104	C00095-01	5	87.07	154	C00369-01	8	88.93
105	C00182-01	5	92.49	155	MZA5395-34	8	89.94
106	MZA1870-20	5	93.16	156	C00098-01	8	97.60
107	C00092-01	5	94.33	157	MZA4134-8	8	107.66
108	MZA5798-39	5	96.73	158	MZA5805-19	8	114.07
109	C00358-01	5	102.75	159	C00183-01	8	121.74
110	C00367-01	5	114.03	160	C00140-01	8	127.10
111	MZA6910-187	5	117.64	161	MZA4677-11	8	131.93
112	C00303-01	5	126.65	162	MZA15278-6	8	132.63
113	MZA5296-6	5	141.54	163	C00193-01	8	132.95
114	C00171-01	5	150.49	164	C00299-01	8	148.99
115	MZA6386-11	5	151.24	165	MZA4786-9	8	157.98
116	MZA3512-186	5	176.96	166	MZA765-24	8	168.12
117	C00110-01	5	215.20	167	MZA5019-59	8	199.45
118	C00091-01	5	216.27	168	MZA5181-10	9	51.55
119	MZA15961-13	6	17.88	169	MZA4720-12	9	56.34
120	C00221-01	6	21.29	170	C00090-01	9	76.23
121	C00381-01	6	23.52	171	C00280-01	9	98.09
122	C00119-01	6	24.53	172	C00349-01	9	100.00
123	MZA1572-17	6	25.59	173	MZA7916-4	9	108.02
124	C00178-01	6	28.49	174	MZA1766-1	9	119.06
125	MZA13020-10	6	75.31	175	MZA816-25	9	138.18
126	MZA4904-16	6	76.71	176	MZA816-29	9	138.18
127	C00117-01	6	88.11	177	C00188-01	9	140.59
128	MZA3590-19	6	90.64	178	C00225-01	9	141.15
129	C00308-01	6	92.87	179	MZA4303-16	9	152.25
130	MZA11985-27	6	97.24	180	MZA3765-7	10	47.43
131	C00243-01	6	125.00	181	MZA1752-36	10	51.12
132	MZA4662-153	6	125.12	182	MZA15331-16	10	53.56
133	MZA5794-13	6	131.63	183	MZA3922-32	10	58.61
134	MZA4503-25	6	146.13	184	C00229-01	10	61.19
135	C00181-01	6	146.69	185	MZA1155-14	10	61.70
136	MZA5361-13	6	176.81	186	C00314-01	10	62.03
137	MZA5529-4	6	179.75	187	C00152-01	10	67.14
138	C00197-01	7	20.96	188	C00086-01	10	71.34
139	C00347-01	7	56.77	189	C00137-01	10	72.36
140	C00237-01	7	64.45	190	MZA13687-14	10	75.78
141	MZA4080-15	7	68.06	191	C00305-01	10	128.40
142	MZA9162-135	7	117.42	192	MZA5435-25	10	129.12
143	MZA5232-11	7	205.29	193	MZA3844-14	10	147.93
144	MZA2487-6	8	45.78	194	MZA1506-18	10	161.54
145	MZA5637-15	8	51.03				
146	MZA1447-89	8	60.46				
147	MZA1978-111	8	73.87				
148	MZA6428-11	8	75.53				
149	MZA3856-10	8	84.38				
150	MZA191-12	8	85.40				

without ambiguity between CML153 and SKV50 which were then included in the QTL analysis. About 6 families were removed from the analysis due to markers inconsistency likely due to either contamination in genotyped samples, or errors in reading. Banding pattern of 344 $F_{2:3}$ individuals was scored as A (CML153 allele type), B (SKV50 allele type), H (hybrid type) and (-) for missing data.

4.3.2 Genetic linkage mapping

The chi-square (X^2) test was carried out to test the Mendelian segregation ratio (expected 1:2:1) for the polymorphic markers which were used to genotype $F_{2:3}$ mapping population. Out of 765 SNP markers, 194 SNP markers were found to be polymorphic and were segregating in Mendelian fashion and were used for linkage analysis and linkage map construction employing GMendel programme of iMAS software version 2.0 assuming Haldane's mapping function (Haldane, 1919). Markers were assigned to linkage groups at logarithm of odds (LOD) 3.0 with a maximum recombination fraction of 0.40. A total of 194 SNP markers were mapped on 10 linkage groups (LGs) spanning 2143.02 cM. The number of markers mapped per linkage group ranged from 6 (LG7) to 33 (LG1). The length of linkage groups was ranged from 152.25 cM (LG9) to 308.76 cM (LG1) with an average interval distance of 10.77 cM indicating comparatively high density SNP linkage map (Table 13). Each of the linkage group could be assigned to one of the ten maize chromosomes. The linkage map constructed on $F_{2:3}$ mapping population of CML153 × SKV50 was used for identification and mapping of QTLs conferring resistance to three foliar pathogens.

4.4 QTL analysis

To identify QTLs for resistance to northern corn leaf blight, sorghum downy mildew and southern corn rust, we used the arcsine transformed phenotypic data on disease incidence from $F_{2:3}$ mapping

population of the cross CML153 × SKV50 along with the genetic linkage map constructed using SNP markers.

4.4.1 QTLs for northern corn leaf blight resistance

For resistance to northern corn leaf blight, QTLs were detected using the disease incidence data from *Kharif* 2012, *Kharif* 2013 and pooled data over two seasons and the results of the same are tabulated in Table 15 and in Fig. 4 – 5 and results are briefly presented below.

***Kharif* -2012**

Three QTL positions were identified for northern corn leaf blight resistance during *Kharif* 2012. One QTL was located on chromosome 2 (*qNCLB-2*) flanked by markers C00359-01 – MZA13360-13 which explained 3.07 *per cent* phenotypic variation with LOD of 3.06. Two QTLs were located on chromosome 8 (*qNCLB-8-1 and qNCLB-8-2)* flanked by markers *viz.,* MZA2487-6 and MZA6428-11 – MZA3856-10 and these two QTLs showed phenotypic variation of 2.46 and 22.97 *per cent* with LOD score of 2.77 and 3.44, respectively. A major northern corn leaf blight QTL was mapped on chromosome 8 (*qNCLB-8-2)* which explained maximum phenotypic variation of 22.97 *per cent*. These three identified QTLs explained a total of 28.50 *per cent* phenotypic variation in *Kharif* season of 2012.

Additive gene effects of these three QTLs ranged from 1.71 to 1.92 and the favorable alleles for these QTLs were contributed by resistant parent SKV50. In *Kharif* season, QTLs located on chromosome 2 (*qNCLB-2*) and on chromosome 8 (*qNCLB-8-2*) exhibited over dominance gene action while another QTL on chromosome 8 (*qNCLB-8-1*) revealed dominance type of gene action (Table 15; Fig. 4 and 5).

Table 15: QTLs detected for northern corn leaf blight resistance during individual seasons and combined over seasons using 344 $F_{2:3}$ families from the cross CML 153 × SKV 50 (Threshold LOD score = 2.50)

Season	Chromosome	Bin Location	Flanking Markers		QTL Position (cM)	Ma×imum LOD score	R^2 (%)	Genetic Effect		Gene Action	Donor Allele	QTL Name
			Left	Right				Additive	Dominance			
***Kharif*, 2012**	2	2.06	C00359-01	MZA13360-13	108.11	3.06	3.07	1.71	3.02	OD	SKV 50	*qNCLB-2*
	8	8.03	MZA2487-6		42.91	2.77	2.46	1.72	2.07	D	SKV 50	*qNCLB-8-1*
	8	8.06	MZA6428-11	MZA3856-10	80.01	3.44	22.97	1.92	3.62	OD	SKV 50	*qNCLB-8-2*
***Kharif*, 2013**	5	5.03	MZA5359-10	MZA3137-17	49.21	4.12	1.00	3.48	4.30	OD	SKV 50	*qNCLB-5-1*
	8	8.03	MZA2487-6		37.81	2.80	1.90	1.65	0.89	D	SKV 50	*qNCLB-8-1*
	8	8.06	MZA6428-11	MZA3856-10	81.31	3.05	9.68	-0.13	3.51	OD	CML 153	*qNCLB-8-2*
Combined	2	2.06	C00359-01	MZA13360-13	108.11	3.22	2.28	1.25	2.33	OD	SKV 50	*qNCLB-2*
	5	5.03	MZA3103-47	MZA533-46	53.21	4.21	1.77	0.74	2.41	OD	SKV 50	*qNCLB-5-2*
	5	5.04 – 5.05	MZA5296-6	C00171-01	142.71	3.40	10.24	3.39	-1.20	D	SKV 50	*qNCLB-5-3*
	8	8.03	MZA2487-6		43.21	3.71	1.64	2.99	1.07	D	SKV 50	*qNCLB-8-1*
	8	8.06	MZA6428-11	MZA3856-10	81.31	3.13	16.34	0.56	2.60	OD	SKV 50	*qNCLB-8-2*

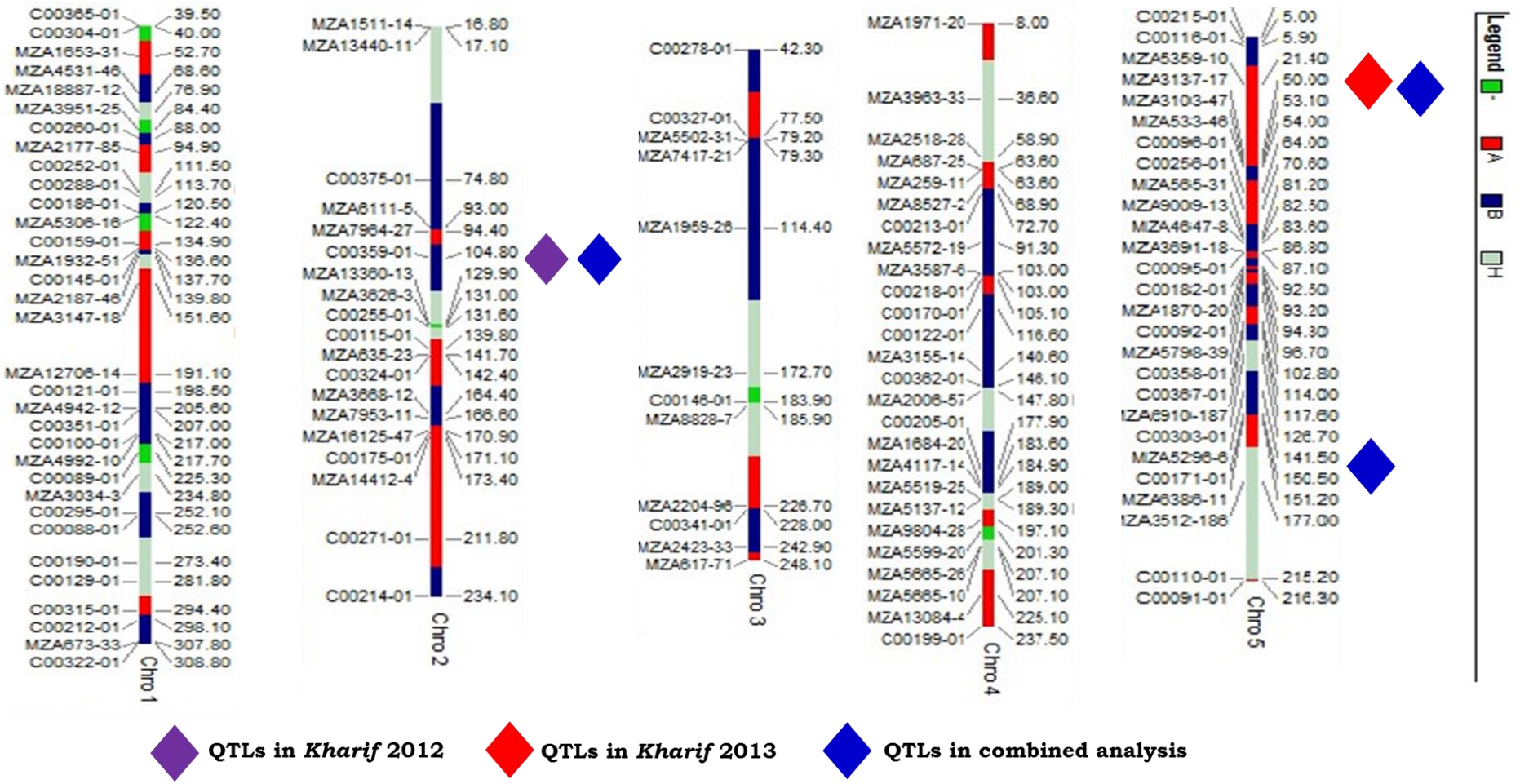


Fig. 4. Linkage map and position of the QTLs associated with northern corn leaf blight resistance mapped from $F_{2:3}$ mapping population of the cross CML153 × SKV50

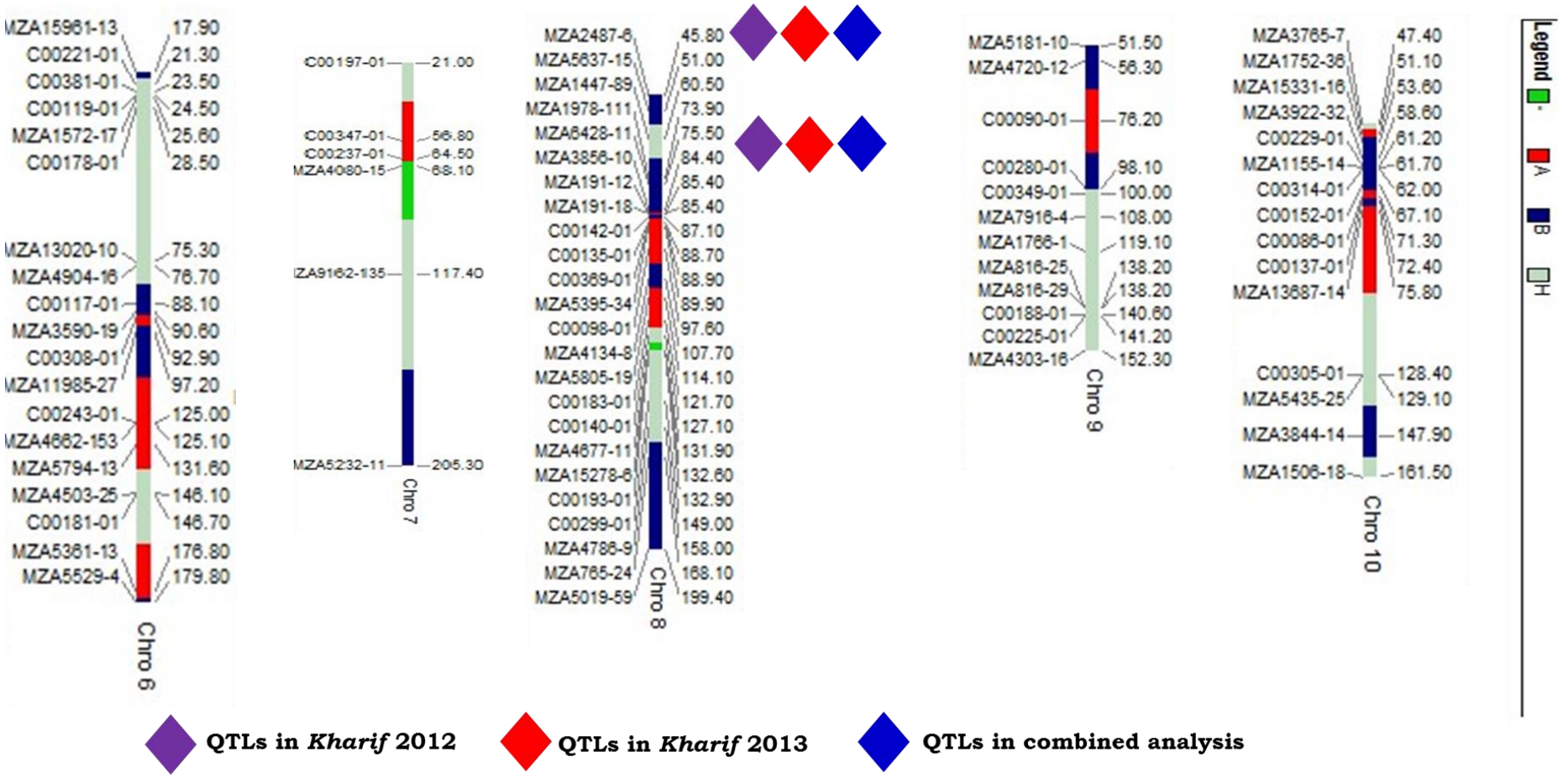

MZA15961-13 17.90
C00221-01 21.30
C00381-01 23.50
C00119-01 24.50
MZA1572-17 25.60
C00178-01 28.50
MZA13020-10 75.30
MZA4904-16 76.70
C00117-01 88.10
MZA3590-19 90.60
C00308-01 92.90
MZA11985-27 97.20
C00243-01 125.00
MZA4662-153 125.10
MZA5794-13 131.60
MZA4503-25 146.10
C00181-01 146.70
MZA5361-13 176.80
MZA5529-4 179.80
Chro 6
C00197-01 21.00
C00347-01 56.80
C00237-01 64.50
MZA4080-15 68.10
IZA9162-135 117.40
MZA5232-11 205.30
Chro 7
MZA2487-6 45.80
MZA5637-15 51.00
MZA1447-89 60.50
MZA1978-111 73.90
MZA6428-11 75.50
MZA3856-10 84.40
MZA191-12 85.40
MZA191-18 85.40
C00142-01 87.10
C00135-01 88.70
C00369-01 88.90
MZA5395-34 89.90
C00098-01 97.60
MZA4134-8 107.70
MZA5805-19 114.10
C00183-01 121.70
C00140-01 127.10
MZA4677-11 131.90
MZA15278-6 132.60
C00193-01 132.90
C00299-01 149.00
MZA4786-9 158.00
MZA765-24 168.10
MZA5019-59 199.40
Chro 8
MZA5181-10 51.50
MZA4720-12 56.30
C00090-01 76.20
C00280-01 98.10
C00349-01 100.00
MZA7916-4 108.00
MZA1766-1 119.10
MZA816-25 138.20
MZA816-29 138.20
C00188-01 140.60
C00225-01 141.20
MZA4303-16 152.30
Chro 9
MZA3765-7 47.40
MZA1752-36 51.10
MZA15331-16 53.60
MZA3922-32 58.60
C00229-01 61.20
MZA1155-14 61.70
C00314-01 62.00
C00152-01 67.10
C00086-01 71.30
C00137-01 72.40
MZA13687-14 75.80
C00305-01 128.40
MZA5435-25 129.10
MZA3844-14 147.90
MZA1506-18 161.50
Chro 10
Legend
A
B
H
QTLs in *Kharif* 2012
QTLs in *Kharif* 2013
QTLs in combined analysis

Fig. 4. Cont'd...

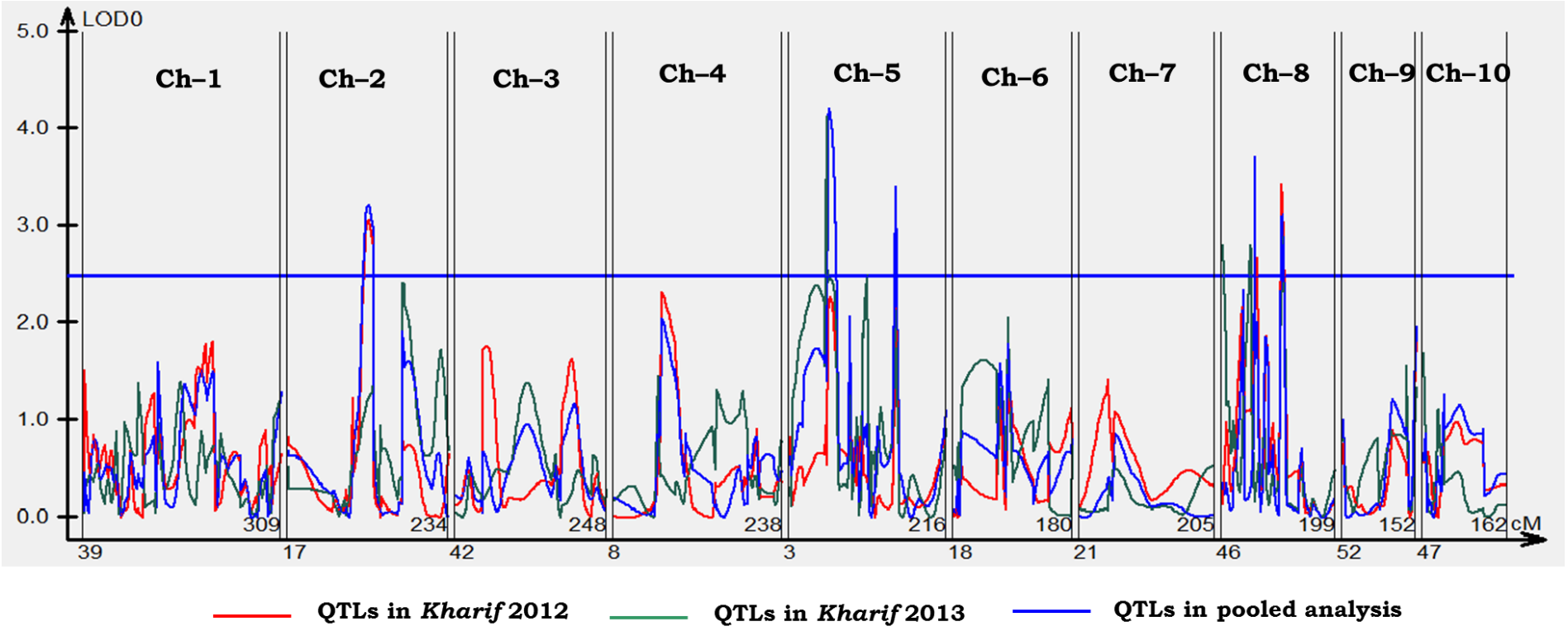


Fig. 5. LOD peak for QTLs conditioning resistance to northern corn leaf blight on chromosomes 2, 5 and 8 in *Kharif* 2012, *Kharif* 2013 and pooled analysis over seasons

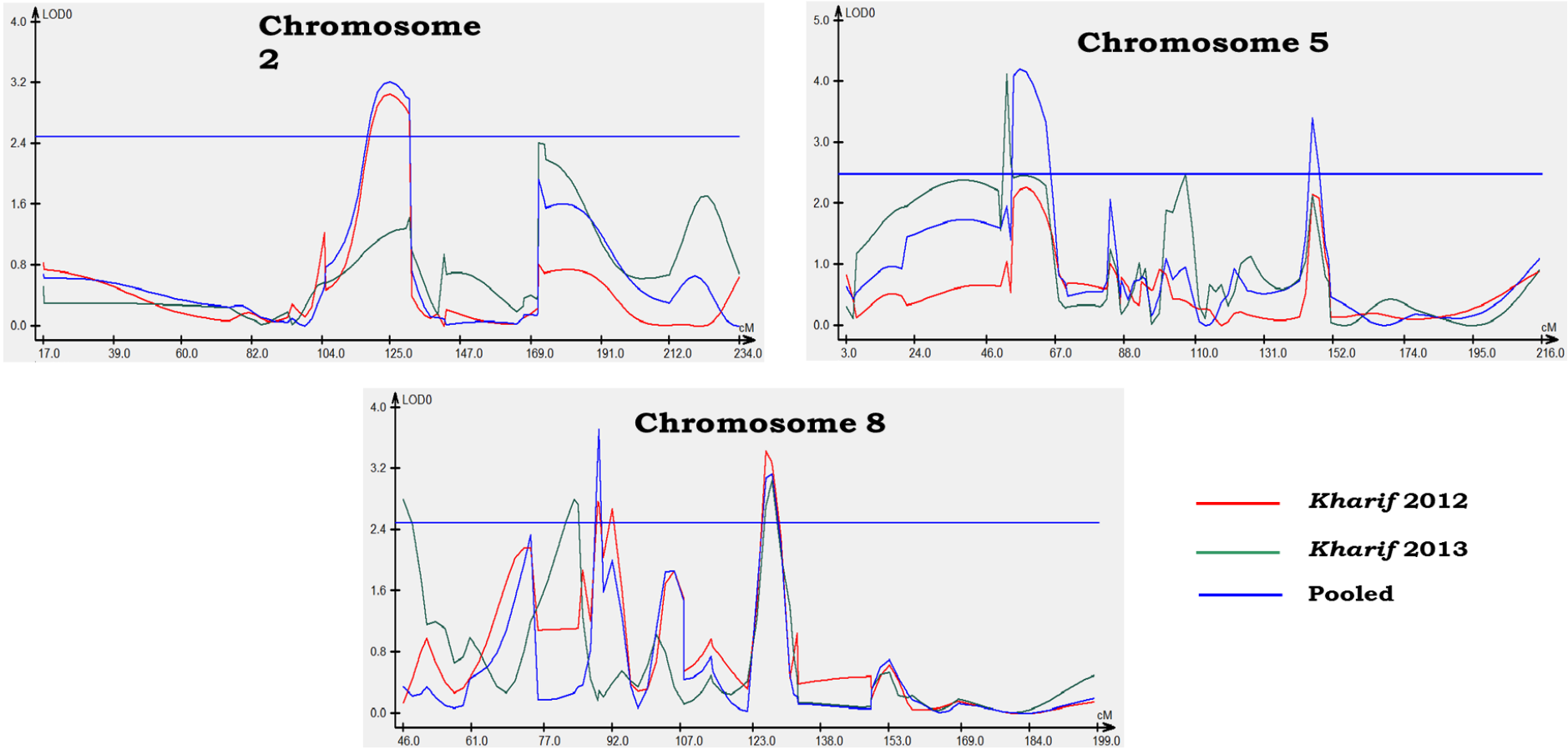

Chromosome 2
LOD0
4.0
3.2
2.4
1.6
0.8
0.0
17.0
39.0
60.0
82.0
104.0
125.0
147.0
169.0
191.0
212.0
234.0
cM
Chromosome 5
LOD0
5.0
4.0
3.0
2.0
1.0
0.0
3.0
24.0
46.0
67.0
88.0
110.0
131.0
152.0
174.0
195.0
216.0
cM
Chromosome 8
LOD0
4.0
3.2
2.4
1.6
0.8
0.0
46.0
61.0
77.0
92.0
107.0
123.0
138.0
153.0
169.0
184.0
199.0
cM
Kharif 2012
Kharif 2013
Pooled

Fig. 5. Cont'd...

***Kharif* -2013**

Three QTL regions conferring resistance to northern corn leaf blight were mapped onto chromosome 5 and chromosome 8. Among these, a QTL bracketed by markers MZA6428-11 – MZA3856-10 on chromosome 8 explained highest phenotypic variation of 16.8 *per cent* with LOD score 3.05 (*qNCLB-8-2*) followed by a QTL on chromosome 8 present adjacent to the marker MZA2487-6 which explained 2.90 *per cent* of phenotypic variation with LOD score of 2.80 (*qNCLB-8-1*). Other QTL on chromosome 5 flanked by markers MZA5359-10 – MZA3137-17 (*qNCLB-5-1*) contributed 2.00 *per cent* to the phenotypic variation with LOD score of 4.12.

Range of additive genetic effects for these QTL ranged from -0.13 to 3.48 and the total phenotypic variation of 21.60 *per cent* was explained by the QTLs identified. Favorable allele for QTL located on chromosome 8 (*qNCLB-8-2*) was contributed by susceptible parent CML153, whereas for other QTLs it was by resistant parent SKV50. In *Kharif* 2013, QTL (*qNCLB-8-1*) on chromosome 8 showed dominance gene action and two QTLs (*qNCLB-8-2 and qNCLB-5-1*) located on chromosome 8 and 5 showed over dominance gene action (Table 15; Fig. 4 and 5).

Pooled analysis

The data from *Kharif* 2012 and *Kharif* 2013 were pooled to detect QTLs for northern corn leaf blight. In combined QTL analysis, five QTLs were detected on chromosomes 2, 5 and 8 flanked by the markers C00359-01 – MZA1336013 (*qNCLB-2*), MZA3103-47 – MZA533-46 (*qNCLB-5-2*), MZA5296-6 – C00171-01 (*qNCLB-5-3*), MZA2487-6 (*qNCLB-8-1*) and MZA6428-11 – MZA3856-10 (*qNCLB-8-2*), respectively. Among these QTLs, three QTLs flanked by markers C00359-01 – MZA1336013 (*qNCLB-2*), MZA2487-6 (*qNCLB-8-1*) and MZA6428-11 – MZA3856-10 (*qNCLB-8-2*) were found to be consistent across two seasons with a LOD

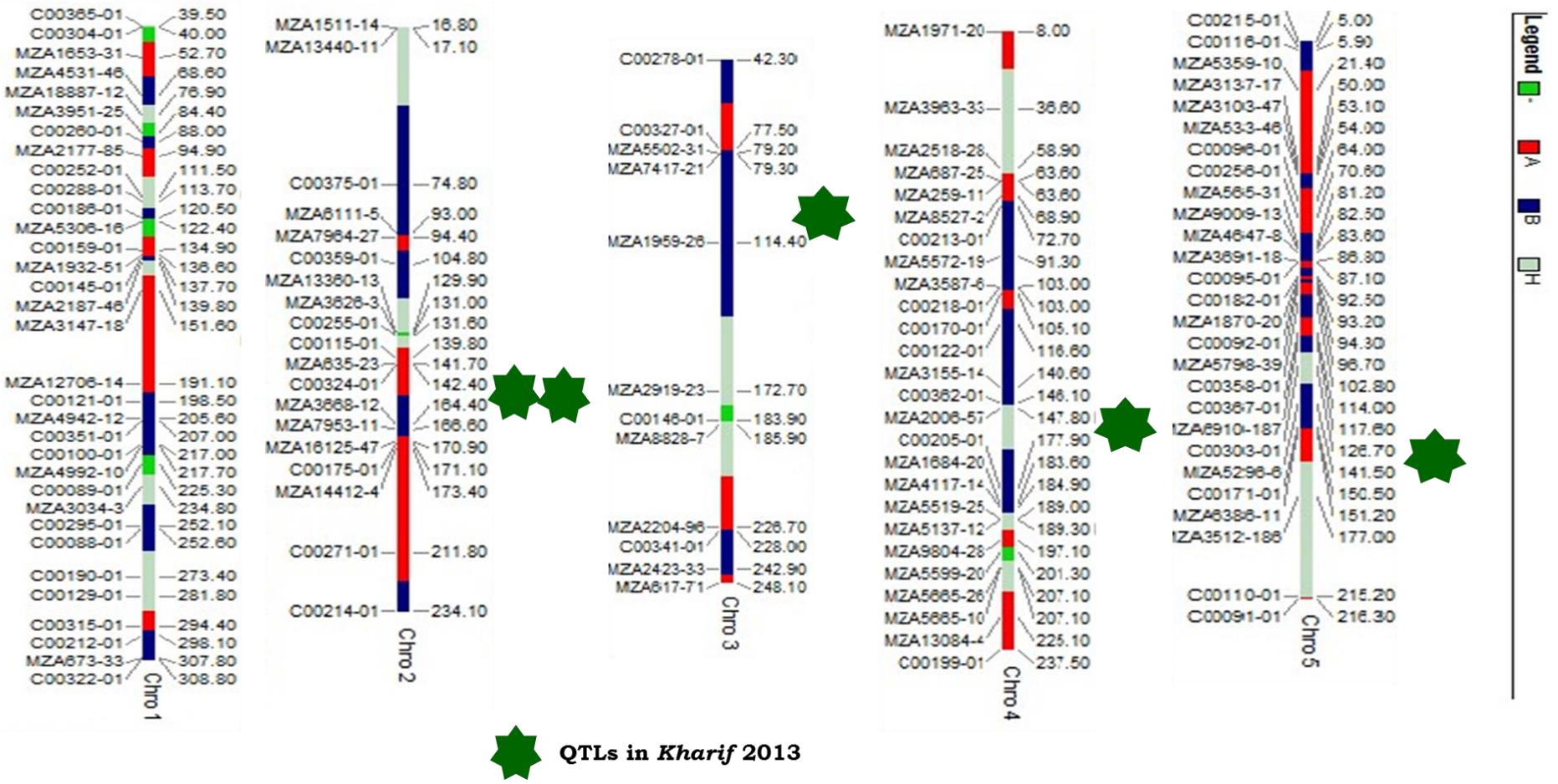


Fig. 6. Genetic linkage map and QTLs conditioning sorghum downy mildew resistance from $F_{2:3}$ mapping population of the cross CML153 × SKV50

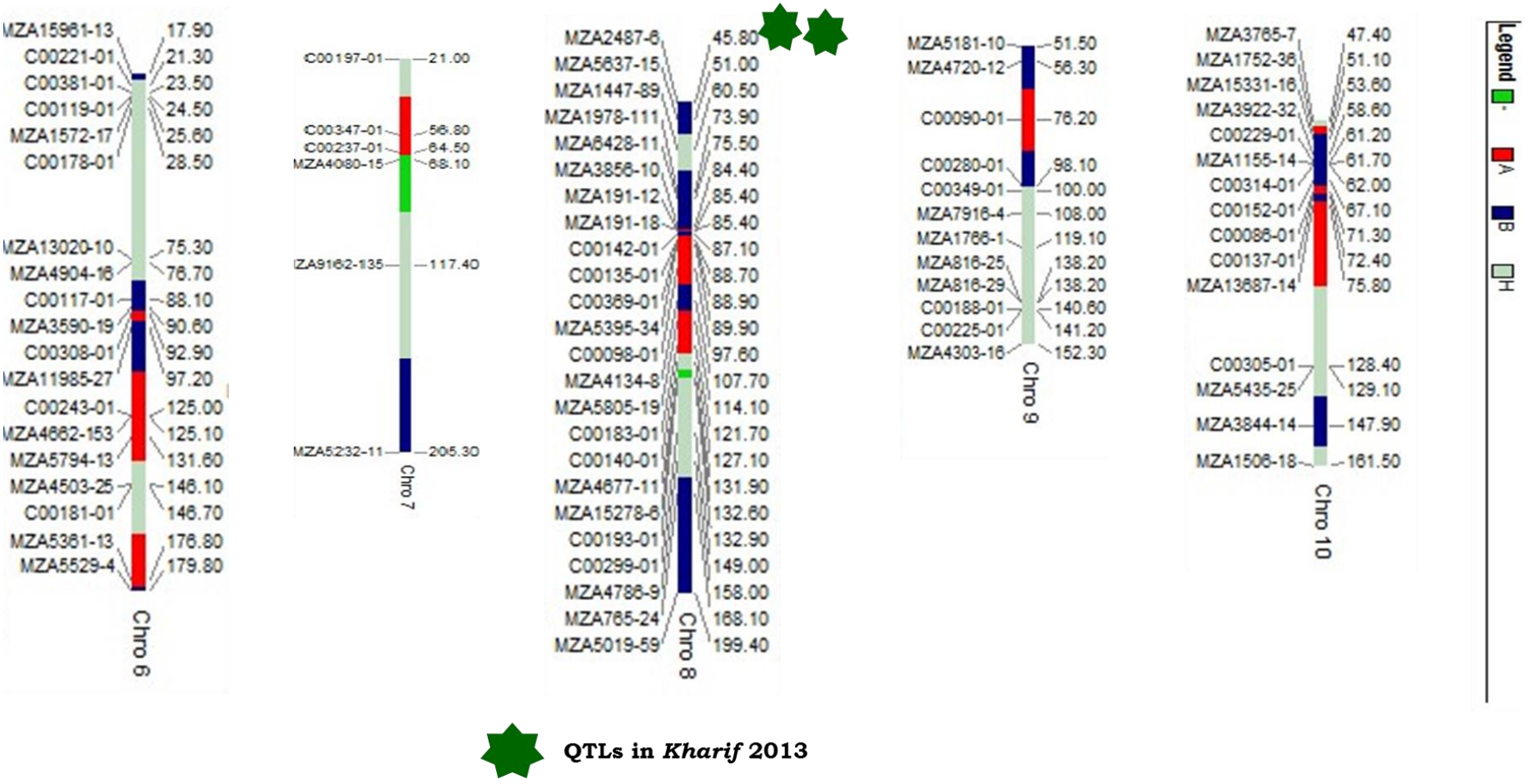

MZA15961-13 17.90
C00221-01 21.30
C00381-01 23.50
C00119-01 24.50
MZA1572-17 25.60
C00178-01 28.50
MZA13020-10 75.30
MZA4904-16 76.70
C00117-01 88.10
MZA3590-19 90.60
C00308-01 92.90
MZA11985-27 97.20
C00243-01 125.00
MZA4662-153 125.10
MZA5794-13 131.60
MZA4503-25 146.10
C00181-01 146.70
MZA5361-13 176.80
MZA5529-4 179.80
Chro 6
C00197-01 21.00
C00347-01 56.80
C00237-01 64.50
MZA4080-15 68.10
IZA9162-135 117.40
MZA5232-11 205.30
Chro 7
MZA2487-6 45.80
MZA5637-15 51.00
MZA1447-89 60.50
MZA1978-111 73.90
MZA6428-11 75.50
MZA3856-10 84.40
MZA191-12 85.40
MZA191-18 85.40
C00142-01 87.10
C00135-01 88.70
C00369-01 88.90
MZA5395-34 89.90
C00098-01 97.60
MZA4134-8 107.70
MZA5805-19 114.10
C00183-01 121.70
C00140-01 127.10
MZA4677-11 131.90
MZA15278-6 132.60
C00193-01 132.90
C00299-01 149.00
MZA4786-9 158.00
MZA765-24 168.10
MZA5019-59 199.40
Chro 8
MZA5181-10 51.50
MZA4720-12 56.30
C00090-01 76.20
C00280-01 98.10
C00349-01 100.00
MZA7916-4 108.00
MZA1766-1 119.10
MZA816-25 138.20
MZA816-29 138.20
C00188-01 140.60
C00225-01 141.20
MZA4303-16 152.30
Chro 9
MZA3765-7 47.40
MZA1752-36 51.10
MZA15331-16 53.60
MZA3922-32 58.60
C00229-01 61.20
MZA1155-14 61.70
C00314-01 62.00
C00152-01 67.10
C00086-01 71.30
C00137-01 72.40
MZA13687-14 75.80
C00305-01 128.40
MZA5435-25 129.10
MZA3844-14 147.90
MZA1506-18 161.50
Chro 10
Legend
A
B
H
QTLs in *Kharif* 2013

Fig. 6. Cont'd...

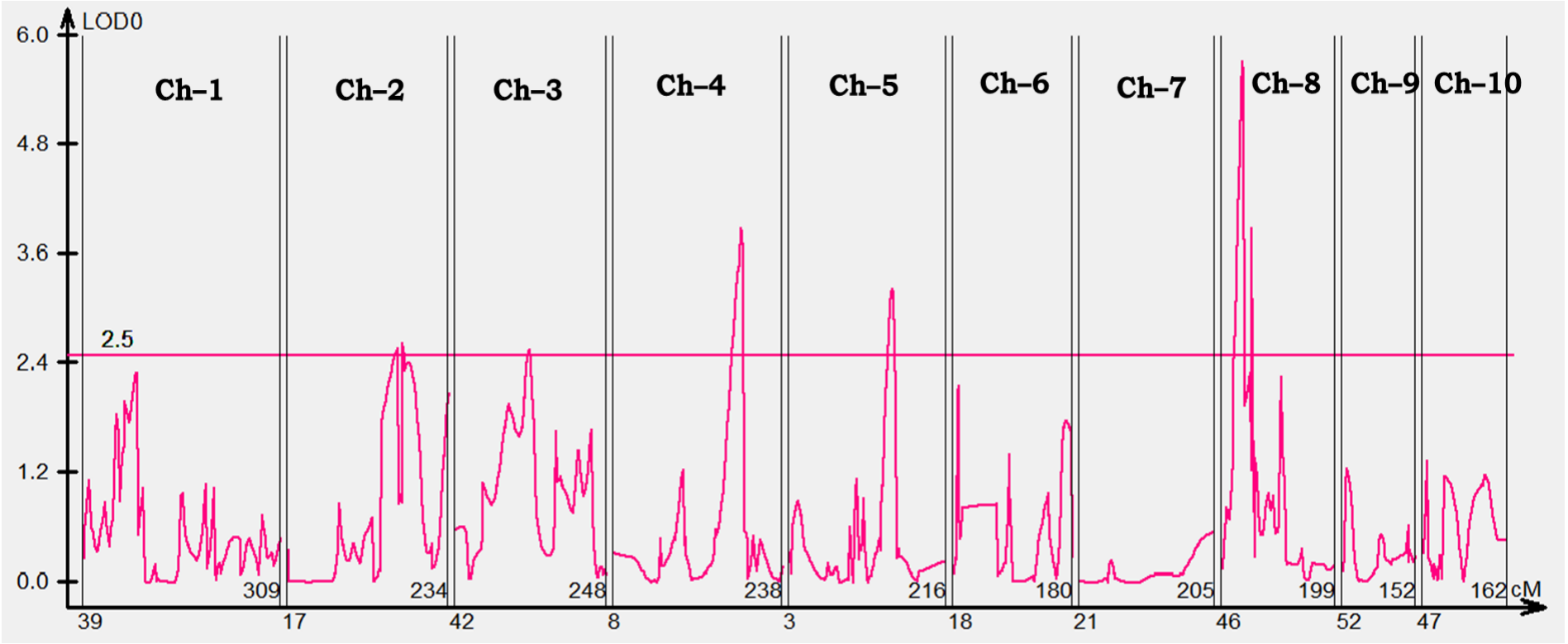


Fig. 7. LOD peak for QTLs conferring resistance to sorghum downy mildew on chromosomes 2, 3, 4, 5 and 8 during *Kharif* 2013

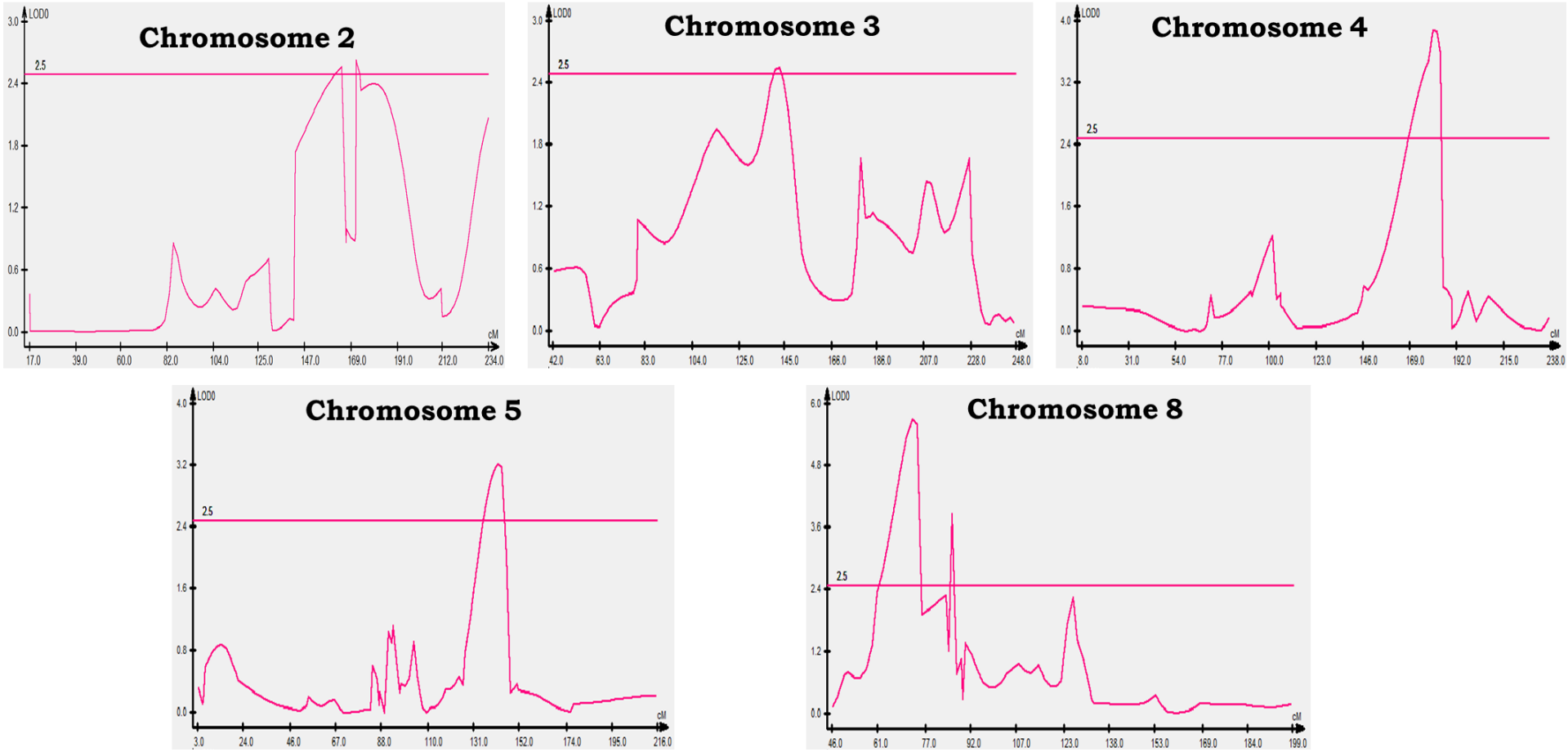

Chromosome 2
LOD0
2.5
cM
Chromosome 3
LOD0
2.5
cM
Chromosome 4
LOD0
2.5
cM
Chromosome 5
LOD0
2.5
cM
Chromosome 8
LOD0
2.5
cM

Fig. 7. Cont'd...

score of 3.22, 3.71 and 3.13. Two novel QTLs were located on chromosome 5 flanked by markers MZA3103-47 – MZA533-46 (*qNCLB-5-2*) and MZA5296-6 – C00171-01 (*qNCLB-5-3*). The QTL *qNCLB-8-2* explained highest phenotypic variation of 16.34 *per cent* followed by *qNCLB-5-3* (10.24 *per cent*). A total phenotypic variation explained by these QTLs was 32.27 *per cent* in the pooled analysis.

Additive genetic effect of these QTLs ranged from 0.74 to 3.39 and the favorable allele for these QTLs was contributed by the resistant parent SKV50. Out of five QTLs, two QTLs (*qNCLB-5-3* and *qNCLB-8-1*) located on chromosome 5 and 8 exhibited dominance and other three QTLs (*qNCLB-2*, *qNCLB-5-2* and *qNCLB-8-2*) on chromosome 2, 5 and 8 showed over dominance gene action in the combined analysis (Table 15; Fig. 4 and 5).

4.4.2 QTLs for sorghum downy mildew resistance

QTLs for resistance to sorghum downy mildew were detected using the disease incidence data from *Kharif* 2013 and the results are presented in Table 16 and depicted in Fig. 6 and briefly outlined below.

Seven QTL intervals for sorghum downy mildew resistance were identified on chromosomes 2, 3, 4, 5 and 8. Out of seven QTLs, two QTLs were located on chromosome 2 (*qsdm-2-1* and *qsdm-2-2*) flanked by the markers C00324-01 – MZA3668-12 with a LOD of 2.57 and 2.63, respectively.

The QTLs bracketed by markers MZA7417-21 – MZA1959-26 (*qsdm-3*), MZA2006-57 – C00205-01 (*qsdm-4*) and C00303-01 – MZA5296-6 (*qsdm-5*) were present on chromosomes 3, 4 and 5 with a LOD of 2.56, 3.89 and 3.22, respectively. Other two QTLs (*qsdm-8-1* and *qsdm-8-2*) adjacent to marker MZA2487-6 were mapped on to chromosome 8 with a peak LOD of 5.72 and 3.89.

Table 16: QTLs detected during *Kharif*, 2013 for sorghum downy mildew resistance using 344 $F_{2:3}$ families from the cross CML153 × SKV50 (Threshold LOD score = 2.50)

Season	Chromosome	Bin Location	Flanking Markers		QTL Position (cM)	Maximum LOD score	R^2 (%)	Genetic Effect		Gene Action	Donor Allele	QTL Name
			Left	Right				Additive	Dominance			
***Kharif*, 2013**	2	2.05-2.06	C00324-01	MZA3668-12	147.61	2.57	20.42	3.11	1.09	PD	SKV 50	*qsdm-2-1*
	2	2.06	C00324-01	MZA3668-12	154.31	2.63	15.43	-2.88	1.35	PD	CML 153	*qsdm-2-2*
	3	3.04	MZA7417-21	MZA1959-26	100.11	2.56	8.28	8.13	-2.24	PD	SKV 50	*qsdm-3*
	4	4.08	MZA2006-57	C00205-01	171.91	3.89	5.10	1.31	-5.90	OD	SKV 50	*qsdm-4*
	5	5.03-5.04	C00303-01	MZA5296-6	138.71	3.22	5.98	3.44	-2.23	PD	SKV 50	*qsdm-5*
	8	8.03	MZA2487-6		26.71	5.72	1.20	1.44	9.49	OD	SKV 50	*qsdm-8-1*
	8	8.03	MZA2487-6		39.61	3.89	1.63	5.23	2.24	PD	SKV 50	*qsdm-8-2*

Maximum phenotypic variation was explained by the QTL (*qsdm-2-1*) present on chromosome 2 (20.42 *per cent*) followed by another QTL (*qsdm-2-2)* on chromosome 2 (15.43 *per cent*), together these two QTLs on chromosome 2 explained 35.85 *per cent* of phenotypic variation. The QTL on chromosome 3 (*qsdm-3*), 4 (*qsdm-4*) and 5 (*qsdm-5*) exhibited phenotypic variation of 8.28, 5.10, and 5.98 *per cent*, respectively whereas two QTLs on chromosome 8 contributed a minor phenotypic variation for sorghum downy mildew resistance.

The range of additive effects was from -2.88 to 8.13 and for all detected QTLs, and the favorable alleles coming from the resistant parent SKV50 except for the QTL (*qsdm-2-2*) located on chromosome 2. Partial dominance type of gene action prevailed for four QTLs detected on chromosome 2 (*qsdm-2-1* and *qsdm-2-2*), chromosome 5 (*qsdm-5*) and chromosome 8 (*qsdm-8-2*) while over dominance type of gene action was noticed for three QTLs which are located on chromosome 3 (*qsdm-3*), chromosome 4 (*qsdm-4*) and chromosome 8 (*qsdm-8-1*) (Table 16; Fig. 6 and 7).

4.4.3 QTLs for southern corn rust resistance

QTLs conferring resistance to southern corn rust are tabulated in Table 17 and portrayed in Fig. 8. Seven QTL regions associated with southern corn rust resistance were distributed over four chromosomes (chromosomes 3, 8, 9 and 10). Two QTLs on chromosome 3 (*qscr-3-1* and *qscr-3-2*) flanked by markers C00278-01 and MZA7417-21 – MZA1959-26 with a LOD of 10.36 and 9.46, explained phenotypic variation of 2.55 and 1.01 *per cent*, respectively. Two QTLs (*qscr-8-1* and *qscr-8-2*) flanked by markers MZA2487-6 and MZA2487-6 – MZA5637-15 were located on chromosome 8 with a LOD of 2.92 and 3.11, displaying a phenotypic variation of 4.17 and 4.71 *per cent*, respectively. On chromosome 9, two QTL (*qscr-9-1* and *qscr-9-2*) intervals were identified with flanking

Table 17: QTLs detected for southern corn rust resistance during *Kharif*, 2013 using 344 $F_{2:3}$ families from the cross CML153 × SKV50 (Threshold LOD score = 2.50)

Season	Chromosome	Bin Location	Flanking Markers		QTL Position (cM)	Maximum LOD score	R^2 (%)	Genetic Effect		Gene Action	Donor Allele	QTL Name
			Left	Right				Additive	Dominance			
***Kharif*, 2013**	3	3.04	C00278-01		18.01	10.36	2.55	15.54	15.64	D	SKV 50	*qscr-3-1*
	3	3.04-3.05	MZA7417-21	MZA1959-26	102.11	9.46	1.01	-16.28	15.09	D	CML 153	*qscr-3-2*
	8	8.03	MZA2487-6		22.71	2.92	4.17	1.67	5.23	OD	SKV 50	*qscr-8-1*
	8	8.03	MZA2487-6	MZA5637-15	48.21	3.11	4.71	8.19	6.79	D	SKV 50	*qscr-8-2*
	9	9.03	MZA4720-12	C00090-01	67.51	2.79	4.67	-2.62	2.08	PD	CML 153	*qscr-9-1*
	9	9.03	C00090-01	C00280-01	88.61	3.26	2.81	4.04	2.69	PD	SKV 50	*qscr-9-2*
	10	10.03	MZA15331-16	MZA3922-32	54.41	9.36	18.59	20.84	9.01	PD	SKV 50	*qscr-10*

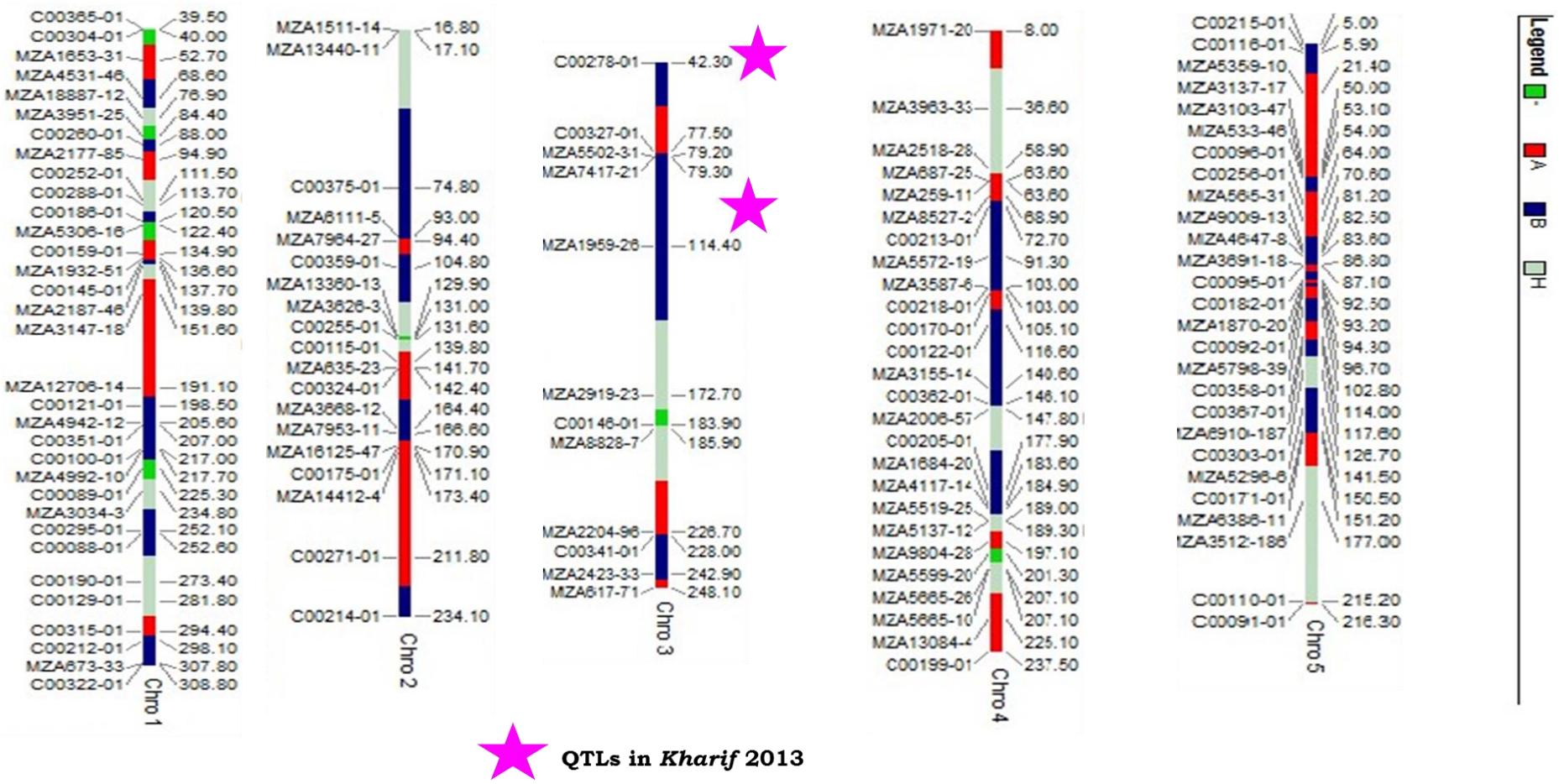


Fig. 8. Genetic linkage map showing marker position and QTLs associated with southern corn rust resistance in the cross CML153 × SKV50

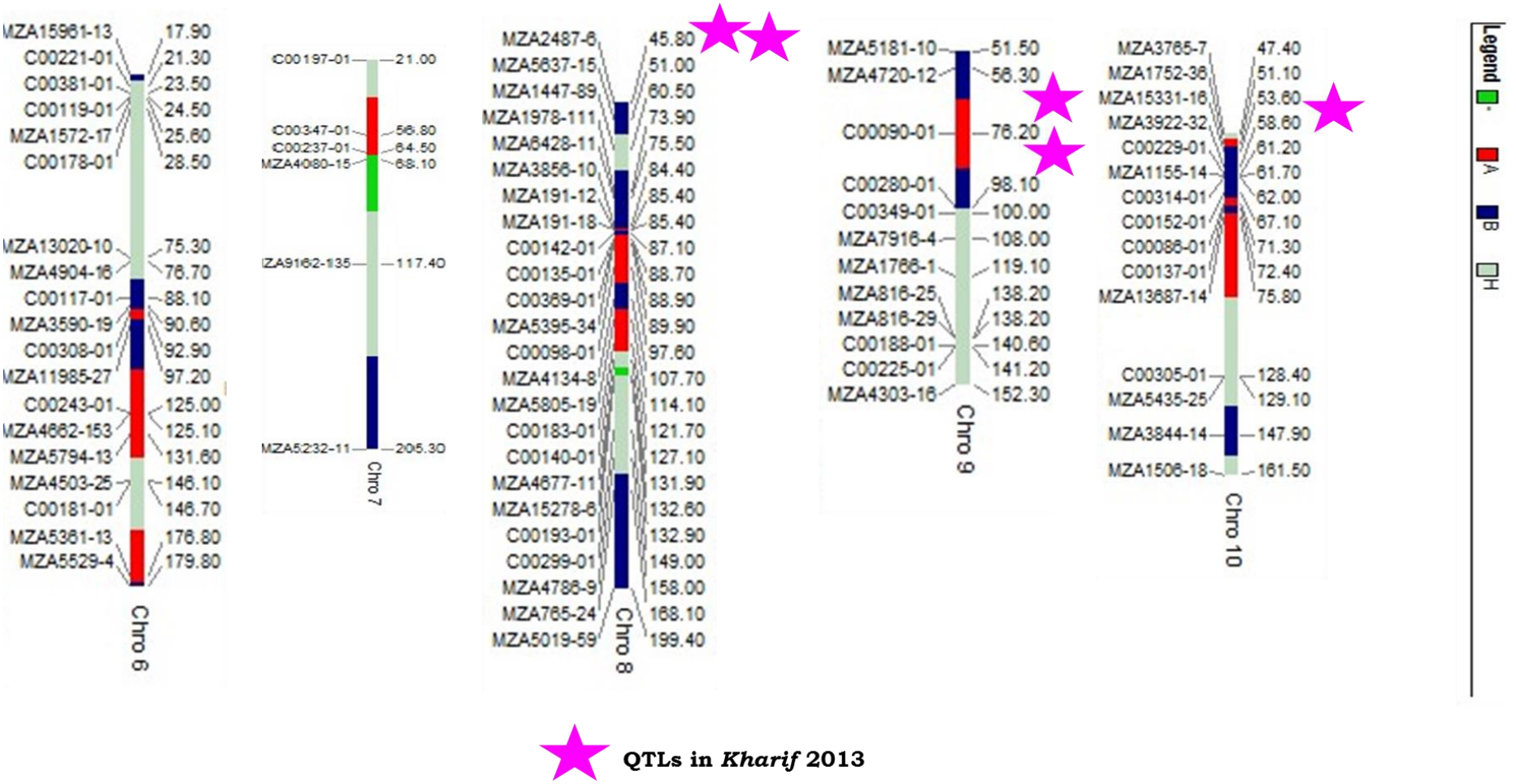

MZA15961-13 17.90
C00221-01 21.30
C00381-01 23.50
C00119-01 24.50
MZA1572-17 25.60
C00178-01 28.50
MZA13020-10 75.30
MZA4904-16 76.70
C00117-01 88.10
MZA3590-19 90.60
C00308-01 92.90
MZA11985-27 97.20
C00243-01 125.00
MZA4662-153 125.10
MZA5794-13 131.60
MZA4503-25 146.10
C00181-01 146.70
MZA5361-13 176.80
MZA5529-4 179.80
Chro 6
C00197-01 21.00
C00347-01 56.80
C00237-01 64.50
MZA4080-15 68.10
IZA9162-135 117.40
MZA5232-11 205.30
Chro 7
MZA2487-6 45.80
MZA5637-15 51.00
MZA1447-89 60.50
MZA1978-111 73.90
MZA6428-11 75.50
MZA3856-10 84.40
MZA191-12 85.40
MZA191-18 85.40
C00142-01 87.10
C00135-01 88.70
C00369-01 88.90
MZA5395-34 89.90
C00098-01 97.60
MZA4134-8 107.70
MZA5805-19 114.10
C00183-01 121.70
C00140-01 127.10
MZA4677-11 131.90
MZA15278-6 132.60
C00193-01 132.90
C00299-01 149.00
MZA4788-9 158.00
MZA765-24 168.10
MZA5019-59 199.40
Chro 8
MZA5181-10 51.50
MZA4720-12 56.30
C00090-01 76.20
C00280-01 98.10
C00349-01 100.00
MZA7916-4 108.00
MZA1766-1 119.10
MZA816-25 138.20
MZA816-29 138.20
C00188-01 140.60
C00225-01 141.20
MZA4303-16 152.30
Chro 9
MZA3765-7 47.40
MZA1752-38 51.10
MZA15331-16 53.60
MZA3922-32 58.60
C00229-01 61.20
MZA1155-14 61.70
C00314-01 62.00
C00152-01 67.10
C00086-01 71.30
C00137-01 72.40
MZA13687-14 75.80
C00305-01 128.40
MZA5435-25 129.10
MZA3844-14 147.90
MZA1506-18 161.50
Chro 10
Legend
A
B
H
QTLs in *Kharif* 2013

Fig. 8. Cont'd...

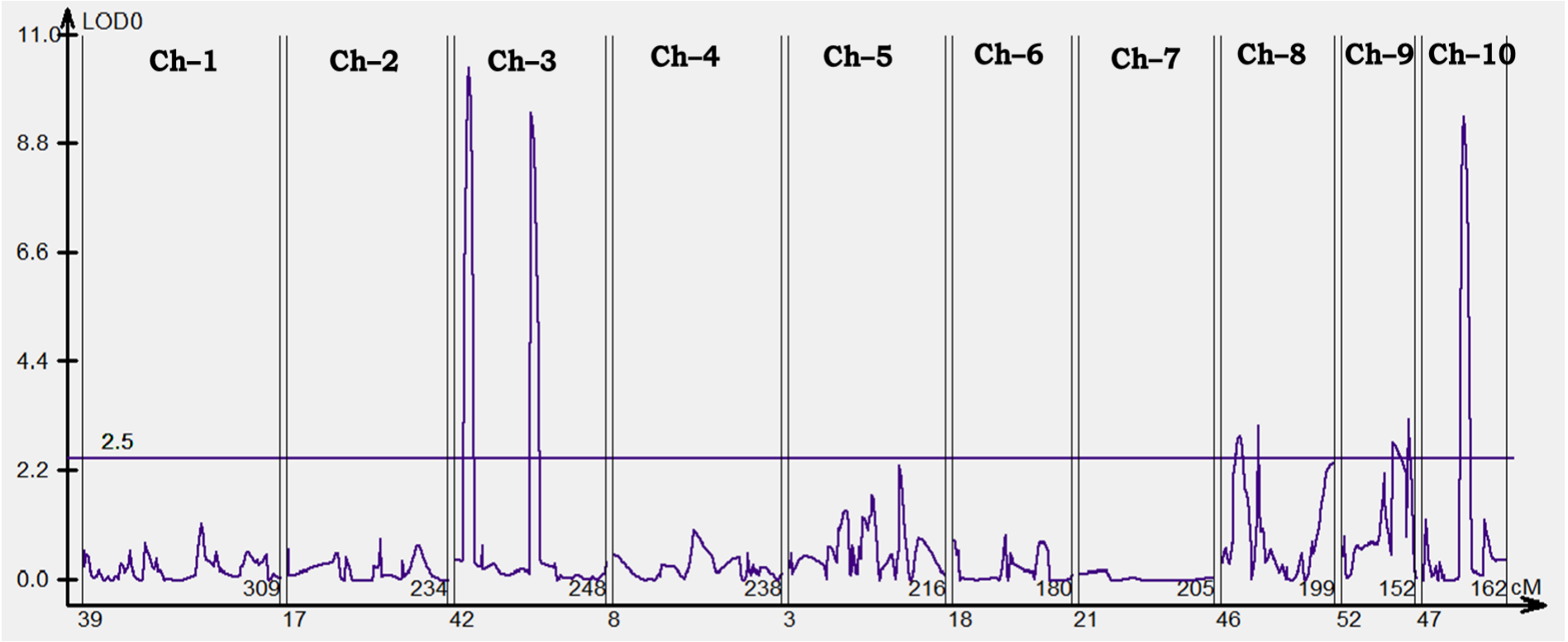


Fig. 9. LOD peak for QTLs associated with resistance to southern corn rust on chromosomes 3, 8 9 and 10 during *Kharif* 2013

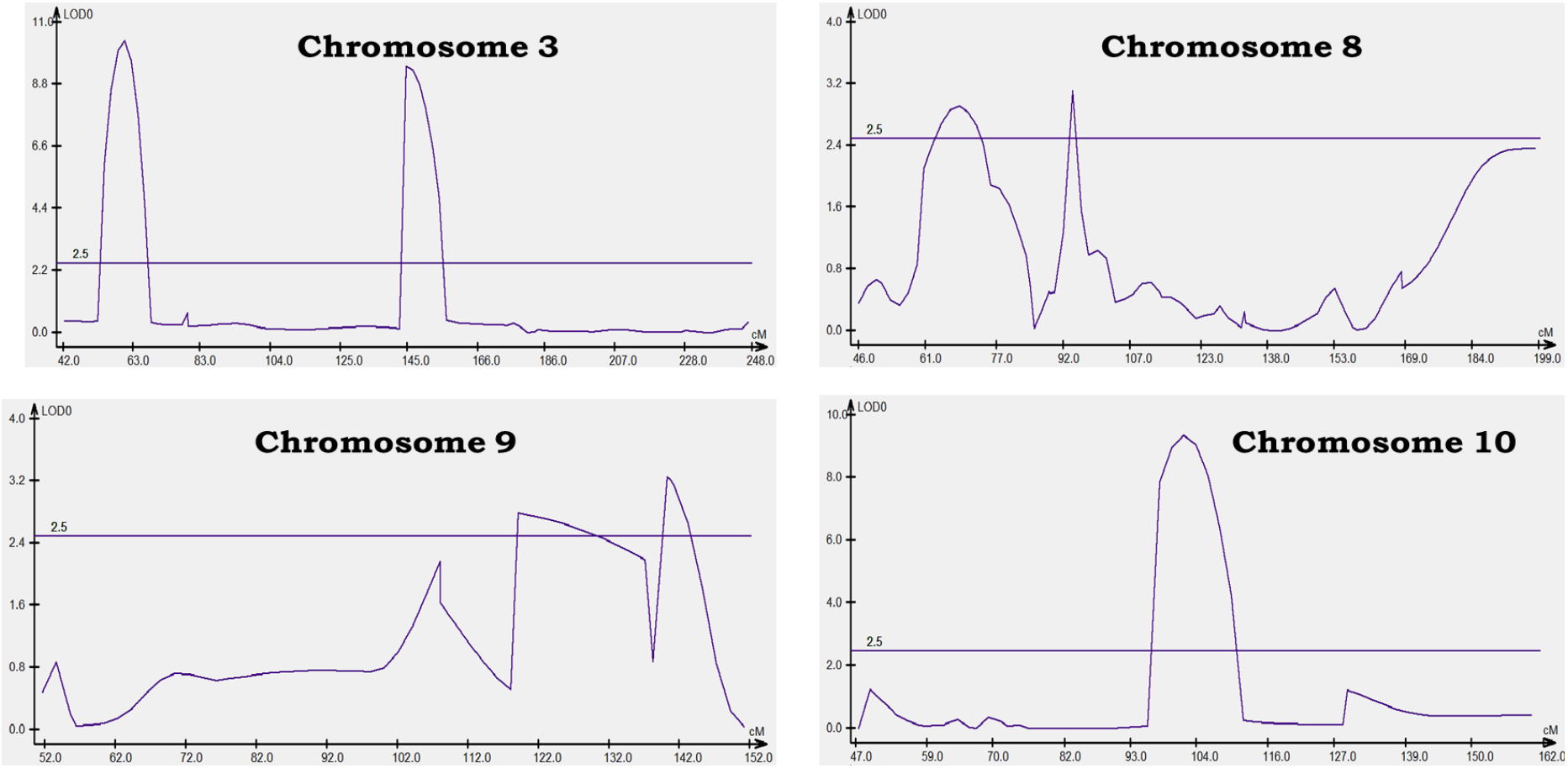

Chromosome 3
LOD0
11.0
8.8
6.6
4.4
2.2
0.0
2.5
cM
42.0
63.0
83.0
104.0
125.0
145.0
166.0
186.0
207.0
228.0
248.0
Chromosome 8
LOD0
4.0
3.2
2.4
1.6
0.8
0.0
2.5
cM
46.0
61.0
77.0
92.0
107.0
123.0
138.0
153.0
169.0
184.0
199.0
Chromosome 9
LOD0
4.0
3.2
2.4
1.6
0.8
0.0
2.5
cM
52.0
62.0
72.0
82.0
92.0
102.0
112.0
122.0
132.0
142.0
152.0
Chromosome 10
LOD0
10.0
8.0
6.0
4.0
2.0
0.0
2.5
cM
47.0
59.0
70.0
82.0
93.0
104.0
116.0
127.0
139.0
150.0
162.0

Fig. 9. Cont'd...

markers MZA4720-12 – C00090-01 and C00090-01 – C00280-01 at peak LOD of 2.79 and 3.26, explaining a phenotypic variation of 4.67 and 2.81 *per cent*, respectively. With phenotypic variation of 18.59 *per cent*, one major QTL (*qscr-10*) conferring resistance to southern corn rust was mapped onto chromosome 10 in the marker interval MZA15331-16 – MZA3922-32 with peak LOD of 9.36.

Wide range of additive effects was observed for all identified QTLs (-16.28 to 20.84). Two QTLs (*qscr-3-2* and *qscr-9-1*) on chromosome 3 and 9 were contributed by susceptible parent CML153 whereas the other five QTLs (*qscr-3-1, qscr-8-1, qscr-8-2, qscr-9-2* and *qscr-10*) on chromosomes 3, 8, 9 and 10 were contributed by resistant parent SKV50. Preponderance of dominance type of gene action was seen in three QTLs (*qscr-3-1* and *qscr-3-2* on chromosome 3, *qscr-8-2* on chromosome 8) while over dominance was prevalent in the QTL *qscr-8-1* present on chromosome 8. Whereas, partial dominance was prevailed in three identified QTLs (*qscr-9-1* and *qscr-9-2* on chromosome 9, *qscr-10* on chromosome 10) (Table 17; Fig. 8 and 9).

4.5 Co-localization of QTLs conferring resistance to multiple foliar pathogens during *Kharif*-2013

Pairwise Pearson correlation co-efficient between three diseases was worked out and presented in Table 18. The results revealed that significant correlation was observed between northern corn leaf blight, sorghum downy mildew and southern corn rust. The highest significant and positive correlation was noticed between northern corn leaf blight and southern corn rust (r = 0.122) whereas positive but non-significant pairwise correlation between sorghum downy mildew and southern corn rust (r = 0.061) was observed. But, negatively significant correlation was revealed between sorghum downy mildew and northern corn leaf blight (r = -0.120). This implies that there are loci (and possibly genes) conferring

Table 18: Pearson correlation co-efficient between mean disease severity for three diseases *viz.*, northern corn leaf blight (NCLB), sorghum downy mildew (SDM) and southern corn rust (SCR) during *Kharif* 2013

Disease	NCLB	SDM	SCR
NCLB	1	-0.120*	0.122*
SDM		1	0.061
SCR			1

*Significant at 5% level of significance

Where,

NCLB – Northern Corn Leaf Blight

SDM – Sorghum Downy Mildew

SCR – Southern Corn Rust

Table 19: QTLs conditioning resistance to northern corn leaf blight (NCLB), southern corn rust (SCR) and sorghum downy mildew (SDM) during *Kharif* 2013 (Threshold LOD score = 2.50)

Co-localized Chromosome Bin	Disease QTLs	Flanking Marker Interval	QTL Position (cM)	Maximum LOD score	R^2 (%)	Genetic Effect		Gene Action	Donor Allele
						Additive	Dominance		
8.03	**NCLB**	MZA2487-6	43.21	3.71	1.64	2.99	1.07	D	SKV 50
	SDM	MZA2487-6	26.71	5.72	1.20	1.44	9.49	OD	SKV 50
	SDM	MZA2487-6	39.61	3.89	1.63	5.23	2.24	PD	SKV 50
	SCR	MZA2487-6	22.71	2.92	4.17	1.67	5.23	OD	SKV 50
	SCR	MZA2487-6 - MZA5637-15	48.21	3.11	4.71	8.19	6.79	D	SKV 50
5.03/5.04	**NCLB**	MZA3103-47 - MZA533-46	53.21	4.21	1.77	0.74	2.41	OD	SKV 50
	NCLB	MZA5296-6 - C00171-01	142.71	3.40	10.24	3.39	-1.20	D	SKV 50
	SDM	C00303-01 - MZA5296-6	138.71	3.22	5.98	3.44	-2.23	PD	SKV 50
3.04	**SDM**	MZA7417-21 MZA1959-26	100.11	2.56	8.28	8.13	-12.24	OD	SKV 50
	SCR	MZA7417-21 - MZA1959-26	102.11	9.46	1.01	-16.28	15.09	D	CML 153
2.06	**NCLB**	C00359-01 - MZA13360-13	108.11	3.22	2.28	1.25	2.33	OD	SKV 50
	SDM	C00324-01 - MZA3668-12	147.61	2.57	20.42	3.11	1.09	PD	SKV 50
	SDM	C00324-01 - MZA3668-12	154.31	2.63	15.43	-2.88	1.35	PD	CML 153

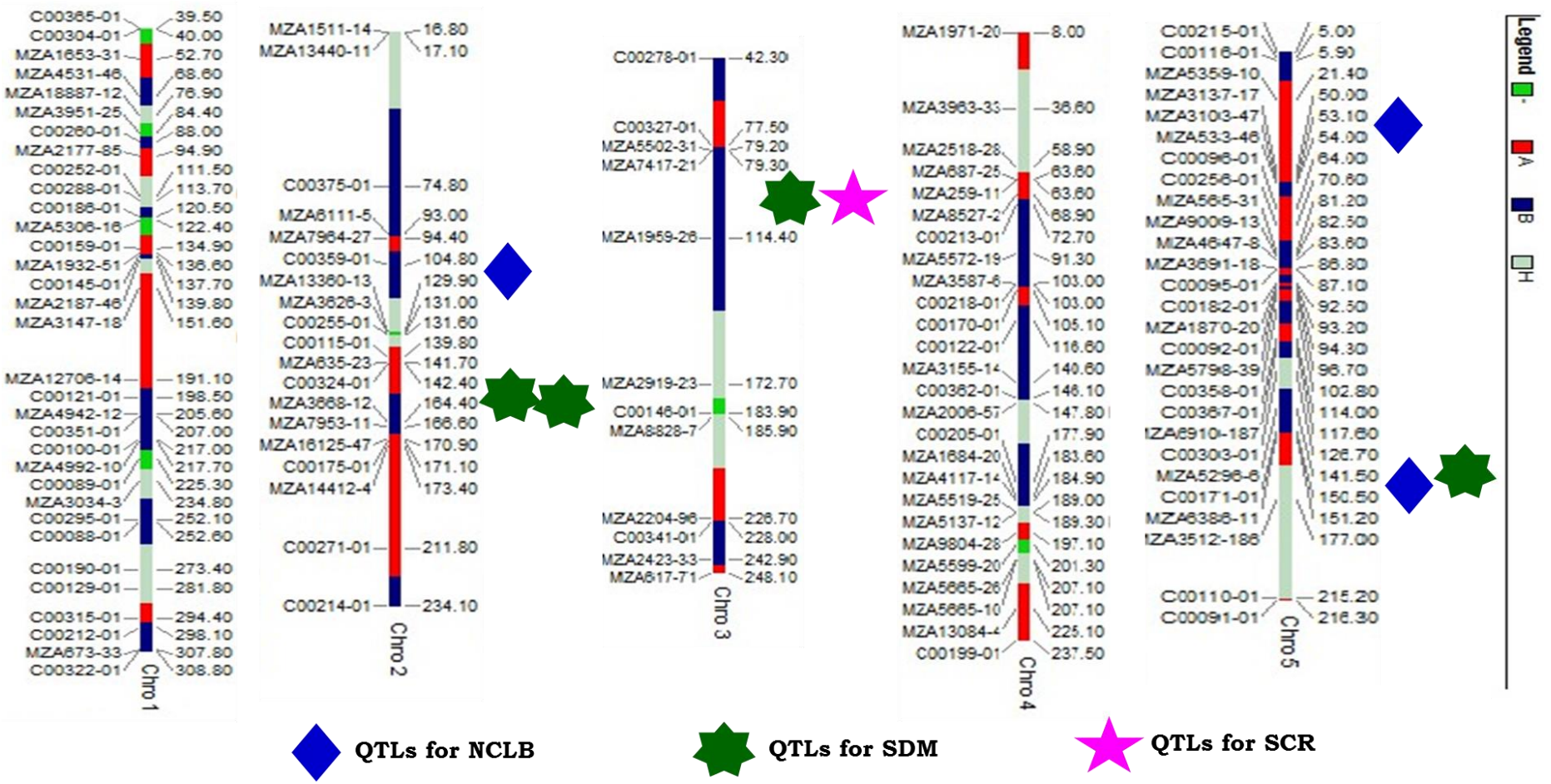


Fig. 10. Position of QTLs conferring resistance to three foliar diseases *viz.*, northern corn leaf blight (NCLB), sorghum downy mildew (SDM) and southern corn rust (SCR) during *Kharif* 2013

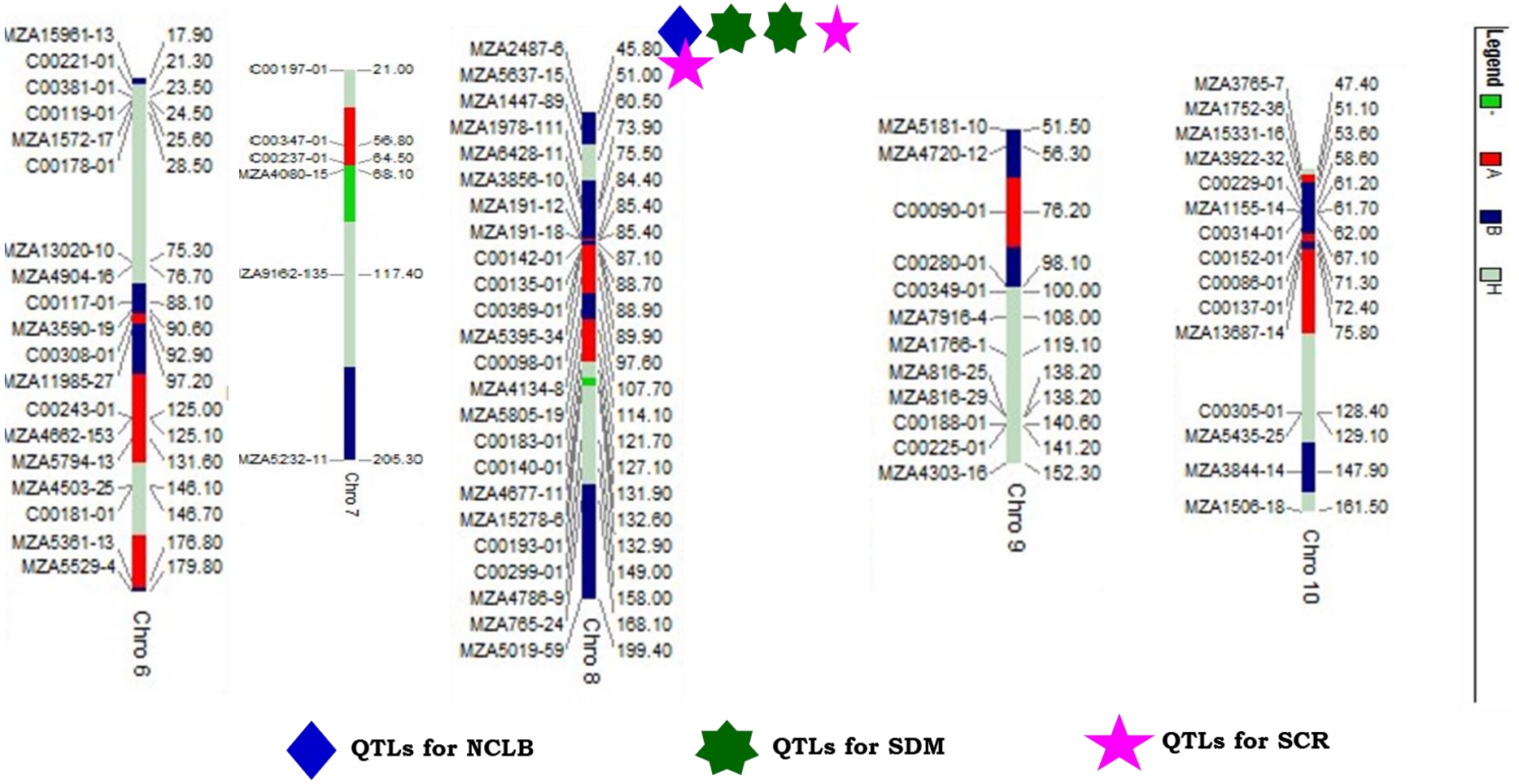


Fig. 10. Position of QTLs conferring resistance to three foliar diseases *viz.*, northern corn leaf blight (NCLB), sorghum downy mildew (SDM) and southern corn rust (SCR) during *Kharif* 2013

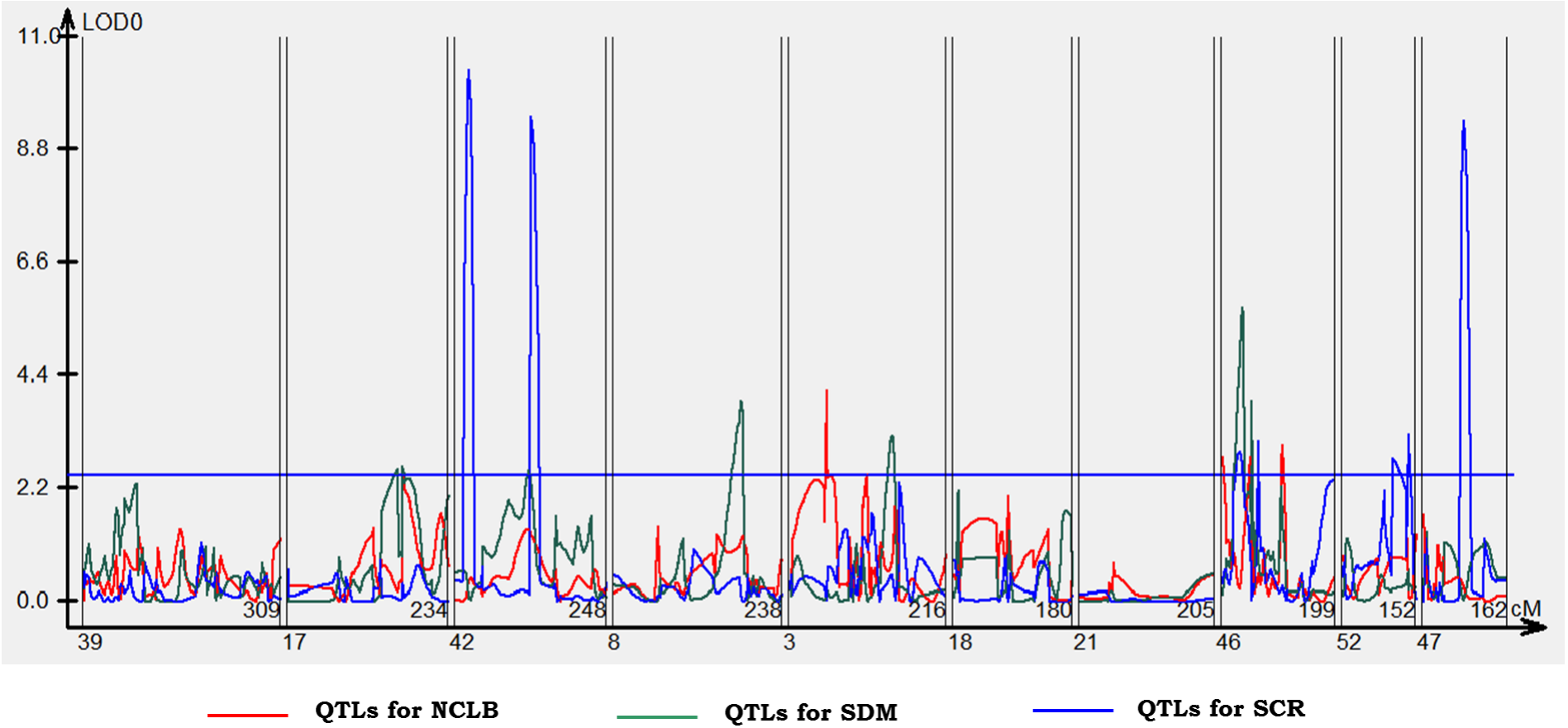


Fig. 11. LOD peak for QTLs conferring resistance to three foliar diseases *viz.*, northern corn leaf blight (NCLB), sorghum downy mildew (SDM) and southern corn rust (SCR) during *Kharif* 2013

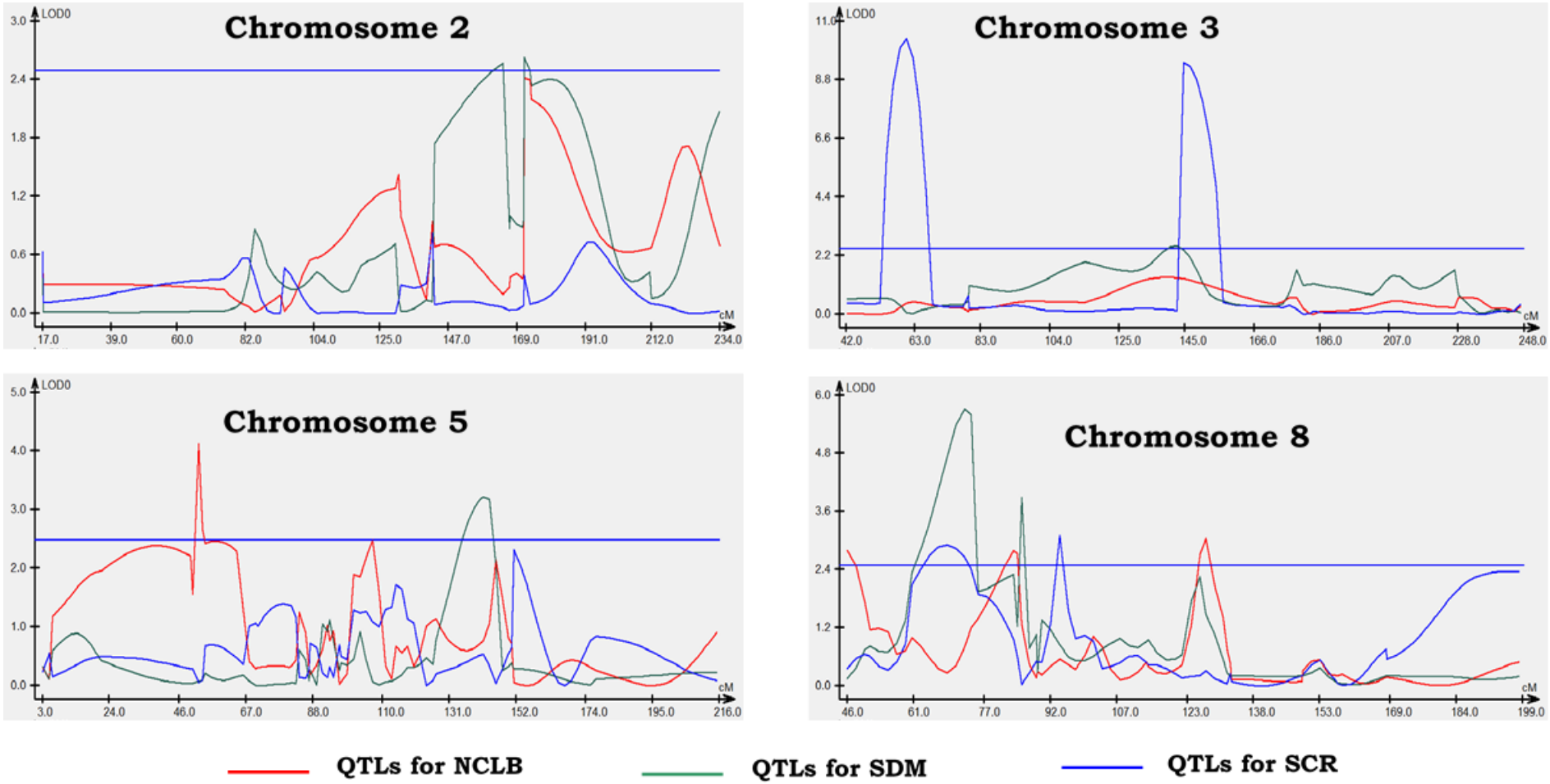

Chromosome 2
LOD0
3.0
2.4
1.8
1.2
0.6
0.0
cM
17.0
39.0
60.0
82.0
104.0
125.0
147.0
169.0
191.0
212.0
234.0
Chromosome 3
LOD0
11.0
8.8
6.6
4.4
2.2
0.0
cM
42.0
63.0
83.0
104.0
125.0
145.0
166.0
186.0
207.0
228.0
248.0
Chromosome 5
LOD0
5.0
4.0
3.0
2.0
1.0
0.0
cM
3.0
24.0
46.0
67.0
88.0
110.0
131.0
152.0
174.0
195.0
216.0
Chromosome 8
LOD0
6.0
4.8
3.6
2.4
1.2
0.0
cM
46.0
61.0
77.0
92.0
107.0
123.0
138.0
153.0
169.0
184.0
199.0
QTLs for NCLB
QTLs for SDM
QTLs for SCR

Fig. 11. Cont'd...

multiple disease resistance to these three diseases in the $F_{2:3}$ population derived from the cross CML153 × SKV50.

In the present study, chromosomal regions harbouring QTLs for northern corn leaf blight, sorghum downy mildew and southern corn rust resistance are co-localized (Table 19 and Fig. 10).

Several QTLs were associated with resistance to more than one disease in this study. In bin 8.03, QTL conferring resistance to all three foliar diseases *viz.*, northern corn leaf blight (one QTL), sorghum downy mildew (two QTLs) and southern corn rust (two QTLs) were co-localized at approximately the same map position (<10 cM difference between the QTL peaks for the three diseases) with common adjacent marker MZA2487-6.

Bin 5.03-5.04 was significantly associated with resistance to northern corn leaf blight and sorghum downy mildew (two QTLs for northern corn leaf blight and one QTL for sorghum downy mildew co-localized with a map distance of <3 cM). This co-localized QTL exhibited 10.24 and 5.98 *per cent* of total phenotypic variation for northern corn leaf blight and sorghum downy mildew, respectively.

QTLs for southern corn rust and sorghum downy mildew resistance were co-localized in bin 3.04 at a map distance of 1 cM with a common flanking marker MZA1959-26; while QTL conferring resistance to northern corn leaf blight and sorghum downy mildew were co-localized in chromosome bin 2.06 (Table 19; Fig. 10 and 11).

Discussion

V. DISCUSSION

Maize (*Zea mays* L.) is an important cereal crop and the demand for its grain is increasing every year because of its diverse uses. As many as 112 diseases affect maize in different parts of the world, caused by fungi, bacteria, viruses and nematodes leading to extensive damage. In India about 61 diseases have been reported to affect the maize crop. Among these, foliar diseases are the primary biotic constraints to maize yields like maize streak virus, sorghum downy mildew, northern corn leaf blight, gray leaf spot, rusts (*Puccinia spp*), ear rot (*Fusarium* and *Diplodia spp*), banded leaf and sheath blights, stalk rots and corn stunt (Payak and Renfro, 1968; Payak *et al.*, 1973; Payak and Sharma, 1985).

Among several foliar diseases, northern corn leaf blight, sorghum downy mildew and southern corn rust are the most devastating diseases prevalent in India and worldwide. Breeding for resistance is a practical, cost-effective means available to manage most of the diseases in different crop species (Fehr, 1987). In many plant breeding programs, development of genotypes with resistance to several foliar diseases simultaneously is an important goal, but it is difficult to achieve because of other important traits for improvement (Castor, 1992). As a consequence, most breeders often do not invest equal effort in improving resistance to all diseases but focus on an individual disease causing the greatest loss (Lambert and White, 1997).

Host-pathogen resistance to diseases is classified as qualitative or quantitative. Qualitative resistance is controlled by single and usually dominant genes that interact with the pathogen on a gene-for-gene basis. Unlike qualitative resistance, plants exhibiting quantitative resistance display levels of resistance that show continuous variation and, usually, incomplete expression. Quantitative resistance is controlled by multiple

genes with smaller but continuous phenotypic effects or by relatively few genes with large environmental influence (Michelmore, 1995). Resistance reaction is expressed as reduced rate of disease development and immunity in some cases. Differences in disease reaction are generally recognized qualitatively by distinctly different lesion types associated with certain genotypes or quantitatively by development of fewer lesions or a slower rate of lesion expansion on more resistant genotypes.

Enormous amount of genetic resources having resistance to individual foliar diseases are available in the country (Hooda *et al.*, 2012). Despite the fact that various sources of resistance exist for foliar diseases, hybrids and inbreds with resistance to single pathogens are not entirely useful because they are likely to be challenged by different pathogens across environments (Pratt *et al.*, 1997). Actual circumstances in the field required varieties having combined resistance to more than one foliar disease, but also to other important abiotic stress factors, and acceptable agronomic characteristics.

Multiple disease resistance in which the same locus conferring resistance to multiple pathogens is both practically and conceptually important. Very few cultivars of maize with resistance to all the three foliar diseases *viz.*, northern corn leaf blight, sorghum downy mildew and southern corn rust along with better combining ability are available in the country necessitating introgression of resistance genes into different genetic backgrounds (heterotic groups). Hence, incorporation of multiple disease resistance in high combiners and various germplasm using DNA markers assisted breeding that eventually aid in development of high yielding disease resistant hybrids suited to specific environments, needs to be given at most importance.

In recent years, advent of molecular markers has revolutionized the breeding procedure in gaining better success. Markers are used in

practical breeding to assist backcrossing of resistance loci into elite cultivars previously developed through conventional means. The usefulness of genetic markers are (1) markers can help in selection of target alleles whose effects are difficult to observe phenotypically such as recessive genes, (2) combining multiple disease resistance genes in a common genotype when effects are known, (3) aid in selection of alleles conferring resistance to diseases that are not regularly expressed in a particular environment and (4) used to select disease resistant gene where phenotypic assays are more expensive than marker assays.

Integrating molecular approaches with traditional breeding for disease resistance could significantly increase the efficiency of selection of desirable resistant genotypes. Most traits of agricultural importance, such as yield and disease resistance are complex and governed by polygenes with quantitative inheritance. There are many regions within the genome, which contain genes that are associated with a polygenic trait and are called quantitative trait loci (QTLs). The identification of QTLs based on conventional phenotypic evaluation is not possible. Molecular markers linked to quantitative traits would be useful in both identifying suitable parents as well as identifying the desirable progeny among the breeding lines. Thus, identifying quantitative trait loci (QTL) for resistance to diseases has greater significance in the present scenario. With the advancements in understanding the genetics of disease resistance and the development of robust screening techniques, identification of QTLs has become more precise.

In the present investigation, six generation mean analysis was employed to know the type of gene action especially components of genetic variation *viz.*, additive, dominance and epistasis effects affecting resistance to northern corn leaf blight and southern corn rust which helps in breeding of a multiple disease resistant cultivars. After ascertaining the genetics of resistance to foliar diseases, $F_{2:3}$ mapping

population developed from diverse parents CML153 and SKV50 segregating for northern corn leaf blight, sorghum downy mildew and southern corn rust diseases was used to quantify the quantitative inheritance through identification of QTLs implicated in resistance to these three foliar diseases. The results of the experiments are discussed under following headings.

5.1 Genetic architecture of resistance to northern corn leaf blight and southern corn rust

5.2 Evaluation of $F_{2:3}$ mapping population against multiple foliar pathogens

5.3 Genetic linkage map construction

5.4 QTL mapping for resistance to multiple foliar pathogen infestation

5.5 Co-localization of QTLs conferring resistance to three foliar diseases

5.1 Genetic architecture of resistance to northern corn leaf blight and southern corn rust in two crosses *viz.*, CM212 × SKV50 and HKI162 × SKV50 of maize

Efficient collection of genetic information and a rapid application of this information to breeding, is clearly a priority in a crop like maize. This is true of quantitative traits, which have genetic complexity and are subjected to environmental fluctuations. Phenotypic selection in these kind of traits is slow due to segregation at numerous loci and due to effects of environment on phenotype. Hence, in order to probe into various gene effects affecting quantitative traits in maize, six generation mean analysis (Hayman, 1958; Jinks and Jones, 1958; Mather and Jinks, 1971) is employed. Though various biometrical approaches are available to elucidate such information, generation mean analysis was used in the present study because of its relative simplicity and statistical

reliability since it is based on first order statistics. Further, detection, estimation and interpretation of non-allelic interaction has progressed much farther at the level of first degree statistics because their effects are less confounded. Kinds of experiments required for their analysis are both smaller and simpler.

In the present investigation, to know the genetics or inheritance of resistance to northern corn leaf blight and southern corn rust, six generation mean analysis was carried out in the crosses CM212 × SKV50 and HKI162 × SKV50. It is clearly indicated from the table 2 that parents are highly diverse for disease severity. Probably because of the diverse nature of the parents, even F_2 and backcross generations also exhibited a wide range in their reaction to northern corn leaf blight and southern corn rust incidence. The adequacy of simple additive-dominance model was tested by A, B, C and D scaling tests of Mather (1949) and joint scaling test of Cavalli (1952) which were significant in both crosses which indicated inadequacy of simple additive-dominance model to explain the observed variation in generation means for northern corn leaf blight and southern corn rust resistance thereby justifying the use of six parameter model for detection of interaction.

After ascertaining the failure of additive-dominance model perfect fit solution was fitted to estimate the magnitude and direction of the digenic interaction effects for the two crosses. The main gene effects such as additive [$\hat{d}$] and dominance [$\hat{h}$], and epistatic gene effects such as additive × additive [$\hat{i}$], additive × dominance [$\hat{j}$] dominance × dominance [$\hat{l}$] gene effects were estimated using perfect fit solutions (Mather and Jinks, 1971), the results of the same are summarised in table 5.

Northern corn leaf blight

The estimates of additive [$\hat{d}$] and dominance [$\hat{h}$] gene effects were significant for northern corn leaf blight suggesting the importance of both additive and dominance gene effects in the inheritance of resistance in both the crosses CM212 × SKV50 and HKI162 × SKV50. Among non-allelic interaction effects, predominance of additive × additive [$\hat{i}$] interaction effect in both crosses and dominance × dominance [$\hat{l}$] interaction effect in the cross CM212 × SKV50 was noticed in controlling the resistance to northern corn leaf blight. The observed differences could be attributed to resistance or susceptible gene frequencies and proportion of dominance and recessive genes possessed by parents (Viana *et al.*, 1999). Jha and Dhawan (1970), Hughes and Hooker (1971), Zhang *et al.* (1983), Sigulus *et al.* (1988), Shankarlingam *et al.* (1989), Pandurangegowda *et al.* (1993b), Mahajan *et al.* (1995), Hettiarachchi *et al.* (2009), Chaudhary and Mani (2010) and Njoroge and Gichuru (2013) also reported both additive as well as non-additive gene action in the expression of resistance to northern corn leaf blight. Duplicate gene interaction based on the significant opposite signs of [$\hat{h}$] and [$\hat{l}$] components was involved in the genetic control in both crosses. Duplicate gene interaction suggested that genetic gain is faster with mild selection and less rapid with very intense selection (Roy, 2000).

Southern corn rust

Preponderance of additive gene effect in the inheritance of southern corn rust was observed in the cross CM212 × SKV50 in which only additive [$\hat{d}$] effect was significant and additive × additive gene effect [$\hat{i}$] was significant in negative direction. Non-significance of dominance gene effect need not necessarily mean that dominance is completely absent, because such results may be obtained due to cancellation of

dominance gene effects at various loci. These results are in agreement with the findings of Pinho *et al.* (1999), So *et al.* (2003), Silva *et al.* (2001) and Vieira *et al.* (2011). In the cross HKI162 × SKV50, both additive and non-additive gene effects were found to be important wherein both [$\hat{d}$] and [$\hat{h}$] gene effects were significant. Among epistatic interaction effects additive × additive [$\hat{i}$] and dominance × dominance [$\hat{l}$] types were negative and significant. Prominence of additive and non-additive gene action in controlling resistance to southern corn rust was also reported by Paterniani *et al.* (2000), Brewbaker (2005), Josue (2007), Authrapun *et al.* (2009) and Brewbaker *et al.* (2011)

The operation of duplicate type of epistasis was noticed in both the crosses since the estimates of [$\hat{h}$] and [$\hat{l}$] had opposite signs. Presence of duplicate type of epistasis for southern corn rust was earlier reported by Ji (2006), Thaitad *et al.* (2008) and Brewbaker *et al.* (2011). Roy (2000) suggested that genetic gain for southern corn rust resistance is faster with moderate intensity of selection.

Number of effective factors and potence ratio

Minimum number of effective factors influencing a trait in F_2 generation and the degree of dominance computed from generation means of F_1 as well as F_2 generation in terms of potence ratio for two crosses CM212 × SKV50 and HKI162 × SKV50 for northern corn leaf blight and southern corn rust were estimated (Table 6) and briefly discussed below.

In the crosses CM212 × SKV50 and HKI162 × SKV50, northern corn leaf blight resistance was under the control of one group or two groups of effective factors. Hooker (1963), Ullstrup (1970), Hughes and Hooker (1971) and Patil *et al.* (1982) also revealed that the resistance to

northern corn leaf blight was conditioned by relatively less number of genes.

Potence ratio in F_1 and F_2 generation of both crosses CM212 × SKV50 and HKI162 × SKV50 revealed the preponderance of partial dominance in the genetic control of resistance to northern corn leaf blight and southern corn rust. These results are in confirmation with the expression of resistant reaction against *Exserohilum turcicum* and *Puccinia polysora* infection in F_1s whereas segregation and recombination in the segregating generations for resistance to northern corn leaf blight and southern corn rust in F_2s in the present study, which was attributed to partial dominance of trait.

In the present study, six generation mean analysis revealed that the genetic architecture of resistance to northern corn leaf blight and southern corn rust was population specific. Hence, appropriate breeding methods are to be adopted for the improvement of each population. Both additive and non-additive components are found important for northern corn leaf blight and southern corn rust resistance, hence recurrent selection programme preferably reciprocal recurrent selection is appropriate to develop improved resistant lines first and then exploit disease resistance in F_1s (Ceballos *et al.*, 1991 and Hettiarachchi *et al.*, 2009).

5.2 Evaluation of $F_{2:3}$ mapping population against multiple foliar pathogens

Highly diverse parents for multiple pathogen infection were used in the development of $F_{2:3}$ progenies which revealed highly significant differences among the progenies for disease reaction against northern corn leaf blight, sorghum downy mildew and southern corn rust pathogens.

To understand the effect of environment on development of these three foliar diseases, $F_{2:3}$ progenies developed from the cross CML153 x SKV50 were evaluated in *Kharif* 2012 and *Kharif* 2013 at ZARS, Mandya. In *Kharif* 2012, the environmental conditions were not congenial for southern corn rust and sorghum downy mildew disease symptom development, hence distribution of southern corn rust and sorghum downy mildew disease among the $F_{2:3}$ progenies was not uniform and the data from *Kharif* 2012 for sorghum downy mildew and southern corn rust were not included in the analysis.

The means of F_3 families for northern corn leaf blight indicated that there was significant difference in each season and pooled over two seasons. The significance of genotypic component of variance in both the seasons and pooled data confirmed the anticipation of genotypic variability within the population. The grand mean of infection of $F_{2:3}$ families in contrast to parental line means for each season indicated that a larger portion of the population tends to show moderate resistance to resistant reaction to northern corn leaf blight. Meanwhile, differences among means indicated that disease pressure varied between seasons *i. e., Kharif* 2012 had the highest disease pressure compared to *Kharif* 2013. In pooled analysis, genotype x season interaction was significant representing the influence of the season/environment on northern corn leaf blight incidence. Assuming random effects model, Bartlett's test proved the homogeneity of error mean sum of squares for Northern corn leaf blight data between *Kharif* 2012 and *Kharif* 2013. Therefore, data from these two seasons were pooled.

The existence of genotypic variability for sorghum downy mildew and southern corn rust incidence within F_3 progenies was revealed by the significant genotypic component of variance in *Kharif* 2013. The mean disease incidence data of $F_{2:3}$ families in contrast to parents mean indicated that a larger portion of the population tends to show

susceptible reaction to sorghum downy mildew and resistant reaction to southern corn rust.

5.2.1 Distribution of the population

The study of descriptive statistics gives basic idea of the breeding material. The characteristics of the $F_{2:3}$ population in respect of multiple disease incidence as indicated by descriptive statistics (Table 11) are discussed below.

The study of distribution properties such as coefficients of skewness and kurtosis provides insight about the nature of gene action (Fisher *et al.*, 1932) and number of genes controlling the trait (Robson, 1956), respectively. All the reported genetic analysis of quantitative traits are based on first degree (gene effects through generation mean analysis) and second degree (components of genetic variances through diallel, line × tester analysis, etc) statistics. Skewness and kurtosis are more powerful than variances which reveal interaction genetic effects (Choo and Reinbergs, 1982). The skewed distribution of a trait in general suggests that the trait is under the control of non-additive gene action, especially epistasis and influenced by environmental variables (Pooni *et al.*, 1977; Kimbeng and Bingham, 1998; Roy, 2000). While positive skewness is associated with complementary gene interactions, negative skewness is associated with duplicate (additive × additive) gene interactions. The genes controlling the trait with skewed distribution tend to be predominantly dominant irrespective of whether they have increasing or decreasing effect on the trait.

Frequency distribution of 344 F_3 progenies from the cross CML153 x SKV50 revealed non-normal distribution for the incidence of northern corn leaf blight, sorghum downy mildew and southern corn rust. Positively skewed distribution was observed for northern corn leaf blight in each season data and pooled data. Skewed distribution was observed

towards resistant parent SKV50 indicating the dominance of resistance. Schechert *et al.* (1999); Welz *et al.* (1999); Brown *et al.* (2001) and Asea *et al.* (2009) also reported approximately or near normal distribution in phenotypic data on $F_{2:3}$ population with the distribution skewed towards the resistant parent.

Distribution of sorghum downy mildew incidence in F_3 progenies was skewed towards susceptible parent CML153 indicating the dominance of susceptibility. This absence of a normal distribution in the phenotypic values of the mapping populations is usually common particularly for diseases such as downy mildews. These results are in agreement with the findings of Agrama *et al.* (1999), George *et al.* (2003) and Nair *et al.* (2005) who also reported the absence of a normal distribution in phenotypic data on RILs with the distribution skewed towards the susceptible parent.

Positively skewed frequency distribution of southern corn rust incidence was noticed in F_3 population and the distribution skewed towards resistant parent SKV50 indicating the dominance of resistance. However, the distribution was made near normal through arcsine transformation of the *per cent* disease incidence data. Approximately or near normal distribution in phenotypic data on $F_{2:3}$ population reported by Holland *et al.* (1998) and Brunelli *et al.* (2002) and the near normal distribution in phenotypic data on RILs was described by Jines *et al.* (2007); Brewbaker *et al.* (2011) and Wanlayaporn *et al.* (2013).

The traits with leptokurtic and platykurtic distribution are controlled by fewer and a large number of genes, respectively. Kurtosis is negative or close to zero in the absence of gene interactions and is positive in the presence of gene interactions (Pooni *et al.*, 1977; Choo and Reinbergs, 1982). Platykurtic and skewed distribution in $F_{2:3}$ population is an evidence for the involvement of relatively large number of

segregating genes and presence of gene interaction in the inheritance of northern corn leaf blight, sorghum downy mildew and southern corn rust resistance.

5.2.2 Genetic parameters in $F_{2:3}$ mapping population

Estimation of genetic variability parameters in a population is a pre-requisite for breeding programme aimed at improving yield, disease resistance and other important characters under consideration. Unless a major portion of variation is heritable, attempts to improve characters by selection would be futile. The success in a crop improvement programme depends on the ability of the breeder to define and assemble the required genetic variability and select for yield indirectly through yield associated and highly heritable characters after eliminating the environmental components of phenotypic variation (Mather, 1949). Therefore, it is necessary to have information on both phenotypic co-efficient of variation and genotypic co-efficient of variation, heritability which helps the breeder to predict the expected genetic advance possible by selection for characters.

The evaluation of segregating progenies helps in estimation of various genetic and non-genetic components of variance. The study of variability provides an opportunity for selecting the desirable genotypes. Heritability is a fraction of variance in phenotypic expression that arises from genetic effects. The nature of the selection units and sampling errors also influence greatly the magnitude of heritability estimates. The heritability estimates in segregating generations helps to know genetic variance, genotype-environment interaction and the progress expected from selection. Knowledge on genetic advance that is expected by applying selection pressure to a segregating population is useful in designing effective breeding programme.

Phenotypic co-efficient of variation (PCV) was higher than the genotypic co-efficient of variation (GCV) for northern corn leaf blight, sorghum downy mildew and southern corn rust indicating the scope for direct selection of resistant genotypes for particular disease. Similar findings have been reported for northern corn leaf blight (Hakiza *et al.*, 2004, Vieira *et al.*, 2009, Vieira *et al.*, 2011); sorghum downy mildew (Yen and Prasanna, 2001, Nair *et al.*, 2004b, Yen *et al.*, 2004, Phumichai *et al.*, 2012, Premalatha *et al.*, 2012, Rashid *et al.*, 2013) and for southern corn rust (So *et al.*, 2003).

High heritability with high genetic advance over mean for northern corn leaf blight, sorghum downy mildew and southern corn rust indicated the reliability of the estimates of variation between F_3 families and reasonable progress in selection is possible for disease resistance traits in this population. Similar results were reported for northern corn leaf blight by Freymark *et al.* (1993, 1994), Schechert *et al.* (1999), Hakiza *et al.* (2004), Balint-Kurti *et al.* (2010) and Zwonitzer *et al.* (2010); for sorghum downy mildew by Sabry *et al.* (2006), Phumichai *et al.* (2012) and Jampatong *et al.* (2013) and for southern corn rust by Holland *et al.* (1998), Jines *et al.* (2007), Brewbaker *et al.* (2011) and Wanlayaporn *et al.* (2013).

5.3 Genetic linkage map construction

The development of genetic linkage map will greatly expedite the ability of breeders to tag and follow the introgression of specific chromosome segments linked to desirable trait from different resistance sources into breeding lines. Linkage map indicates the position and relative genetic distances between markers along the chromosomes. The most important use of linkage map is to identify chromosomal locations containing genes and QTLs associated with traits of interest. Construction of genetic linkage map is necessary to apply marker-

assisted selection (MAS) tool in crop improvement programme. The reports on the construction of genetic linkage map in maize are numerous (Senior *et al.*, 1996, Chin *et al.*, 1996, Agrama *et al.*, 1999, Brown *et al.*, 2001, Nair *et al.*, 2001, George *et al.*, 2003, Jines *et al.*, 2007, Zwonitzer *et al.*, 2010, Jampatong *et al.*, 2013, Wanlayaporn *et al.*, 2013). Due to the availability of enormous genomic resources and high level of polymorphism there has been rapid progress in developing genetic and physical maps in maize (Davis *et al.*, 1999; Sharopova *et al.*, 2002).

In the present investigation, genotype frequencies among the 344 $F_{2:3}$ progenies satisfied the expected Mendelian segregation ratios for 194 SNP markers used to construct the linkage map. Ten linkage groups were formed using SNP markers which were very similar to the published maps at LOD score of 3. The total map distance covered about 2143.02 cM with 194 markers spanning all 10 chromosomes with an average interval length of 10.77 cM (Table 13). It is also important to mention that the genetic linkage map constructed in this study is relatively high density map. The markers used in the present investigation were verified with known positions on IBM2 Neighbor's consensus public linkage map (*http://www.maizegdb.org*).

5.4 QTL mapping for resistance to multiple foliar diseases

The importance of disease in reducing total maize yield and the diversity of pathogens affecting this crop have been reviewed recently in the light of worldwide food security (Ali and Yan, 2012). In that review, the importance of molecular breeding was highlighted as a central framework for overcoming supply shortage of maize because of disease. Among molecular breeding strategies, quantitative trait loci (QTL) identification which in turn helps in selection of resistant genotypes

through marker assisted selection (MAS) has proven to be a powerful tool for breeding resistant cultivars.

Several foliar diseases cause economic damage to maize, fortunately, the prevalence of these diseases varies depending on the region or season (Smith, 1999). Based on the economic importance and severity worldwide and in India, northern corn leaf blight, sorghum downy mildew and southern corn rust diseases were considered for identifying QTLs for resistance in this study.

Mapping population ($F_{2:3}$ progenies) which descended from parents contrasting for three foliar diseases is the main requirement to perform proper QTL analyses. This was clearly accomplished and the *per cent* disease incidence of the highly resistant parent was low and the other parent used in the study was highly susceptible (Table 10). These differences between parents were clearly noticeable in the field (Plate 2). Parental polymorphism is another requirement for dissection of QTLs. The *per cent* polymorphism of SNP markers between parents CML153 and SKV50 which were used in the development of mapping population to identify QTLs in the study was 25.35% (Table 13). Mapping population comprising of 344 $F_{2:3}$ progenies genotyped with 194 SNPs (Table 14) was used to identify QTLs for three diseases. The near normal distribution pattern of F_3 progenies suggested the reliability of the $F_{2:3}$ mapping population for identification of QTLs for resistance to northern corn leaf blight, sorghum downy mildew and southern corn rust.

5.4.1 Mapping of QTLs conferring resistance to northern corn leaf blight

In this study, genomic locations of QTLs for northern corn leaf blight were identified on chromosomes 2, 5 and 8. The QTLs on chromosomes 5 and 8 were major with high *per cent* of the phenotypic variance for resistance. The QTL detected on chromosome 8 at position

Table 20a: Comparison of QTLs identified with earlier reported QTLs for resistance to northern corn leaf blight

Mapping Population	Marker Type	Genome Coverage (cM)	Chromosome bin location	QTL position (cM)	Flanking Marker interval	Additive effect	R^2 (%)	Reference
F2:3 (344)	SNP (194)	2143.02	2.06	108.11	C00359-01 - MZA13360-13	1.25	2.28	Present Study
			5.03	53.21	MZA3103-47 - MZA533-46	0.74	1.77	
			5.04 – 5.05	142.71	MZA5296-6 - C00171-01	3.39	10.24	
			8.03	43.21	MZA2487-6	2.99	1.64	
			8.06	81.31	MZA6428-11 - MZA3856-10	0.56	16.34	
F2:3 (256)	RFLP (110)	1853.00	2.05-2.06	46.00	umc371 - umc381	0.27	9.40	Schechert et al. (1999)
			5.03	74.00	umcO0l - bn15.40	0.33	7.00	
			8.06	106.00	bn112.30 - umc323	0.29	11.80	
F2:3 (220)	RFLP (87) and SSR (7)	1799.00	5.03	86.00	Umc27a - Umc43	-3.43	6.50	Welz et al. (1999)
			5.04	112.00	csu36a - bnl7.71	-6.47	13.40	
			8.02-8.03	2.00	umc103a - bngl669	4.29	7.80	
			8.06	74.00	umc17b - npi268a	7.19	19.20	
F2:3	RFLP (110)	1853.00	5.03	74.00	bnl10.06 - bnl7.71	-16.30	15.00	Welz and Geiger (2000)
			5.04-5.05	136.00	bnl5.40 - npi461	-7.60	6.90	
			8.06	76.00	bnl7.08 - bnl8.26	-11.80	13.00	
F2:3 (157)	RFLP (88)	-	2.05-2.06	82.00	u131	–1.73	6.70	Brown et al. (2001)
			8.06	86.00	n268	–1.41	12.00	
IBM RILs (302)	SSR	-	2.06	37.03	bnlg2277 - umc1262	–0.42	4.80	BalintKurti et al. (2010)
			8.06	89.50	psb107a - bnlg666	0.70	10.40	
F2:3 (410)	SSR	-	5.04	92.00	phi330507 - umc1221	0.018	-	Asea et al. (2009)
			8.06	108.00	mmc0181 - umc1724	-0.064	-	
RILs (109)	SSR and SNP	1697.30	2.05-2.06	48.40	PZA03699.1 - PZA03228.4	0.20	7.80	Zwonitzer et al. (2010)
			8.02	26.30	PHM9695.8 - PZA02955.3	0.27	12.80	
			8.05-8.06	76.20	PHM3993.28 - PZA03612.2	0.19	8.30	

**Values in parenthesis are mapping population size and number of markers*

81.31 cM with LOD score of 3.13 explained 16.34% of the phenotypic variance followed by the QTL on chromosome 5 at position 142.71 with LOD value of 3.40 explained 10.24 *per cent* in combined analysis. When considered together total variance explained by these QTLs was 26.58 *per cent*.

The UMC reference map of maize (Davis *et al.*, 1999) allows comparision of QTL positions across different experiments and different genetic backgrounds (Lin *et al.*, 1995; Tuberosa *et al.*, 2002). The major QTLs on chromosome bins 5.04-5.05 and 8.06 were also reported by Freymark *et al.* (1993, 1994) in bins 5.03-5.05 and 8.03-8.06; Dingersidissen *et al.* (1996) in bins 5.03 and 8.06; Schechert *et al.* (1999) in bins 5.03-5.05 and 8.06; Welz *et al.* (1999) in bins 5.03-5.04 and 8.02-8.06; Welz and Geiger, (2000) in bins 5.04 and 8.06; Brown *et al.* (2001) in bin 8.06; Asea *et al.* (2009) in bins 5.04 and 8.06 and Chung *et al.* (2011) in bins 5.03 and 8.06 (Table 20a). This supports the suggestion from Chung *et al.* (2010) who fine mapped northern corn leaf blight QTL on chromosome bin 8.06, that a major QTL region on chromosome 8 affects the response to northern corn leaf blight. Similarly important role of QTLs on chromosome 5 at bin 4 was deciphered as an additional QTL to QTLs already known in bins 5.03, 5.04 and 5.05 by Welz *et al.* (1999); Asea *et al.* (2009) and Chung *et al.* (2011).

Another QTL in the present study in the chromosome bin 2.06 was also reported earlier by Schechert *et al.* (1999); Welz *et al.* (1999); Brown *et al.* (2001); Ping *et al.* (2007); Balint-Kurti *et al.* (2010) and Zwonitzer *et al.* (2010). On the other hand, QTLs located on chromosomes 1, 3, 4, 6, 7 and 9 described in other studies were not found in our studies (Schechert *et al.*, 1999; Welz *et al.*, 1999; Brown *et al.*, 2001; Balint-Kurti *et al.*, 2010; Zwonitzer *et al.*, 2010 and Chung *et al.*, 2011).

Critical analysis of all available results indicated that partial dominance (PD), dominance (D) and over dominance (OD) were most frequent for resistance to northern corn leaf blight. The major QTL identified in our study at bin location 8.06 had an over dominant (OD) gene action. It could be confirmed that northern corn leaf blight resistance alleles in SKV50 are a good source for MAS supported maize breeding program. Similar findings were observed by Welz *et al.* (1999); Welz and Geiger, (2000); Brown *et al.* (2001); Chung *et al.* (2010); Zwonitzer *et al.* (2010) and Chung *et al.* (2011).

One of the major goals of QTL mapping for resistance to northern corn leaf blight is to locate markers that can be broadly used for MAS in a breeding program. One major concern against using MAS has been the lack of consistency of QTLs across environments. Results from Stuber *et al.* (1987) suggested the use of QTLs with little QTL × environment interaction. This was substantiated in the present study as QTLs in bin 2.06 and 8.06 were detected in both the seasons and in pooled analysis. Earlier workers (Brown *et al.*, 2001, Asea *et al.*, 2009 and Balint-Kurti *et al.*, 2010) reported the most likelihood peaks that were identified in all environments and differed only in the level of significance and the size of estimated genetic effects. Conclusively the markers associated with QTLs in bin 2.06 (C00359-01 and MZA13360-13) and 8.06 (MZA6428-11 and MZA3856-10) are the favourites to be used to transfer resistance alleles to susceptible lines (Table 15). Marker assisted selection will be more effective in delivering such lines, and it will dramatically minimize expensive and laborious field screening (Gupta *et al.*, 2008; Ali and Yan, 2012). An approach integrating both phenotypic selection and MAS would be more effective in order to enhance northern corn leaf blight resistance in maize.

5.4.2 QTL mapping for resistance to sorghum downy mildew

Quantitative trait loci (QTLs) for resistance to sorghum downy mildew were located on chromosomes 2, 3, 4, 5 and 8 in the present study. The QTLs on chromosomes 2 and 3 were major with high *per cent* of the phenotypic variance for resistance with 35.85% and 8.28%, respectively. The two QTLs detected on chromosome 2 at position 147.61 and 154.31 cM with LOD score of 2.57 and 2.63 collectively explained 35.85 *per cent* of the total phenotypic variance. The QTL on chromosome 3 at position 100.11 cM with LOD value of 2.56 explained 8.28 *per cent* of the phenotypic variance followed by QTL on chromosome 5 at position 138.71 with LOD value of 3.22 and 5.98 *per cent* of the phenotypic variance. When considered together, total variance explained by all these QTLs was 50.11 *per cent*. The major QTL on chromosome 2 was also reported by Sabry *et al.* (2006) which explained 70 *per cent* phenotypic variation. Nair *et al.* (2005) and Jampatong *et al.* (2013) also identified major QTLs on chromosome 3 and chromosome 5 thereby verifying the significance of QTLs identified in the present study (Table 20b).

The QTLs for sorghum downy mildew resistance have been identified till now on nine of ten maize chromosomes, indicating a complex genetic situation. As in this study, QTLs located in the bins 2.05 and 2.06 were also detected in bins 2.04 (Nair *et al.* 2005), 2.06 (George *et al.*, 2003; Phumichai *et al.*, 2012) and 2.09 (George *et al.*, 2003; Sabry *et al.*, 2006; Phumichai *et al.*, 2012; Jampatong *et al.*, 2013). This supports the suggestion from Sabry *et al.* (2006), that a major region on chromosome 2 affects the response to downy mildew. Similarly important role of QTLs on chromosome 3 at bin 4 was interpreted as an additional QTL to QTLs already known in bins 3.04 by Nair *et al.* (2005) and Jampatong *et al.* (2013), respectively.

Table 20b: Comparison of QTLs identified with earlier reported QTLs conferring resistance to sorghum downy mildew

Mapping Population	Marker Type	Genome Coverage (cM)	Chromosome bin location	QTL position (cM)	Flanking Marker interval	Additive effect	R^2 (%)	Reference
F2:3 (344)	SNP (194)	2143.02	2.05-2.06	147.61	C00324-01 - MZA3668-12	3.11	20.42	Present Study
			2.06	154.31	C00324-01 - MZA3668-12	-2.88	15.43	
			3.04	100.11	MZA1959-26 - MZA2919-23	8.13	8.28	
			5.03-5.04	138.71	C00303-01 - MZA5296-6	3.44	5.98	
RILs (135)	RFLP (143)	2117.00	2.06	158	umc55a – csu154a	4.00	3.40	George *et al.* (2003)
BC1F2 (177)	SSR (89)	1681.50	2.04–2.05	0.01	bnlg1018 - bnlg371	-4.98	5.40	Nair et al. (2005)
			3.04–3.05	55.6	umc1223-bnlg420	-7.13	14.90	
F2 (220)	RFLP (60)	1265.00	3.04	-	phi073 - bnlg1350	-	4.00	Sabry et al. (2006)
F2:3 (251)	SSR (195)	2415.20	3.04	157.81	umc2002 - umc1117	5.60	6.79	Jampatong et al. (2013)
			5.03	114.01	bnlg1902	11.52	10.60	

**Values in parenthesis are mapping population size and number of markers*

The other two QTLs identified in the present investigation in the bins 4.08 and 5.03-5.04 were in correspondence with QTLs reported by Jampatong *et al.* (2013). Two new QTLs were located in the chromosome bin 8.03 and which were not published before. On the other hand, fifteen QTLs described in other studies were not found in our studies (Agrama *et al.*, 1999; George *et al.*, 2003; Nair *et al.*, 2005; Sabry *et al.*, 2006; Phumichai *et al.*, 2012 and Jampatong *et al.*, 2013).

A cross-comparison of all available results indicated that partial dominance (PD) and additive gene action were most frequent for resistance to sorghum downy mildew. The major QTLs identified in the present study at bin location 2.05-2.06 had a partial dominant (PD) gene action. Sorghum downy mildew resistance alleles from the resistant parent SKV50 are useful for effective marker assisted selection (George *et al.*, 2003; Sabry *et al.*, 2006 and Jampatong *et al.*, 2013) while sorghum downy mildew resistant alleles in CML153 are also a good contender for MAS supported maize breeding program as described by Nair *et al.* (2005).

5.4.3 Identification of QTLs for southern corn rust resistance

The *per cent* southern corn rust disease severity of the highly resistant parent was low and the other parent used in the study was highly susceptible (73.66 *per cent*), this shows that highly contrasting parents for southern corn rust severity were used for development of mapping population for identification of QTLs conditioning resistance to southern corn rust.

From the present investigation, seven QTLs were identified on chromosomes 3, 8, 9 and 10 which conditioned the resistance to southern corn rust. The major QTL detected on chromosome 10 at 54.41 cM with LOD of 9.36 explained 18.59% of the phenotypic variance. This major QTL was located on short arm of chromosome 10 (bin 10.01-10.03)

Table 20c: Comparison of QTLs identified with previously reported QTLs for southern corn rust resistance

Mapping Population	Marker Type	Genome Coverage (cM)	Chromosome bin location	QTL position (cM)	Flanking Marker interval	Additive effect	R^2 (%)	Reference
F2:3 (344)	SNP (194)	2143.02	3.04	18.01	C00278-01	15.54	2.55	Present Study
			8.03	22.71	MZA2487-6	1.67	4.17	
			8.03	48.21	MZA2487-6 - MZA5637-15	8.19	4.71	
			9.03	67.51	MZA4720-12 - C00090-01	-2.62	4.67	
			10.03	54.41	MZA15331-16 - MZA3922-32	20.84	18.59	
F2 (307)	RFLP (86)	-	3.04	64.00	Umc3 - Umc96	-	6.90	Jiang et al. (1999)
			9.03	55.00	Umc53 - Bnl5.09	-	5.60	
RILs (143)	SSR (113)	1993	8.03	51.72	Umc1360 - Umc1034	0.16	2.00	Jines et al. (2007)
			9.01	31.42	Bnlg1401 - Phi022	-0.14	1.50	
			10.00	6.01	Umc1380 - Bnlg1451	1.27	82.70	
RILs (89)	SSR (157)	2123.1	9.01	20.00	MACT2B8 - phi068	−0.89	18.70	Wanlayaporn et al. (2013)
			10.03	3.90	umc1246 - umc1239	−1.06	15.20	

**Values in parenthesis are mapping population size and number of markers*

in different populations as reported by Chen *et al.* (2004); Jiang *et al.* (1999); Holland *et al.* (1998); Jines *et al.* (2007) and Wanlayaporn *et al.* (2013). All these QTLs were tightly linked with Rpp9 gene which is resistant to southern corn rust race PP.9 and also adjacently linked to Rp1, common rust resistance gene, located on short arm of chromosome10.

Other QTLs in this study, located in the bins 3.04 and 3.05 were also detected earlier in bins 3.05 (Holland *et al.*, 1998) and in 3.04 (Jiang *et al.*, 1999); QTLs in the bins 8.03 and 9.03 corresponded with the QTLs reported by Brunelli *et al.* 2002 (in bin 9.03); Jines *et al.* 2007 (in bins 8.03 and 9.01) and Wanlayaporn *et al.* 2013 (in bins 9.01) (Table 20c).

Preponderance of partial dominance (PD), dominance (D) and over dominance (OD) gene action was most frequent for resistance to southern corn rust. The present results indicated that partial dominant (PD) gene action was associated with major QTL regions in the bin 10.03. The donor rust resistant alleles coming from both the parent's *viz.*, resistant parent SKV50 and susceptible parent CML153 are important for marker assisted selection for rust resistance as described by Jines *et al.* (2007) and Wanlayaporn *et al.* (2013).

The lack of a common element between the QTLs identified for northern corn leaf blight, sorghum downy mildew and southern corn rust in different populations could be due to a combination of various factors, including: (1) the type and size of the mapping population used; (2) segregation of different sets of QTLs in different crosses; (3) detection of a QTL in a segregating population only if both parental lines contributed different alleles of the QTL; (4) epistatic interaction between QTLs in each of the mapping populations (Beavis and Keim 1996; Kearsey and Pooni, 1996; Bohn *et al.*,1997). Beavis *et al.* (1991) recorded that a comparison of data for QTL localization in different segregating populations for

characters such as disease resistance reveals only a few QTLs that are common across populations. This is particularly relevant because of the fact that different climatic and growing conditions at individual environments might affect the expression of QTL involved in developmental, morphological and chemical characters affecting resistance against specific pathogens. Despite these possibilities, the detection of major QTLs for resistance against specific pathogen(s) contributed by diverse resistant lines could provide an opportunity for pyramiding multiple genes that might control different components of resistance.

5.5 Co-localization of QTLs conferring resistance to three foliar diseases

In maize, enormous amount of valuable information exists in the literature concerning QTL conditioning resistance to diseases that may be exploited to improve durable multiple disease resistance (MDR). Multiple disease resistance (MDR), in which the same chromosome locus conditioning resistance to multiple pathogens, is both practically and conceptually important.

Developing maize inbred lines with multiple disease resistance is a high priority in many breeding programs in the areas where increasing intensity of maize production has resulted in maize being produced essentially year-round in many areas with environments that are favourable to disease development. This was achieved by clustering of disease resistance genes in maize and in other crops (McMullen and Simcox, 1995; Pflieger *et al.*, 2001; Yuan *et al.*, 2003; Randall *et al.*, 2005) and suggests several potential applications to enhance the efficiency of marker assisted selection for pyramiding resistance genes as emphasized in integrated disease management. It is interesting to note that some QTL characterized as having minor effects are located on the

same bin positions for major QTL for other diseases. Identification of mapped resistance loci for multiple pathogens provides the opportunity for co-localizing resistance loci. Such resistance gene combinations could provide more durable protection (Simmonds, 1985) against the variable number of pathogens than resistance conferred to single pathogen only. In the present study, QTLs for northern corn leaf blight, sorghum downy mildew and southern corn rust resistance were co-localized based on the bin locations and chromosomal regions where QTLs were detected.

Associations between resistances to northern corn leaf blight, sorghum downy mildew and southern corn rust was significant in the $F_{2:3}$ mapping population segregating for multiple disease incidence (table 18) and these associations were likely caused either by alleles that confer high levels of resistance to one disease and lower levels to others whose effects are undetectable by QTL analysis, or by alleles that confer low gain undetectable by QTL analysis with varied levels of resistance to two or more diseases. Thirteen QTLs associated with resistance to two or more diseases (Table 19 and Fig. 10) would seem to provide good evidence for loci conditioning resistance to one or more diseases, either by carrying a single gene conferring resistance to two or more diseases or two closely linked genes conferring disease-specific resistances to different diseases in $F_{2:3}$ population.

The inbred SKV50 with resistance to foliar diseases *viz*., northern corn leaf blight, sorghum downy mildew and southern corn rust can be used for pyramiding resistance QTLs. The study indicated the potential of using several target QTLs present in the chromosome bin 8.03 for resistance to northern corn leaf blight, sorghum downy mildew and southern corn rust; bins 5.03-5.04, 2.06 for resistance to northern corn leaf blight and sorghum downy mildew and bin 3.04 for resistance to sorghum downy mildew and southern corn rust for marker assisted

selection to pyramid quantitative resistance to multiple foliar pathogens as described by Pratt and Gordon (2006).

Future line of work

1. Genetics of resistance to northern corn leaf blight and southern corn rust unravelled from the present study may be confirmed by using more number of crosses having large population size.

2. Reciprocal recurrent selection can be followed to improve northern corn leaf blight and southern corn rust disease resistance in both the populations of maize CM212 x SKV50 and HKI162 x SKV50.

3. Resistant F_3 progenies identified in the study can be forwarded for further generations to obtain resistant inbred lines.

4. Pyramiding of major QTLs identified on the chromosome 5 and 8 (for resistance to northern corn leaf blight); QTLs on chromosome 2 and 3 (for resistance to sorghum downy mildew) and QTL on the chromosome 10 (for resistance to southern corn rust) into susceptible inbreds.

5. The multiple disease resistant loci on chromosome bin 8.03 with common flanking marker MZA2487-6 can be used to pyramid quantitative resistance into elite susceptible breeding lines against multiple foliar pathogen infestation.

Summary

VI. SUMMARY

The present investigation was undertaken during *Kharif* 2011, *Rabi* 2011-12, summer 2012, *Kharif* 2012, *Rabi* 2012-13 and *Kharif* 2013 at the Zonal Agricultural Research Station, V. C. Farm, Mandya. The objective was to study the genetics of resistance to northern corn leaf blight and southern corn rust in two crosses of maize *viz.*, CM212 × SKV50 and HKI162 × SKV50 through generation mean analysis and identification of QTLs conferring resistance and co-localization of QTLs conditioning resistance to three foliar diseases *viz.*, northern corn leaf blight, sorghum downy mildew and southern corn rust using $F_{2:3}$ mapping population.

The salient features of the present study on maize are summarized below.

1. The scaling tests as well as joint scaling tests revealed that the inadequacy of simple additive-dominance model and justifying the use of six parameter model for the detection of gene interactions in both the crosses CM212 × SKV50 and HKI162 × SKV50 for resistance to northern corn leaf blight and southern corn rust.

2. The six generation mean analysis indicated significance of additive, dominance and additive × additive gene effects for northern corn leaf blight and southern corn rust. Based on the signs of [$\hat{h}$] and [$\hat{l}$] gene effects, duplicate gene interaction was evident in the inheritance of northern corn leaf blight and southern corn rust in both the crosses of maize CM212 × SKV50 and HKI162 × SKV50.

3. Minimum number of effective factors estimated in respect of northern corn leaf blight and southern corn rust ranged from one group or two groups in these two crosses of maize.

4. In both the crosses, the degree of dominance in terms of potence ratio for northern corn leaf blight and southern corn rust resistance indicated that the genetic control of resistance was under partial dominance, since the value of potence ratio was less than one.

5. The $F_{2:3}$ mapping population was developed using two contrasting inbred lines for disease reaction *viz.*, CML153 (susceptible) and SKV50 (resistant) was phenotyped during *Kharif* 2012 and *Kharif* 2013 in the national disease screening nurseries for northern corn leaf blight, sorghum downy mildew and southern corn rust disease.

6. Analysis of variance showed significant variation among the progenies, seasons and genotype × season interaction for northern corn leaf blight incidence indicating that performance of progenies was different in different seasons and seasons were adequately different with respect to weather conditions. Significant seasonal effect observed in the analysis indicated that the disease development was highly influenced by weather conditions *viz.*, rainfall, temperature and relative humidity.

7. Presence of genotypic variability was noticed for sorghum downy mildew and southern corn rust incidence within F_3 progenies, as evident from the significant variation among the progenies in the ANOVA during *Kharif* 2013.

8. In F_3 progenies, the *per cent* disease incidence showed high PCV and GCV for all three diseases *viz.*, northern corn leaf blight, sorghum downy mildew and southern corn rust suggesting ample scope for selection for disease resistance. High heritability coupled with high GAM observed for these three foliar diseases indicated substantial amount of heritable variation for the trait.

9. Frequency distribution of F_3 progenies did not show normal distribution for disease incidence. Distribution of northern corn leaf blight and southern corn rust skewed towards resistant parent SKV50 indicating dominance of resistance, while in case of sorghum downy mildew distribution was skewed towards susceptible parent CML153 indicating dominance of susceptibility. However, the distribution was made near normal through arcsine transformation of the per cent disease incidence data.

10. Linkage analysis using 194 polymorphic markers in $F_{2:3}$ progenies revealed 10 linkage groups spanning 2143.02 cM with average distance of 10.77 cM.

11. The QTL analysis revealed two QTLs *viz.*, *qNCLB-8-1* and *qNCLB-8-2* for resistance to northern corn leaf blight were detected as consensus QTLs in both the seasons and in pooled data. The major QTL (*qNCLB-8-2*) flanked by markers MZA6428-11 – MZA3856-10 contributed highest (16.34%) phenotypic variance for northern corn leaf blight resistance and the other QTL *qNCLB-5-3* flanked by markers MZA5296-6 – C00171-01 explained the phenotypic variance of 10.24 *per cent*.

12. Three major QTLs (*qsdm-2-1* and *qsdm-2-2* in bin 2.06 collectively explained the phenotypic variation of 35.85%; *qsdm*-3 in bin 3.04 explained 8.28% variation) conferring resistance to sorghum downy mildew were identified. These markers and QTLs can be used for MAS for sorghum downy mildew resistance.

13. With phenotypic variation of 18.59 *per cent*, one major QTL (*qscr-10*) conditioning resistance to southern corn rust was mapped onto chromosome 10 in the marker interval MZA15331-16 – MZA3922-32.

14. QTLs were detected in bin 5.04-5.05 and 8.06 for northern corn leaf blight; in bin 2.06 and 3.04 for sorghum downy mildew and in bin 10.01-10.03 for southern corn rust and the markers associated with these QTLs are required to be used to transfer resistance alleles to susceptible lines.

15. Significant pairwise correlation/association observed between northern corn leaf blight, sorghum downy mildew and southern corn rust incidence indicated that there are loci (and possibly genes) conferring multiple disease resistance in the population studied.

16. Co-localization of QTLs for resistance to multiple foliar diseases was observed in bin 8.03 wherein QTLs conferring resistance to northern corn leaf blight (one QTL), sorghum downy mildew (two QTLs) and southern corn rust (two QTLs) were located with common adjacent marker MZA2487-6.

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Appendices

Appendix 1: List of the 344 $F_{2:3}$ progenies developed from the cross CML153 × SKV50

Sl. No.	Pedigree	Sl. No.	Pedigree	Sl. No.	Pedigree
1	(CML153 × SKV50)-1	46	(CML153 × SKV50)-85	91	(CML153 × SKV50)-171
2	(CML153 × SKV50)-3	47	(CML153 × SKV50)-86	92	(CML153 × SKV50)-173
3	(CML153 × SKV50)-6	48	(CML153 × SKV50)-91	93	(CML153 × SKV50)-174
4	(CML153 × SKV50)-7	49	(CML153 × SKV50)-92	94	(CML153 × SKV50)-178
5	(CML153 × SKV50)-8	50	(CML153 × SKV50)-93	95	(CML153 × SKV50)-180
6	(CML153 × SKV50)-9	51	(CML153 × SKV50)-94	96	(CML153 × SKV50)-182
7	(CML153 × SKV50)-10	52	(CML153 × SKV50)-96	97	(CML153 × SKV50)-185
8	(CML153 × SKV50)-12	53	(CML153 × SKV50)-99	98	(CML153 × SKV50)-187
9	(CML153 × SKV50)-16	54	(CML153 × SKV50)-101	99	(CML153 × SKV50)-188
10	(CML153 × SKV50)-17	55	(CML153 × SKV50)-103	100	(CML153 × SKV50)-189
11	(CML153 × SKV50)-18	56	(CML153 × SKV50)-104	101	(CML153 × SKV50)-191
12	(CML153 × SKV50)-19	57	(CML153 × SKV50)-105	102	(CML153 × SKV50)-192
13	(CML153 × SKV50)-21	58	(CML153 × SKV50)-107	103	(CML153 × SKV50)-194
14	(CML153 × SKV50)-22	59	(CML153 × SKV50)-108	104	(CML153 × SKV50)-195
15	(CML153 × SKV50)-23	60	(CML153 × SKV50)-113	105	(CML153 × SKV50)-197
16	(CML153 × SKV50)-28	61	(CML153 × SKV50)-114	106	(CML153 × SKV50)-198
17	(CML153 × SKV50)-29	62	(CML153 × SKV50)-115	107	(CML153 × SKV50)-200
18	(CML153 × SKV50)-30	63	(CML153 × SKV50)-116	108	(CML153 × SKV50)-201
19	(CML153 × SKV50)-31	64	(CML153 × SKV50)-117	109	(CML153 × SKV50)-203
20	(CML153 × SKV50)-34	65	(CML153 × SKV50)-119	110	(CML153 × SKV50)-204
21	(CML153 × SKV50)-35	66	(CML153 × SKV50)-120	111	(CML153 × SKV50)-205
22	(CML153 × SKV50)-36	67	(CML153 × SKV50)-124	112	(CML153 × SKV50)-206
23	(CML153 × SKV50)-39	68	(CML153 × SKV50)-126	113	(CML153 × SKV50)-207
24	(CML153 × SKV50)-41	69	(CML153 × SKV50)-127	114	(CML153 × SKV50)-208
25	(CML153 × SKV50)-45	70	(CML153 × SKV50)-129	115	(CML153 × SKV50)-209
26	(CML153 × SKV50)-46	71	(CML153 × SKV50)-132	116	(CML153 × SKV50)-212
27	(CML153 × SKV50)-47	72	(CML153 × SKV50)-133	117	(CML153 × SKV50)-214
28	(CML153 × SKV50)-48	73	(CML153 × SKV50)-134	118	(CML153 × SKV50)-217
29	(CML153 × SKV50)-49	74	(CML153 × SKV50)-136	119	(CML153 × SKV50)-218
30	(CML153 × SKV50)-52	75	(CML153 × SKV50)-139	120	(CML153 × SKV50)-222
31	(CML153 × SKV50)-55	76	(CML153 × SKV50)-141	121	(CML153 × SKV50)-224
32	(CML153 × SKV50)-58	77	(CML153 × SKV50)-142	122	(CML153 × SKV50)-225
33	(CML153 × SKV50)-60	78	(CML153 × SKV50)-143	123	(CML153 × SKV50)-228
34	(CML153 × SKV50)-61	79	(CML153 × SKV50)-145	124	(CML153 × SKV50)-230
35	(CML153 × SKV50)-62	80	(CML153 × SKV50)-146	125	(CML153 × SKV50)-235
36	(CML153 × SKV50)-63	81	(CML153 × SKV50)-149	126	(CML153 × SKV50)-237
37	(CML153 × SKV50)-64	82	(CML153 × SKV50)-150	127	(CML153 × SKV50)-238
38	(CML153 × SKV50)-65	83	(CML153 × SKV50)-153	128	(CML153 × SKV50)-239
39	(CML153 × SKV50)-69	84	(CML153 × SKV50)-154	129	(CML153 × SKV50)-240
40	(CML153 × SKV50)-70	85	(CML153 × SKV50)-157	130	(CML153 × SKV50)-241
41	(CML153 × SKV50)-75	86	(CML153 × SKV50)-158	131	(CML153 × SKV50)-242
42	(CML153 × SKV50)-79	87	(CML153 × SKV50)-161	132	(CML153 × SKV50)-244
43	(CML153 × SKV50)-80	88	(CML153 × SKV50)-162	133	(CML153 × SKV50)-245
44	(CML153 × SKV50)-81	89	(CML153 × SKV50)-169	134	(CML153 × SKV50)-246
45	(CML153 × SKV50)-82	90	(CML153 × SKV50)-170	135	(CML153 × SKV50)-248

Appendix 1 Cont'd……

Sl. No.	Pedigree	Sl. No.	Pedigree	Sl. No.	Pedigree
136	(CML153 × SKV50)-250	181	(CML153 × SKV50)-327	226	(CML153 × SKV50)-408
137	(CML153 × SKV50)-251	182	(CML153 × SKV50)-329	227	(CML153 × SKV50)-413
138	(CML153 × SKV50)-252	183	(CML153 × SKV50)-330	228	(CML153 × SKV50)-417
139	(CML153 × SKV50)-253	184	(CML153 × SKV50)-331	229	(CML153 × SKV50)-418
140	(CML153 × SKV50)-255	185	(CML153 × SKV50)-332	230	(CML153 × SKV50)-419
141	(CML153 × SKV50)-256	186	(CML153 × SKV50)-335	231	(CML153 × SKV50)-423
142	(CML153 × SKV50)-257	187	(CML153 × SKV50)-338	232	(CML153 × SKV50)-424
143	(CML153 × SKV50)-258	188	(CML153 × SKV50)-342	233	(CML153 × SKV50)-426
144	(CML153 × SKV50)-259	189	(CML153 × SKV50)-343	234	(CML153 × SKV50)-427
145	(CML153 × SKV50)-260	190	(CML153 × SKV50)-345	235	(CML153 × SKV50)-431
146	(CML153 × SKV50)-262	191	(CML153 × SKV50)-346	236	(CML153 × SKV50)-433
147	(CML153 × SKV50)-265	192	(CML153 × SKV50)-347	237	(CML153 × SKV50)-442
148	(CML153 × SKV50)-266	193	(CML153 × SKV50)-348	238	(CML153 × SKV50)-451
149	(CML153 × SKV50)-268	194	(CML153 × SKV50)-351	239	(CML153 × SKV50)-452
150	(CML153 × SKV50)-269	195	(CML153 × SKV50)-352	240	(CML153 × SKV50)-453
151	(CML153 × SKV50)-270	196	(CML153 × SKV50)-353	241	(CML153 × SKV50)-468
152	(CML153 × SKV50)-271	197	(CML153 × SKV50)-354	242	(CML153 × SKV50)-469
153	(CML153 × SKV50)-272	198	(CML153 × SKV50)-355	243	(CML153 × SKV50)-473
154	(CML153 × SKV50)-274	199	(CML153 × SKV50)-356	244	(CML153 × SKV50)-474
155	(CML153 × SKV50)-276	200	(CML153 × SKV50)-357	245	(CML153 × SKV50)-476
156	(CML153 × SKV50)-279	201	(CML153 × SKV50)-362	246	(CML153 × SKV50)-477
157	(CML153 × SKV50)-283	202	(CML153 × SKV50)-363	247	(CML153 × SKV50)-485
158	(CML153 × SKV50)-286	203	(CML153 × SKV50)-364	248	(CML153 × SKV50)-486
159	(CML153 × SKV50)-287	204	(CML153 × SKV50)-365	249	(CML153 × SKV50)-487
160	(CML153 × SKV50)-289	205	(CML153 × SKV50)-367	250	(CML153 × SKV50)-491
161	(CML153 × SKV50)-290	206	(CML153 × SKV50)-371	251	(CML153 × SKV50)-494
162	(CML153 × SKV50)-292	207	(CML153 × SKV50)-372	252	(CML153 × SKV50)-498
163	(CML153 × SKV50)-294	208	(CML153 × SKV50)-375	253	(CML153 × SKV50)-499
164	(CML153 × SKV50)-295	209	(CML153 × SKV50)-380	254	(CML153 × SKV50)-500
165	(CML153 × SKV50)-296	210	(CML153 × SKV50)-381	255	(CML153 × SKV50)-501
166	(CML153 × SKV50)-298	211	(CML153 × SKV50)-382	256	(CML153 × SKV50)-505
167	(CML153 × SKV50)-301	212	(CML153 × SKV50)-383	257	(CML153 × SKV50)-511
168	(CML153 × SKV50)-302	213	(CML153 × SKV50)-384	258	(CML153 × SKV50)-515
169	(CML153 × SKV50)-303	214	(CML153 × SKV50)-386	259	(CML153 × SKV50)-516
170	(CML153 × SKV50)-309	215	(CML153 × SKV50)-389	260	(CML153 × SKV50)-517
171	(CML153 × SKV50)-310	216	(CML153 × SKV50)-390	261	(CML153 × SKV50)-520
172	(CML153 × SKV50)-311	217	(CML153 × SKV50)-391	262	(CML153 × SKV50)-524
173	(CML153 × SKV50)-314	218	(CML153 × SKV50)-393	263	(CML153 × SKV50)-525
174	(CML153 × SKV50)-315	219	(CML153 × SKV50)-396	264	(CML153 × SKV50)-526
175	(CML153 × SKV50)-317	220	(CML153 × SKV50)-400	265	(CML153 × SKV50)-529
176	(CML153 × SKV50)-319	221	(CML153 × SKV50)-402	266	(CML153 × SKV50)-531
177	(CML153 × SKV50)-320	222	(CML153 × SKV50)-403	267	(CML153 × SKV50)-533
178	(CML153 × SKV50)-321	223	(CML153 × SKV50)-404	268	(CML153 × SKV50)-538
179	(CML153 × SKV50)-322	224	(CML153 × SKV50)-405	269	(CML153 × SKV50)-540
180	(CML153 × SKV50)-324	225	(CML153 × SKV50)-406	270	(CML153 × SKV50)-541

Appendix 1 Cont'd……

Sl. No.	Pedigree	Sl. No.	Pedigree
271	(CML153 × SKV50)-542	316	(CML153 × SKV50)-643
272	(CML153 × SKV50)-543	317	(CML153 × SKV50)-644
273	(CML153 × SKV50)-546	318	(CML153 × SKV50)-646
274	(CML153 × SKV50)-548	319	(CML153 × SKV50)-648
275	(CML153 × SKV50)-551	320	(CML153 × SKV50)-652
276	(CML153 × SKV50)-554	321	(CML153 × SKV50)-653
277	(CML153 × SKV50)-555	322	(CML153 × SKV50)-655
278	(CML153 × SKV50)-559	323	(CML153 × SKV50)-657
279	(CML153 × SKV50)-561	324	(CML153 × SKV50)-667
280	(CML153 × SKV50)-562	325	(CML153 × SKV50)-668
281	(CML153 × SKV50)-563	326	(CML153 × SKV50)-669
282	(CML153 × SKV50)-565	327	(CML153 × SKV50)-673
283	(CML153 × SKV50)-568	328	(CML153 × SKV50)-674
284	(CML153 × SKV50)-573	329	(CML153 × SKV50)-681
285	(CML153 × SKV50)-577	330	(CML153 × SKV50)-683
286	(CML153 × SKV50)-578	331	(CML153 × SKV50)-685
287	(CML153 × SKV50)-579	332	(CML153 × SKV50)-687
288	(CML153 × SKV50)-581	333	(CML153 × SKV50)-697
289	(CML153 × SKV50)-585	334	(CML153 × SKV50)-699
290	(CML153 × SKV50)-587	335	(CML153 × SKV50)-700
291	(CML153 × SKV50)-588	336	(CML153 × SKV50)-702
292	(CML153 × SKV50)-589	337	(CML153 × SKV50)-703
293	(CML153 × SKV50)-591	338	(CML153 × SKV50)-706
294	(CML153 × SKV50)-592	339	(CML153 × SKV50)-707
295	(CML153 × SKV50)-593	340	(CML153 × SKV50)-708
296	(CML153 × SKV50)-596	341	(CML153 × SKV50)-709
297	(CML153 × SKV50)-597	342	(CML153 × SKV50)-710
298	(CML153 × SKV50)-598	343	(CML153 × SKV50)-712
299	(CML153 × SKV50)-600	344	(CML153 × SKV50)-717
300	(CML153 × SKV50)-601		
301	(CML153 × SKV50)-602		
302	(CML153 × SKV50)-605		
303	(CML153 × SKV50)-606		
304	(CML153 × SKV50)-609		
305	(CML153 × SKV50)-611		
306	(CML153 × SKV50)-612		
307	(CML153 × SKV50)-615		
308	(CML153 × SKV50)-616		
309	(CML153 × SKV50)-619		
310	(CML153 × SKV50)-620		
311	(CML153 × SKV50)-625		
312	(CML153 × SKV50)-634		
313	(CML153 × SKV50)-637		
314	(CML153 × SKV50)-638		
315	(CML153 × SKV50)-639		

Appendix 2: List of SNPs used for parental polymorphism survey between CML153 and SKV50

Sl No.	SNP Marker Name	Chrom osome	Distance	Sl No.	SNP Marker Name	Chrom osome	Distance
1	MZA175-25	1	10.83	65	MZA5622-21	1	155.21
2	C00270-01	1	17.65	66	MZA4300-6	1	159.73
3	MZA2244-142	1	25.14	67	MZA5727-5	1	164.25
4	MZA7935-15	1	27.07	68	MZA5597-15	1	179.27
5	MZA4997-11	1	27.93	69	MZA15268-18	1	180.04
6	MZA4997-17	1	27.93	70	MZA5480-17	1	180.73
7	MZA6238-36	1	31.96	71	MZA3627-11	1	185.38
8	MZA13094-8	1	34.93	72	MZA12706-14	1	191.11
9	C00370-01	1	35.22	73	MZA1438-34	1	192.6
10	C00365-01	1	39.47	74	C00104-01	1	195.58
11	C00304-01	1	40.01	75	MZA6043-19	1	196.63
12	C00364-01	1	41.99	76	MZA3690-10	1	197.32
13	MZA1653-31	1	52.7	77	MZA3690-23	1	197.32
14	C00124-01	1	67.46	78	C00121-01	1	198.53
15	MZA3226-15	1	68.55	79	MZA15871-11	1	199.22
16	MZA4531-46	1	68.63	80	MZA5484-22	1	199.61
17	MZA13619-5	1	68.7	81	MZA12693-8	1	202.81
18	C00240-01	1	74.4	82	C00298-01	1	202.9
19	MZA18887-12	1	76.93	83	C00248-01	1	204.32
20	MZA3951-25	1	84.39	84	MZA759-24	1	205.1
21	MZA835-25	1	85.73	85	MZA4942-12	1	205.62
22	C00260-01	1	88.02	86	MZA2478-22	1	206.46
23	MZA4597-14	1	89.91	87	C00351-01	1	207.01
24	MZA4359-21	1	91.69	88	MZA13405-8	1	212.04
25	MZA3726-129	1	94.56	89	C00100-01	1	216.98
26	C00344-01	1	94.58	90	MZA4992-10	1	217.73
27	MZA2177-85	1	94.92	91	MZA4926-16	1	217.98
28	MZA11000-21	1	95.33	92	C00251-01	1	223.74
29	MZA4313-17	1	97.78	93	C00089-01	1	225.25
30	C00185-01	1	100.27	94	MZA16605-19	1	226.47
31	C00094-01	1	102.16	95	MZA3034-3	1	234.76
32	MZA4913-18	1	102.88	96	MZA14475-3	1	235.45
33	MZA12323-17	1	105.5	97	MZA14475-7	1	235.45
34	MZA2130-29	1	107.69	98	MZA5526-25	1	246.55
35	MZA574-14	1	111.02	99	C00295-01	1	252.14
36	C00252-01	1	111.47	100	C00088-01	1	252.62
37	C00179-01	1	113.27	101	C00239-01	1	263.36
38	C00288-01	1	113.65	102	MZA595-30	1	273.41
39	MZA5098-25	1	117.39	103	C00190-01	1	273.41
40	MZA1950-71	1	117.97	104	MZA720-29	1	274.17
41	MZA5481-94	1	119.29	105	MZA3563-17	1	275.63
42	C00186-01	1	120.49	106	C00292-01	1	276.64
43	MZA5306-16	1	122.39	107	MZA18705-23	1	277.4
44	MZA4185-13	1	125.95	108	MZA2184-28	1	278.17
45	MZA4185-17	1	125.95	109	C00129-01	1	281.8
46	MZA9418-11	1	133.73	110	MZA13191-8	1	290.59
47	C00159-01	1	134.91	111	MZA13191-6	1	290.59
48	MZA12633-15	1	135.12	112	C00315-01	1	294.42
49	MZA3463-18	1	135.79	113	C00087-01	1	295.06
50	MZA1932-51	1	136.59	114	C00212-01	1	298.1
51	C00200-01	1	136.6	115	C00234-01	1	298.96
52	C00310-01	1	136.65	116	C00158-01	1	299.66
53	MZA1725-34	1	137.68	117	MZA9807-9	1	303.24
54	C00145-01	1	137.7	118	MZA1275-22	1	304.76
55	C00083-01	1	138.39	119	C00296-01	1	305.91
56	MZA4695-5	1	138.5	120	MZA673-33	1	307.76
57	MZA8074-6	1	139.61	121	C00322-01	1	308.76
58	MZA2187-46	1	139.83	122	MZA1317-17	1	313.44
59	MZA2187-34	1	139.83	123	MZA7616-35	1	316.26
60	MZA3147-18	1	151.6	124	MZA4752-14	1	318.51
61	C00333-01	1	152.45	125	C00163-01	2	7.59
62	MZA1968-22	1	152.75	126	MZA5817-15	2	9.32
63	MZA14519-8	1	153.89	127	MZA4951-8	2	10.1
64	MZA17698-8	1	154.33	128	MZA1511-26	2	16.81

Appendix 2 Cont'd……

Sl No.	SNP Marker Name	Chromo some	Distance	Sl No.	SNP Marker Name	Chromo some	Distance
129	MZA1511-14	2	16.81	195	MZA3094-23	2	247.41
130	MZA13440-11	2	17.05	196	MZA4259-5	3	7.21
131	C00339-01	2	17.05	197	C00264-01	3	9.21
132	MZA5535-8	2	25.19	198	C00259-01	3	19.6
133	MZA5628-11	2	27.48	199	MZA12859-10	3	28.32
134	C00376-01	2	30.61	200	MZA15475-27	3	33.4
135	MZA12952-13	2	31.28	201	MZA7672-7	3	37.84
136	MZA3334-6	2	33.38	202	C00278-01	3	42.31
137	MZA3309-8	2	37.29	203	MZA4204-69	3	48.89
138	C00118-01	2	37.62	204	MZA4145-18	3	66.13
139	C00151-01	2	38.47	205	MZA1960-37	3	70.21
140	MZA12979-9	2	46.62	206	MZA2343-25	3	70.31
141	MZA14783-14	2	51.22	207	MZA2766-14	3	73.36
142	MZA5822-15	2	53.53	208	MZA13823-7	3	75.3
143	MZA482-23	2	63.64	209	MZA15899-9	3	76.1
144	C00149-01	2	70.19	210	C00157-01	3	77.45
145	C00375-01	2	74.84	211	C00327-01	3	77.49
146	MZA4425-25	2	85.68	212	MZA2324-23	3	77.73
147	MZA6111-5	2	93.05	213	MZA12153-9	3	77.73
148	MZA7964-27	2	94.4	214	MZA15474-5	3	77.8
149	MZA1962-33	2	96.01	215	MZA5502-31	3	79.23
150	C00196-01	2	96.44	216	MZA7417-21	3	79.28
151	MZA4586-12	2	101.93	217	MZA15449-10	3	84.28
152	MZA4780-38	2	104.57	218	MZA1745-16	3	89.22
153	C00359-01	2	104.85	219	MZA4339-79	3	90.17
154	C00136-01	2	105.46	220	MZA4955-12	3	96.91
155	C00231-01	2	107.39	221	C00318-01	3	97.53
156	MZA10404-8	2	110.62	222	MZA9914-11	3	103.97
157	MZA10321-11	2	125.68	223	MZA13420-11	3	103.97
158	MZA3457-6	2	125.68	224	C00249-01	3	103.99
159	MZA3457-29	2	125.68	225	C00120-01	3	105.02
160	MZA4880-179	2	129.91	226	MZA2885-31	3	112.97
161	MZA13360-13	2	129.91	227	MZA1959-26	3	114.44
162	MZA883-16	2	129.91	228	MZA17210-5	3	135.23
163	MZA3626-3	2	130.96	229	C00269-01	3	151.4
164	C00356-01	2	131.15	230	C00236-01	3	159.18
165	C00255-01	2	131.56	231	MZA351-36	3	159.38
166	MZA4620-24	2	133.91	232	MZA824-17	3	159.76
167	C00102-01	2	136.28	233	MZA13673-53	3	160.3
168	MZA13648-11	2	136.54	234	C00169-01	3	163.24
169	C00357-01	2	136.96	235	C00085-01	3	167.85
170	C00115-01	2	139.75	236	MZA2919-23	3	172.71
171	C00220-01	2	140.26	237	C00287-01	3	172.71
172	MZA635-23	2	141.65	238	C00141-01	3	175.73
173	C00324-01	2	142.36	239	MZA3688-14	3	178.06
174	C00109-01	2	155.52	240	MZA9672-9	3	181.32
175	C00342-01	2	155.73	241	C00146-01	3	183.9
176	MZA4196-27	2	159.9	242	MZA8828-7	3	185.88
177	MZA499-19	2	162.16	243	MZA3352-19	3	188.77
178	MZA3055-9	2	163.57	244	MZA13742-5	3	199.96
179	MZA3668-12	2	164.36	245	MZA3342-31	3	211.14
180	C00093-01	2	165.38	246	C00233-01	3	213.4
181	MZA7953-11	2	166.63	247	MZA2672-19	3	222.29
182	MZA793-25	2	166.88	248	MZA2204-96	3	226.71
183	MZA16125-47	2	170.93	249	C00341-01	3	228
184	C00175-01	2	171.11	250	MZA3852-15	3	233.91
185	MZA14412-4	2	173.36	251	MZA3852-23	3	233.91
186	C00261-01	2	178.19	252	C00194-01	3	237.42
187	MZA2773-30	2	182.4	253	MZA2423-33	3	242.86
188	C00337-01	2	194.11	254	MZA13174-18	3	246.5
189	C00274-01	2	203.08	255	MZA617-71	3	248.13
190	C00271-01	2	211.83	256	C00099-01	4	4.25
191	C00108-01	2	217.33	257	MZA1971-20	4	8.01
192	C00384-01	2	226.23	258	MZA3712-18	4	9.68
193	C00112-01	2	229.94	259	MZA1184-26	4	15.97
194	C00214-01	2	234.07	260	MZA14235-15	4	18.8

Appendix 2 Cont'd……

Sl No.	SNP Marker Name	Chromosome	Distance	Sl No.	SNP Marker Name	Chromosome	Distance
261	MZA2438-28	4	21.72	327	C00277-01	5	5.3
262	MZA3301-28	4	36.28	328	C00116-01	5	5.91
263	MZA3963-33	4	36.57	329	MZA3061-315	5	9.2
264	C00321-01	4	42.56	330	MZA5359-10	5	21.43
265	C00291-01	4	53.68	331	MZA13122-43	5	25.5
266	MZA2518-28	4	58.89	332	MZA14671-9	5	34.47
267	MZA687-25	4	63.56	333	MZA3762-18	5	40.19
268	MZA259-11	4	63.65	334	C00297-01	5	45.4
269	MZA8527-2	4	68.86	335	MZA15223-38	5	45.71
270	MZA8283-23	4	72.57	336	MZA3137-17	5	50.05
271	C00213-01	4	72.7	337	MZA3103-47	5	53.13
272	MZA2159-8	4	84.12	338	MZA533-46	5	53.98
273	C00134-01	4	86.3	339	MZA9676-10	5	55.52
274	MZA15427-11	4	87.43	340	C00336-01	5	60.62
275	MZA3112-5	4	90.59	341	C00096-01	5	63.97
276	MZA5572-19	4	91.27	342	C00195-01	5	66.55
277	MZA13623-14	4	91.27	343	C00202-01	5	66.86
278	C00113-01	4	91.58	344	C00256-01	5	70.56
279	C00177-01	4	92.34	345	C00114-01	5	76.28
280	MZA4469-13	4	93.97	346	MZA13942-10	5	79.7
281	MZA14717-2	4	97.62	347	C00165-01	5	79.81
282	MZA14055-6	4	100.27	348	MZA565-31	5	81.21
283	MZA1307-11	4	101.19	349	MZA9009-13	5	82.53
284	MZA3587-6	4	103	350	C00312-01	5	82.68
285	C00218-01	4	103	351	MZA4647-8	5	83.61
286	C00267-01	4	104.72	352	MZA12992-5	5	86.46
287	C00170-01	4	105.09	353	MZA3691-15	5	86.77
288	C00187-01	4	113.03	354	MZA3691-18	5	86.77
289	MZA1505-31	4	113.94	355	C00106-01	5	86.87
290	C00122-01	4	116.62	356	C00095-01	5	87.07
291	MZA15864-8	4	117.96	357	MZA6795-4	5	89.17
292	C00257-01	4	125.34	358	MZA3402-11	5	89.43
293	C00126-01	4	128.7	359	C00161-01	5	91.65
294	C00123-01	4	130.12	360	MZA2769-43	5	92.46
295	MZA9635-30	4	133.64	361	C00182-01	5	92.49
296	C00272-01	4	136.79	362	MZA1870-20	5	93.16
297	MZA3155-14	4	140.61	363	MZA3171-5	5	93.62
298	C00362-01	4	146.12	364	MZA2614-14	5	93.98
299	MZA2006-57	4	147.84	365	C00379-01	5	94.22
300	C00330-01	4	154.26	366	C00092-01	5	94.33
301	MZA3637-14	4	160.09	367	C00180-01	5	95
302	MZA14618-11	4	160.09	368	MZA4165-14	5	95.05
303	MZA3637-15	4	160.09	369	MZA13675-17	5	95.05
304	C00162-01	4	169.96	370	MZA13675-18	5	95.05
305	C00284-01	4	170.4	371	MZA5798-39	5	96.73
306	C00133-01	4	171.62	372	C00279-01	5	98.12
307	C00103-01	4	173.94	373	C00289-01	5	99.75
308	MZA4348-16	4	176.75	374	MZA2348-66	5	102.12
309	C00205-01	4	177.94	375	C00358-01	5	102.75
310	C00361-01	4	179.79	376	C00307-01	5	103.69
311	MZA1684-20	4	183.57	377	C00184-01	5	105.15
312	C00273-01	4	184.18	378	C00348-01	5	113.92
313	MZA4117-14	4	184.88	379	C00367-01	5	114.03
314	MZA5519-25	4	189.03	380	C00156-01	5	117.37
315	C00335-01	4	189.08	381	MZA6910-187	5	117.64
316	MZA5137-12	4	189.26	382	C00245-01	5	122.87
317	MZA9804-28	4	197.14	383	C00227-01	5	123.94
318	MZA5599-20	4	201.25	384	C00303-01	5	126.65
319	MZA5665-26	4	207.05	385	C00265-01	5	127.33
320	MZA5665-10	4	207.05	386	MZA13696-11	5	132.87
321	MZA2100-21	4	211.05	387	MZA1899-157	5	137.13
322	MZA13084-4	4	225.12	388	C00125-01	5	137.74
323	MZA4310-112	4	231.35	389	C00201-01	5	140.37
324	C00199-01	4	237.51	390	MZA5337-18	5	141.08
325	MZA662-27	5	2.82	391	MZA5296-6	5	141.54
326	C00215-01	5	5	392	C00302-01	5	143.63

Appendix 2 Cont'd……

Sl No.	SNP Marker Name	Chrom osome	Distance	Sl No.	SNP Marker Name	Chrom osome	Distance
393	C00171-01	5	150.49	459	MZA2108-61	6	146.65
394	MZA6386-11	5	151.24	460	C00181-01	6	146.69
395	MZA7908-20	5	152.87	461	C00283-01	6	146.87
396	MZA1558-19	5	155.13	462	MZA1956-90	6	148.75
397	MZA532-23	5	155.79	463	MZA4978-27	6	151.67
398	MZA4616-7	5	164.16	464	MZA7922-8	6	156.97
399	C00174-01	5	167.1	465	MZA5361-13	6	176.81
400	MZA9161-15	5	168.28	466	MZA3466-69	6	179.75
401	C00204-01	5	172.45	467	MZA5529-4	6	179.75
402	MZA3512-186	5	176.96	468	MZA4468-13	6	179.9
403	MZA4349-6	5	187.26	469	C00197-01	7	20.96
404	MZA4349-3	5	187.26	470	MZA9241-13	7	36.65
405	MZA3612-19	5	205.1	471	MZA3078-12	7	42.95
406	C00110-01	5	215.2	472	MZA4135-15	7	45.84
407	C00091-01	5	216.27	473	MZA2691-31	7	49.96
408	MZA13639-13	5	219.15	474	MZA3676-33	7	53.58
409	MZA2865-8	5	226.8	475	C00347-01	7	56.77
410	MZA3019-38	6	5.42	476	C00371-01	7	60.51
411	MZA15961-13	6	17.88	477	C00237-01	7	64.45
412	C00107-01	6	19.66	478	C00378-01	7	67.21
413	C00221-01	6	21.29	480	MZA4285-20	7	72.85
414	MZA15873-12	6	21.68	481	C00238-01	7	73.54
415	C00206-01	6	22.62	482	MZA4353-31	7	74.73
416	C00111-01	6	22.68	483	C00355-01	7	76.13
417	C00381-01	6	23.52	484	MZA4818-15	7	76.99
418	C00372-01	6	23.59	485	MZA15501-6	7	77.21
419	C00353-01	6	23.81	486	MZA15501-9	7	77.21
420	C00360-01	6	23.94	487	MZA12830-14	7	77.42
421	C00363-01	6	24.06	488	MZA904-21	7	77.51
422	C00119-01	6	24.53	489	C00242-01	7	99.44
423	MZA1572-17	6	25.59	490	MZA5766-12	7	109.43
424	C00246-01	6	25.62	491	C00189-01	7	111.93
425	MZA8327-18	6	26.19	492	MZA9162-135	7	117.42
426	C00178-01	6	28.49	493	C00138-01	7	118.21
427	MZA5347-6	6	31.63	494	MZA16437-20	7	120.57
428	MZA12904-7	6	32.32	495	C00230-01	7	120.63
429	MZA8909-12	6	34.19	496	MZA6608-5	7	121.47
430	MZA15035-9	6	35.32	497	C00343-01	7	127.17
431	C00105-01	6	44.82	498	MZA112-8	7	133.59
432	C00338-01	6	67.9	499	MZA1912-20	7	138.94
433	C00382-01	6	71.66	500	MZA1912-23	7	138.94
434	MZA13451-15	6	72.41	501	MZA424-13	7	144.48
435	C00153-01	6	72.57	502	MZA424-16	7	144.48
436	MZA1190-3	6	73.05	503	MZA3435-6	7	161.79
437	MZA13020-10	6	75.31	504	MZA7898-10	7	171.51
438	C00285-01	6	76.34	505	MZA10225-15	7	172.88
439	MZA4904-16	6	76.71	506	C00313-01	7	176.82
440	MZA12794-47	6	78.32	507	C00262-01	7	182.29
441	C00191-01	6	85.6	508	C00263-01	7	184.57
442	C00117-01	6	88.11	509	C00329-01	7	191.43
443	C00097-01	6	90.41	510	C00306-01	7	197.18
444	MZA3590-19	6	90.64	511	C00176-01	7	204.9
445	C00308-01	6	92.87	512	MZA2871-188	7	205.17
446	C00172-01	6	95	513	MZA5232-11	7	205.29
447	MZA11985-27	6	97.24	514	MZA5218-14	8	19.27
448	C00144-01	6	115.1	515	C00132-01	8	29.9
449	MZA15251-3	6	119.3	516	C00350-01	8	36.75
450	MZA15251-5	6	119.3	517	MZA4512-38	8	36.86
451	C00243-01	6	125	518	MZA9126-15	8	42.76
452	MZA4662-153	6	125.12	519	C00216-01	8	42.8
453	C00128-01	6	130.38	520	MZA2487-6	8	45.78
454	MZA5794-13	6	131.63	521	MZA5637-15	8	51.03
455	MZA597-12	6	133.27	522	MZA9695-8	8	55.06
456	MZA4748-16	6	136.44	523	C00250-01	8	55.76
457	MZA16607-11	6	142.68	524	MZA1447-89	8	60.46
458	MZA4503-25	6	146.13	525	C00228-01	8	62.4

Appendix 2 Cont'd……

Sl No.	SNP Marker Name	Chrom osome	Distance	Sl No.	SNP Marker Name	Chrom osome	Distance
526	MZA5158-13	8	67.16	592	MZA1218-6	9	40.61
527	MZA1978-111	8	73.87	593	C00326-01	9	41.62
528	MZA6428-11	8	75.53	594	C00208-01	9	42.02
529	MZA2350-14	8	80.39	595	C00082-01	9	46.23
530	MZA2350-17	8	80.39	596	MZA5181-10	9	51.55
531	MZA3856-10	8	84.38	597	MZA4720-12	9	56.34
532	MZA191-12	8	85.4	598	MZA7584-7	9	60.18
533	MZA191-18	8	85.4	599	MZA5185-13	9	61.63
534	MZA4968-10	8	85.63	600	MZA4495-14	9	73.45
535	C00142-01	8	87.12	601	MZA229-15	9	73.97
536	C00135-01	8	88.7	602	MZA1871-19	9	75.09
537	C00369-01	8	88.93	603	MZA2101-21	9	75.68
538	C00148-01	8	89.05	604	C00084-01	9	75.79
539	MZA4552-6	8	89.54	605	MZA3893-18	9	75.99
540	MZA5395-34	8	89.94	606	C00090-01	9	76.23
541	MZA11114-10	8	90.47	607	C00226-01	9	85.75
542	MZA11114-7	8	90.47	608	MZA13183-12	9	85.79
543	C00168-01	8	91.03	609	MZA2278-86	9	87.58
544	MZA5235-8	8	91.47	610	C00293-01	9	93.08
545	MZA1534-45	8	93.08	611	C00280-01	9	98.09
546	C00098-01	8	97.6	612	C00155-01	9	99.05
547	MZA4134-8	8	107.66	613	C00349-01	9	100
548	C00275-01	8	112.15	614	MZA4905-6	9	100.98
549	MZA934-19	8	112.15	615	C00266-01	9	101.23
550	MZA3993-16	8	113.21	616	MZA7916-4	9	108.02
551	MZA3993-28	8	113.21	617	MZA1599-84	9	108.75
552	MZA5805-19	8	114.07	618	MZA13582-6	9	111.02
553	MZA10525-11	8	117.62	619	MZA4689-49	9	113.14
554	MZA10525-9	8	117.62	620	MZA794-26	9	117.22
555	MZA5468-25	8	119.07	621	MZA1766-1	9	119.06
556	MZA14152-18	8	120.09	622	MZA816-25	9	138.18
557	C00281-01	8	121.5	623	MZA816-29	9	138.18
558	MZA4203-11	8	121.62	624	MZA11226-13	9	139.59
559	C00183-01	8	121.74	625	C00188-01	9	140.59
560	MZA448-23	8	121.75	626	MZA4604-18	9	141.08
561	MZA15744-10	8	122.25	627	C00225-01	9	141.15
562	MZA523-21	8	122.32	628	C00316-01	9	141.22
563	C00140-01	8	127.1	629	MZA4303-16	9	152.25
564	C00173-01	8	128.65	630	MZA15445-25	9	158.17
565	MZA4757-14	8	130.23	631	MZA13681-12	9	166.82
566	MZA12749-13	8	130.3	632	MZA1911-173	9	168.73
567	MZA4677-11	8	131.93	633	C00276-01	9	174.61
568	MZA15278-6	8	132.63	634	MZA3631-47	10	30.74
569	C00193-01	8	132.95	635	MZA2828-83	10	45.19
570	C00241-01	8	133.53	636	MZA3765-7	10	47.43
571	C00223-01	8	134.25	637	C00258-01	10	48.6
572	MZA3465-6	8	137.15	638	MZA5740-9	10	48.84
573	MZA232-30	8	144.37	639	MZA1752-36	10	51.12
574	MZA1834-47	8	145.37	640	MZA15331-16	10	53.56
575	MZA4560-54	8	148.65	641	C00311-01	10	54.87
576	C00299-01	8	148.99	642	MZA3896-9	10	56.98
577	MZA4786-9	8	157.98	643	C00247-01	10	57.9
578	C00268-01	8	163.29	644	MZA4973-6	10	58.12
579	MZA13493-12	8	163.56	645	MZA3911-11	10	58.56
580	MZA765-24	8	168.12	646	MZA3922-32	10	58.61
581	MZA14046-9	8	181.57	647	C00211-01	10	59.22
582	MZA14104-23	8	193.83	648	MZA8352-4	10	59.28
583	MZA3312-23	8	194.24	649	C00282-01	10	60.81
584	MZA3337-23	8	194.78	650	MZA6822-4	10	60.88
585	MZA2749-10	8	199.04	651	MZA4066-11	10	61.15
586	MZA5019-59	8	199.45	652	MZA3931-17	10	61.16
587	C00130-01	8	202.23	653	MZA1812-32	10	61.18
588	C00160-01	9	12.11	654	C00229-01	10	61.19
589	MZA3925-79	9	12.22	655	MZA1155-14	10	61.7
590	C00368-01	9	21.43	656	C00314-01	10	62.03
591	MZA11946-17	9	34.53	657	C00331-01	10	63.01

Appendix 2 Cont'd……

Sl No.	SNP Marker Name	Chrom osome	Distance	Sl No.	SNP Marker Name	Chrom osome	Distance
658	MZA2770-19	10	63.71	724	C03360-2	0	0
659	C00154-01	10	66.67	725	C03384-2	0	0
660	C00152-01	10	67.14	726	C03359-2	0	0
661	MZA12990-15	10	67.34	727	C03342-2	0	0
662	MZA537-22	10	70.46	728	C03358-2	0	0
663	C00332-01	10	70.76	729	C03373-2	0	0
664	C00086-01	10	71.34	730	C03348-2	0	0
665	MZA12625-18	10	71.34	731	C03372-2	0	0
666	MZA18195-6	10	71.34	732	C03382-2	0	0
667	C00137-01	10	72.36	733	C03383-2	0	0
668	MZA13687-14	10	75.78	734	C03346-2	0	0
669	MZA1576-25	10	78.95	735	C03363-2	0	0
670	C00334-01	10	85.03	736	C03404-2	0	0
671	MZA18513-156	10	99.69	737	C03371-2	0	0
672	MZA15868-56	10	101.24	738	C03401-2	0	0
673	C00150-01	10	116.02	739	C03392-2	0	0
674	C00317-01	10	123.31	740	C03391-2	0	0
675	C00309-01	10	125.22	741	C03368-2	0	0
676	C00346-01	10	126.67	742	C03376-2	0	0
677	C00305-01	10	128.4	743	C03349-2	0	0
678	MZA3941-11	10	129.06	744	C03347-2	0	0
679	MZA5435-25	10	129.12	745	C03381-2	0	0
680	C00198-01	10	131.53	746	C03402-2	0	0
681	MZA3844-14	10	147.93	747	C03405-2	0	0
682	MZA3736-11	10	154.43	748	C03389-2	0	0
683	MZA10750-26	10	160.4	749	C03390-2	0	0
684	MZA1506-18	10	161.54	750	C03375-2	0	0
685	MZA1506-23	10	161.54	751	C03399-2	0	0
686	MZA16788-6	0	0	752	C03367-2	0	0
687	MZA563-9	0	0	753	C03352-2	0	0
688	C00209-01	0	0	754	C03379-2	0	0
689	C00222-01	0	0	755	C03341-2	0	0
690	C00300-01	0	0	756	C03369-2	0	0
691	C00217-01	0	0	757	C03397-2	0	0
692	C00253-01	0	0	758	C03396-2	0	0
693	C00254-01	0	0	759	C03385-2	0	0
694	C00319-01	0	0	760	C03377-2	0	0
695	C00380-01	0	0	761	C03357-2	0	0
696	C00286-01	0	0	762	C03366-2	0	0
697	C00345-01	0	0	763	C03388-2	0	0
698	C00207-01	0	0	764	C03374-2	0	0
699	C00127-01	0	0	765	C03395-2	0	0
700	C00328-01	0	0				
701	C00166-01	0	0				
702	C00101-01	0	0				
703	C00210-01	0	0				
704	C00219-01	0	0				
705	C00290-01	0	0				
706	C00352-01	0	0				
707	C00354-01	0	0				
708	C00383-01	0	0				
709	C03355-2	0	0				
710	C03364-2	0	0				
711	C03353-2	0	0				
712	C03393-2	0	0				
713	C03387-2	0	0				
714	C03380-2	0	0				
715	C03370-2	0	0				
716	C03394-2	0	0				
717	C03378-2	0	0				
718	C03386-2	0	0				
719	C03400-2	0	0				
720	C03362-2	0	0				
721	C03361-2	0	0				
722	C03403-2	0	0				
723	C03365-2	0	0				

Appendix 3: Arcsine transformed phenotypic data of $F_{2:3}$ progenies in *kharif-2012* and *kharif*-2013

Sl. No.	Pedigree	NCLB Severity (%)		SCR Severity (%)	SDM incidence (%)
		Kharif 2012	*Kharif 2013*	*Kharif 2013*	*Kharif 2013*
1	(CML153 × SKV50)-1	37.75	24.34	25.91	49.68
2	(CML153 × SKV50)-3	54.71	41.54	29.24	73.02
3	(CML153 × SKV50)-6	52.43	45.55	45.22	69.95
4	(CML153 × SKV50)-7	45.70	45.55	49.03	54.61
5	(CML153 × SKV50)-8	44.03	46.13	31.41	58.32
6	(CML153 × SKV50)-9	37.25	40.38	42.42	54.98
7	(CML153 × SKV50)-10	35.50	43.84	38.16	30.13
8	(CML153 × SKV50)-12	47.85	42.69	29.35	48.72
9	(CML153 × SKV50)-16	49.09	42.69	9.21	45.55
10	(CML153 × SKV50)-17	50.75	45.55	24.72	49.68
11	(CML153 × SKV50)-18	58.58	44.98	33.73	73.02
12	(CML153 × SKV50)-19	54.46	58.03	39.21	73.02
13	(CML153 × SKV50)-21	49.78	50.16	29.19	65.75
14	(CML153 × SKV50)-22	46.89	45.55	35.01	48.84
15	(CML153 × SKV50)-23	48.81	50.16	27.04	43.29
16	(CML153 × SKV50)-28	40.27	38.63	48.72	73.02
17	(CML153 × SKV50)-29	47.85	36.86	36.93	64.50
18	(CML153 × SKV50)-30	46.62	43.84	25.74	9.30
19	(CML153 × SKV50)-31	41.54	46.70	17.26	65.75
20	(CML153 × SKV50)-34	55.88	46.13	30.63	47.28
21	(CML153 × SKV50)-35	48.57	35.65	36.78	49.68
22	(CML153 × SKV50)-36	58.58	53.11	20.15	73.02
23	(CML153 × SKV50)-39	44.03	50.75	32.12	65.88
24	(CML153 × SKV50)-41	62.00	47.85	30.76	35.15
25	(CML153 × SKV50)-45	38.38	37.45	26.60	61.91
26	(CML153 × SKV50)-46	50.75	47.28	45.94	57.05
27	(CML153 × SKV50)-47	36.26	43.26	40.72	40.09
28	(CML153 × SKV50)-48	53.37	48.43	37.68	65.47
29	(CML153 × SKV50)-49	52.43	31.29	37.64	63.69
30	(CML153 × SKV50)-52	49.45	48.43	27.43	45.17
31	(CML153 × SKV50)-55	53.11	40.96	8.21	31.43
32	(CML153 × SKV50)-58	59.16	47.85	24.59	14.55
33	(CML153 × SKV50)-60	46.41	36.86	29.43	59.22
34	(CML153 × SKV50)-61	41.70	33.20	32.75	18.94
35	(CML153 × SKV50)-62	57.55	47.28	25.25	73.02
36	(CML153 × SKV50)-63	39.22	33.82	6.51	52.47
37	(CML153 × SKV50)-64	55.73	46.70	36.70	45.84
38	(CML153 × SKV50)-65	47.85	33.20	44.26	60.09
39	(CML153 × SKV50)-69	50.02	44.41	33.20	73.02
40	(CML153 × SKV50)-70	46.62	41.54	45.95	62.12
41	(CML153 × SKV50)-75	54.71	40.96	57.83	62.32
42	(CML153 × SKV50)-79	50.75	40.38	45.75	73.02
43	(CML153 × SKV50)-80	43.84	41.54	49.78	55.34
44	(CML153 × SKV50)-81	27.44	38.04	34.60	34.13
45	(CML153 × SKV50)-82	43.55	36.86	43.55	24.30

Appendix 3 Cont'd……

Sl. No.	Pedigree	NCLB Severity (%)		SCR Severity (%)	SDM incidence (%)
		Kharif 2012	*Kharif 2013*	*Kharif 2013*	*Kharif 2013*
46	(CML153 × SKV50)-85	44.03	44.41	39.45	73.02
47	(CML153 × SKV50)-86	44.98	42.69	31.04	58.88
48	(CML153 × SKV50)-91	49.09	38.63	22.49	73.02
49	(CML153 × SKV50)-92	39.94	44.41	18.43	29.88
50	(CML153 × SKV50)-93	51.48	36.86	25.06	40.92
51	(CML153 × SKV50)-94	43.55	45.55	25.32	44.89
52	(CML153 × SKV50)-96	39.22	32.57	31.65	48.14
53	(CML153 × SKV50)-99	37.25	33.20	31.83	73.02
54	(CML153 × SKV50)-101	53.71	43.26	37.52	73.02
55	(CML153 × SKV50)-103	44.98	36.26	31.04	34.13
56	(CML153 × SKV50)-104	49.29	40.96	37.74	34.69
57	(CML153 × SKV50)-105	59.98	39.22	31.04	73.02
58	(CML153 × SKV50)-107	53.71	46.70	42.10	73.02
59	(CML153 × SKV50)-108	41.39	40.40	20.16	64.25
60	(CML153 × SKV50)-113	56.77	35.65	34.60	50.70
61	(CML153 × SKV50)-114	39.22	40.38	44.98	33.44
62	(CML153 × SKV50)-115	44.03	44.41	39.70	60.98
63	(CML153 × SKV50)-116	62.40	39.80	28.36	29.10
64	(CML153 × SKV50)-117	50.75	45.55	25.32	73.02
65	(CML153 × SKV50)-119	63.41	44.98	30.90	73.02
66	(CML153 × SKV50)-120	66.62	40.38	36.72	66.40
67	(CML153 × SKV50)-124	51.73	39.22	35.42	37.42
68	(CML153 × SKV50)-126	56.77	36.86	30.46	22.44
69	(CML153 × SKV50)-127	61.66	51.33	24.67	73.02
70	(CML153 × SKV50)-129	55.88	44.41	33.20	34.13
71	(CML153 × SKV50)-132	55.22	43.84	33.20	49.68
72	(CML153 × SKV50)-133	50.75	33.82	26.55	59.87
73	(CML153 × SKV50)-134	52.22	35.05	17.81	43.48
74	(CML153 × SKV50)-136	55.73	43.26	25.32	73.02
75	(CML153 × SKV50)-139	56.77	44.98	38.23	47.93
76	(CML153 × SKV50)-141	51.73	48.43	30.38	16.94
77	(CML153 × SKV50)-142	46.62	39.80	36.21	23.96
78	(CML153 × SKV50)-143	54.31	37.45	40.92	19.04
79	(CML153 × SKV50)-145	41.39	34.44	36.98	34.13
80	(CML153 × SKV50)-146	52.71	54.31	39.22	60.44
81	(CML153 × SKV50)-149	53.71	49.58	33.20	60.63
82	(CML153 × SKV50)-150	62.23	36.26	27.99	48.73
83	(CML153 × SKV50)-153	46.26	49.58	4.37	46.14
84	(CML153 × SKV50)-154	39.22	40.96	35.15	45.40
85	(CML153 × SKV50)-157	40.38	40.38	16.69	44.25
86	(CML153 × SKV50)-158	58.58	36.86	31.04	54.16
87	(CML153 × SKV50)-161	62.23	41.54	37.95	47.87
88	(CML153 × SKV50)-162	54.71	40.38	42.10	60.30
89	(CML153 × SKV50)-169	59.98	39.22	31.26	52.27
90	(CML153 × SKV50)-170	55.88	36.26	32.14	49.17

Appendix 3 Cont'd……

Sl. No.	Pedigree	NCLB Severity (%)		SCR Severity (%)	SDM incidence (%)
		Kharif 2012	*Kharif 2013*	*Kharif 2013*	*Kharif 2013*
91	(CML153 × SKV50)-171	45.94	40.96	25.32	44.98
92	(CML153 × SKV50)-173	61.41	39.22	37.74	52.50
93	(CML153 × SKV50)-174	54.14	32.57	30.87	49.36
94	(CML153 × SKV50)-178	54.04	44.98	31.36	48.87
95	(CML153 × SKV50)-180	58.58	39.80	22.49	48.65
96	(CML153 × SKV50)-182	41.15	38.04	27.99	41.76
97	(CML153 × SKV50)-185	49.45	35.65	23.26	43.97
98	(CML153 × SKV50)-187	64.45	47.85	6.55	49.83
99	(CML153 × SKV50)-188	57.82	44.98	8.01	55.25
100	(CML153 × SKV50)-189	55.73	46.70	30.27	52.47
101	(CML153 × SKV50)-191	54.46	43.26	37.23	58.57
102	(CML153 × SKV50)-192	54.14	44.41	31.26	50.74
103	(CML153 × SKV50)-194	59.32	45.55	31.83	49.38
104	(CML153 × SKV50)-195	55.53	47.85	17.81	57.24
105	(CML153 × SKV50)-197	59.51	51.92	32.29	48.42
106	(CML153 × SKV50)-198	45.94	32.57	32.14	46.12
107	(CML153 × SKV50)-200	47.85	31.29	4.37	44.50
108	(CML153 × SKV50)-201	45.94	34.44	22.49	48.67
109	(CML153 × SKV50)-203	48.57	30.64	32.41	48.17
110	(CML153 × SKV50)-204	65.88	43.26	43.84	46.04
111	(CML153 × SKV50)-205	37.25	35.05	42.10	35.48
112	(CML153 × SKV50)-206	42.52	31.94	36.83	39.53
113	(CML153 × SKV50)-207	30.46	35.05	36.83	60.54
114	(CML153 × SKV50)-208	39.22	36.26	26.55	51.39
115	(CML153 × SKV50)-209	38.24	59.32	4.37	49.39
116	(CML153 × SKV50)-212	49.78	50.16	26.55	57.17
117	(CML153 × SKV50)-214	44.98	46.13	13.74	46.72
118	(CML153 × SKV50)-217	48.17	42.11	15.81	49.79
119	(CML153 × SKV50)-218	49.92	42.69	27.30	45.92
120	(CML153 × SKV50)-222	44.98	37.45	13.28	48.74
121	(CML153 × SKV50)-224	39.86	35.05	22.49	49.17
122	(CML153 × SKV50)-225	39.22	33.82	42.10	38.67
123	(CML153 × SKV50)-228	43.71	43.84	23.98	43.47
124	(CML153 × SKV50)-230	50.75	42.69	22.49	46.59
125	(CML153 × SKV50)-235	42.52	26.55	32.89	39.23
126	(CML153 × SKV50)-237	31.94	29.99	42.10	52.41
127	(CML153 × SKV50)-238	50.75	34.44	23.98	53.30
128	(CML153 × SKV50)-239	43.07	36.26	15.32	38.96
129	(CML153 × SKV50)-240	48.43	34.44	26.58	61.35
130	(CML153 × SKV50)-241	44.27	47.28	23.41	44.68
131	(CML153 × SKV50)-242	43.61	39.22	16.96	46.05
132	(CML153 × SKV50)-244	39.22	29.99	17.65	47.20
133	(CML153 × SKV50)-245	32.41	33.82	30.58	50.14
134	(CML153 × SKV50)-246	24.45	38.04	25.81	49.46
135	(CML153 × SKV50)-248	38.24	36.26	34.04	46.63

Appendix 3 Cont'd……

Sl. No.	Pedigree	NCLB Severity (%)		SCR Severity (%)	SDM incidence (%)
		Kharif 2012	*Kharif 2013*	*Kharif 2013*	*Kharif 2013*
136	(CML153 × SKV50)-250	30.46	37.45	23.98	49.14
137	(CML153 × SKV50)-251	37.53	44.98	30.32	47.80
138	(CML153 × SKV50)-252	48.26	46.13	36.21	40.03
139	(CML153 × SKV50)-253	40.51	46.70	36.36	50.74
140	(CML153 × SKV50)-255	51.48	38.63	36.21	45.25
141	(CML153 × SKV50)-256	38.38	30.64	31.70	45.08
142	(CML153 × SKV50)-257	44.03	27.96	17.65	47.51
143	(CML153 × SKV50)-258	36.68	39.22	6.91	45.95
144	(CML153 × SKV50)-259	45.80	47.28	17.65	51.57
145	(CML153 × SKV50)-260	47.53	40.38	6.55	42.83
146	(CML153 × SKV50)-262	55.73	39.22	15.77	47.77
147	(CML153 × SKV50)-265	44.98	43.84	26.55	46.51
148	(CML153 × SKV50)-266	39.94	39.80	25.81	41.57
149	(CML153 × SKV50)-268	41.15	46.13	18.43	32.44
150	(CML153 × SKV50)-269	39.22	49.58	26.55	45.23
151	(CML153 × SKV50)-270	48.43	34.44	30.46	46.98
152	(CML153 × SKV50)-271	40.96	40.96	29.74	47.01
153	(CML153 × SKV50)-272	43.55	49.58	22.49	53.90
154	(CML153 × SKV50)-274	41.15	55.53	17.15	51.57
155	(CML153 × SKV50)-276	43.55	37.45	23.26	51.52
156	(CML153 × SKV50)-279	42.11	40.96	20.76	48.17
157	(CML153 × SKV50)-283	34.08	42.69	29.88	46.79
158	(CML153 × SKV50)-286	41.54	41.54	28.48	46.10
159	(CML153 × SKV50)-287	41.54	40.38	17.65	45.78
160	(CML153 × SKV50)-289	47.28	39.80	9.21	44.46
161	(CML153 × SKV50)-290	36.68	53.71	31.70	63.26
162	(CML153 × SKV50)-292	39.22	44.98	31.70	53.17
163	(CML153 × SKV50)-294	46.89	51.92	25.64	43.28
164	(CML153 × SKV50)-295	49.09	50.75	27.58	46.72
165	(CML153 × SKV50)-296	46.41	45.55	25.32	44.50
166	(CML153 × SKV50)-298	36.26	39.22	15.67	50.21
167	(CML153 × SKV50)-301	40.67	42.11	7.86	53.73
168	(CML153 × SKV50)-302	48.57	40.38	27.30	52.06
169	(CML153 × SKV50)-303	53.11	43.26	27.99	56.52
170	(CML153 × SKV50)-309	55.00	37.45	28.51	49.87
171	(CML153 × SKV50)-310	44.98	37.45	32.28	48.03
172	(CML153 × SKV50)-311	39.22	38.04	19.83	40.00
173	(CML153 × SKV50)-314	45.80	29.99	26.84	53.67
174	(CML153 × SKV50)-315	44.98	33.82	34.04	49.46
175	(CML153 × SKV50)-317	48.57	38.63	32.14	48.42
176	(CML153 × SKV50)-319	47.85	37.45	29.15	32.44
177	(CML153 × SKV50)-320	58.58	29.32	25.82	47.27
178	(CML153 × SKV50)-321	39.22	47.85	31.59	50.74
179	(CML153 × SKV50)-322	49.92	39.80	20.97	47.75
180	(CML153 × SKV50)-324	55.99	46.13	23.78	42.52

Appendix 3 Cont'd……

Sl. No.	Pedigree	NCLB Severity (%)		SCR Severity (%)	SDM incidence (%)
		Kharif 2012	*Kharif 2013*	*Kharif 2013*	*Kharif 2013*
181	(CML153 × SKV50)-327	47.85	47.28	34.24	41.63
182	(CML153 × SKV50)-329	53.71	35.65	26.86	41.19
183	(CML153 × SKV50)-330	41.15	38.04	7.48	53.07
184	(CML153 × SKV50)-331	40.88	38.04	27.14	44.85
185	(CML153 × SKV50)-332	35.65	32.57	39.66	53.35
186	(CML153 × SKV50)-335	48.43	36.26	42.59	49.17
187	(CML153 × SKV50)-338	50.02	37.45	42.82	57.73
188	(CML153 × SKV50)-342	63.41	39.22	36.33	27.57
189	(CML153 × SKV50)-343	44.98	50.75	30.50	35.06
190	(CML153 × SKV50)-345	49.29	43.84	32.15	19.26
191	(CML153 × SKV50)-346	59.98	48.43	33.20	47.48
192	(CML153 × SKV50)-347	46.41	35.05	23.98	52.16
193	(CML153 × SKV50)-348	50.75	30.64	25.81	48.30
194	(CML153 × SKV50)-351	42.11	51.92	12.67	36.83
195	(CML153 × SKV50)-352	39.22	36.86	26.58	45.38
196	(CML153 × SKV50)-353	49.09	36.86	23.98	34.62
197	(CML153 × SKV50)-354	40.67	33.20	17.65	45.56
198	(CML153 × SKV50)-355	48.26	40.38	13.28	33.07
199	(CML153 × SKV50)-356	44.03	36.26	33.20	34.78
200	(CML153 × SKV50)-357	49.09	35.65	36.21	37.16
201	(CML153 × SKV50)-362	31.39	33.20	33.47	37.44
202	(CML153 × SKV50)-363	57.55	32.57	30.58	39.18
203	(CML153 × SKV50)-364	44.98	38.04	37.66	33.28
204	(CML153 × SKV50)-365	40.19	34.44	34.28	32.23
205	(CML153 × SKV50)-367	53.11	36.26	26.55	49.16
206	(CML153 × SKV50)-371	46.41	36.26	41.66	44.85
207	(CML153 × SKV50)-372	40.19	39.22	48.30	45.22
208	(CML153 × SKV50)-375	40.88	41.54	46.46	34.45
209	(CML153 × SKV50)-380	50.75	40.38	32.76	48.80
210	(CML153 × SKV50)-381	54.71	37.45	16.60	39.57
211	(CML153 × SKV50)-382	39.22	33.82	32.89	39.03
212	(CML153 × SKV50)-383	47.85	35.65	17.65	30.21
213	(CML153 × SKV50)-384	48.81	35.65	19.61	34.80
214	(CML153 × SKV50)-386	53.71	26.55	33.88	40.83
215	(CML153 × SKV50)-389	31.62	39.80	39.09	34.85
216	(CML153 × SKV50)-390	34.08	39.22	35.72	31.75
217	(CML153 × SKV50)-391	43.34	43.26	22.78	50.92
218	(CML153 × SKV50)-393	46.41	39.22	41.12	41.55
219	(CML153 × SKV50)-396	39.80	38.63	13.59	44.10
220	(CML153 × SKV50)-400	40.88	34.44	35.42	42.37
221	(CML153 × SKV50)-402	50.75	40.38	23.07	35.08
222	(CML153 × SKV50)-403	54.31	36.86	25.64	40.13
223	(CML153 × SKV50)-404	46.13	40.96	33.76	40.45
224	(CML153 × SKV50)-405	44.16	37.45	17.65	43.65
225	(CML153 × SKV50)-406	51.59	31.94	27.58	53.22

Appendix 3 Cont'd……

Sl. No.	Pedigree	NCLB Severity (%)		SCR Severity (%)	SDM incidence (%)
		Kharif 2012	*Kharif 2013*	*Kharif 2013*	*Kharif 2013*
226	(CML153 × SKV50)-408	39.22	44.41	17.65	35.33
227	(CML153 × SKV50)-413	54.71	39.80	21.29	36.22
228	(CML153 × SKV50)-417	49.58	35.65	24.35	44.42
229	(CML153 × SKV50)-418	44.98	29.99	7.48	34.85
230	(CML153 × SKV50)-419	50.75	37.45	23.26	43.02
231	(CML153 × SKV50)-423	50.75	31.94	21.49	36.41
232	(CML153 × SKV50)-424	50.10	29.99	19.74	54.54
233	(CML153 × SKV50)-426	49.09	31.29	23.15	53.76
234	(CML153 × SKV50)-427	43.07	24.34	9.21	42.26
235	(CML153 × SKV50)-431	47.85	40.96	25.06	41.79
236	(CML153 × SKV50)-433	47.28	34.44	27.26	51.01
237	(CML153 × SKV50)-442	43.34	34.44	23.15	34.74
238	(CML153 × SKV50)-451	49.09	36.26	23.15	39.32
239	(CML153 × SKV50)-452	41.15	36.26	4.37	40.57
240	(CML153 × SKV50)-453	51.73	42.69	25.95	30.18
241	(CML153 × SKV50)-468	55.53	44.41	20.97	39.32
242	(CML153 × SKV50)-469	44.16	43.84	10.13	38.60
243	(CML153 × SKV50)-473	58.03	41.54	26.55	37.71
244	(CML153 × SKV50)-474	34.23	44.98	34.22	40.35
245	(CML153 × SKV50)-476	56.77	32.57	30.88	47.37
246	(CML153 × SKV50)-477	62.00	47.28	24.67	38.30
247	(CML153 × SKV50)-485	49.09	44.41	35.22	40.03
248	(CML153 × SKV50)-486	58.89	33.82	39.22	44.56
249	(CML153 × SKV50)-487	44.03	36.26	37.48	44.24
250	(CML153 × SKV50)-491	47.85	35.65	37.66	41.80
251	(CML153 × SKV50)-494	56.77	45.55	36.36	43.48
252	(CML153 × SKV50)-498	41.39	46.70	31.04	37.60
253	(CML153 × SKV50)-499	53.28	39.22	29.15	47.93
254	(CML153 × SKV50)-500	42.69	42.11	29.88	47.04
255	(CML153 × SKV50)-501	38.38	42.69	33.76	40.61
256	(CML153 × SKV50)-505	43.55	31.29	33.20	46.66
257	(CML153 × SKV50)-511	37.25	30.64	36.21	41.38
258	(CML153 × SKV50)-515	41.15	36.26	36.21	45.29
259	(CML153 × SKV50)-516	43.55	32.57	42.10	31.01
260	(CML153 × SKV50)-517	49.78	36.26	43.53	39.88
261	(CML153 × SKV50)-520	49.29	40.38	43.38	45.97
262	(CML153 × SKV50)-524	42.11	40.38	34.22	39.10
263	(CML153 × SKV50)-525	36.86	42.11	36.21	41.82
264	(CML153 × SKV50)-526	30.81	38.04	37.74	44.76
265	(CML153 × SKV50)-529	51.92	31.29	39.22	38.97
266	(CML153 × SKV50)-531	37.25	37.45	36.93	35.77
267	(CML153 × SKV50)-533	41.75	43.84	33.20	48.47
268	(CML153 × SKV50)-538	44.27	39.22	32.89	31.75
269	(CML153 × SKV50)-540	34.74	36.26	33.20	35.64
270	(CML153 × SKV50)-541	46.41	47.28	38.23	47.88

Appendix 3 Cont'd……

Sl. No.	Pedigree	NCLB Severity (%)		SCR Severity (%)	SDM incidence (%)
		Kharif 2012	*Kharif 2013*	*Kharif 2013*	*Kharif 2013*
271	(CML153 × SKV50)-542	34.57	39.80	35.15	38.82
272	(CML153 × SKV50)-543	44.16	35.05	39.09	36.41
273	(CML153 × SKV50)-546	37.75	46.70	42.75	42.14
274	(CML153 × SKV50)-548	38.24	39.80	37.23	57.67
275	(CML153 × SKV50)-551	40.51	43.84	37.48	44.51
276	(CML153 × SKV50)-554	50.75	47.28	32.41	46.97
277	(CML153 × SKV50)-555	40.88	46.13	40.52	47.72
278	(CML153 × SKV50)-559	44.98	32.57	42.82	43.28
279	(CML153 × SKV50)-561	46.62	38.04	27.30	73.02
280	(CML153 × SKV50)-562	43.07	40.38	19.74	73.02
281	(CML153 × SKV50)-563	50.75	43.26	29.88	55.59
282	(CML153 × SKV50)-565	42.11	29.99	33.76	54.61
283	(CML153 × SKV50)-568	36.86	31.29	31.04	55.22
284	(CML153 × SKV50)-573	44.98	27.26	24.98	56.53
285	(CML153 × SKV50)-577	33.20	29.99	28.82	69.95
286	(CML153 × SKV50)-578	47.85	37.45	33.20	44.41
287	(CML153 × SKV50)-579	35.25	33.20	33.76	39.72
288	(CML153 × SKV50)-581	50.75	38.04	24.08	73.02
289	(CML153 × SKV50)-585	44.98	30.64	24.67	57.71
290	(CML153 × SKV50)-587	47.85	39.22	32.14	47.28
291	(CML153 × SKV50)-588	35.50	38.63	36.21	65.18
292	(CML153 × SKV50)-589	52.71	28.65	42.10	40.09
293	(CML153 × SKV50)-591	37.53	36.26	39.37	57.71
294	(CML153 × SKV50)-592	44.98	23.57	28.27	48.82
295	(CML153 × SKV50)-593	38.24	27.26	36.98	42.71
296	(CML153 × SKV50)-596	48.43	40.96	34.60	51.63
297	(CML153 × SKV50)-597	44.16	39.80	29.88	55.22
298	(CML153 × SKV50)-598	48.81	40.96	26.55	62.42
299	(CML153 × SKV50)-600	37.75	33.20	28.27	41.35
300	(CML153 × SKV50)-601	33.20	29.99	41.00	34.85
301	(CML153 × SKV50)-602	41.70	26.55	46.01	73.02
302	(CML153 × SKV50)-605	40.88	40.96	36.98	73.02
303	(CML153 × SKV50)-606	28.87	39.22	28.82	67.54
304	(CML153 × SKV50)-609	38.38	37.45	29.15	69.64
305	(CML153 × SKV50)-611	46.13	34.44	31.41	65.18
306	(CML153 × SKV50)-612	47.28	31.94	35.15	36.41
307	(CML153 × SKV50)-615	33.20	30.64	40.18	57.71
308	(CML153 × SKV50)-616	38.38	31.94	39.81	73.02
309	(CML153 × SKV50)-619	50.75	38.63	33.20	49.08
310	(CML153 × SKV50)-620	48.81	39.80	36.21	73.02
311	(CML153 × SKV50)-625	56.77	40.96	41.14	64.74
312	(CML153 × SKV50)-634	44.98	42.11	43.55	70.04
313	(CML153 × SKV50)-637	47.85	38.63	41.18	58.32
314	(CML153 × SKV50)-638	39.22	39.22	37.17	33.05
315	(CML153 × SKV50)-639	36.86	33.82	31.83	52.81

Appendix 3 Cont'd……

Sl. No.	Pedigree	NCLB Severity (%)		SCR Severity (%)	SDM incidence (%)
		Kharif 2012	Kharif 2013	Kharif 2013	Kharif 2013
316	(CML153 × SKV50)-643	50.75	42.69	31.90	45.84
317	(CML153 × SKV50)-644	56.77	31.94	42.10	45.84
318	(CML153 × SKV50)-646	44.98	35.65	41.14	56.62
319	(CML153 × SKV50)-648	46.41	38.04	42.10	57.05
320	(CML153 × SKV50)-652	33.20	31.94	40.66	67.05
321	(CML153 × SKV50)-653	39.22	44.98	41.97	30.97
322	(CML153 × SKV50)-655	39.22	27.26	36.21	45.84
323	(CML153 × SKV50)-657	52.71	37.45	36.21	27.61
324	(CML153 × SKV50)-667	49.29	38.04	42.83	73.02
325	(CML153 × SKV50)-668	44.98	47.85	32.85	64.85
326	(CML153 × SKV50)-669	42.69	43.26	28.27	61.91
327	(CML153 × SKV50)-673	39.22	28.65	37.66	73.02
328	(CML153 × SKV50)-674	38.04	41.25	46.42	44.20
329	(CML153 × SKV50)-681	61.09	47.85	46.51	32.57
330	(CML153 × SKV50)-683	39.22	31.94	37.48	47.93
331	(CML153 × SKV50)-685	45.62	36.86	36.69	40.09
332	(CML153 × SKV50)-687	42.69	46.70	43.55	49.68
333	(CML153 × SKV50)-697	41.15	36.86	37.48	29.88
334	(CML153 × SKV50)-699	43.55	24.34	31.59	40.09
335	(CML153 × SKV50)-700	49.29	38.63	27.30	58.88
336	(CML153 × SKV50)-702	44.98	47.28	29.08	73.02
337	(CML153 × SKV50)-703	47.13	44.98	27.30	49.68
338	(CML153 × SKV50)-706	40.38	37.45	30.65	64.31
339	(CML153 × SKV50)-707	44.16	39.80	36.21	69.95
340	(CML153 × SKV50)-708	42.11	38.04	34.04	44.20
341	(CML153 × SKV50)-709	44.98	35.65	32.89	73.02
342	(CML153 × SKV50)-710	51.92	42.69	29.88	40.09
343	(CML153 × SKV50)-712	36.86	47.28	24.54	73.02
344	(CML153 × SKV50)-717	47.85	24.34	14.66	0.00