MODE OF INHERITANCE OF RESISTANCE TO COFFEE WILT DISEASE (*Gibberella xylarioides*Heim and Saccas) IN ARABICA COFFEE (*Coffea arabica* L.) GENOTYPES

MSc THESIS

BY

ADMIKEW GETANEH

MARCH, 2017

JIMMA, ETHIOPIA

MODE OF INHERITANCE OF RESISTANCE TO COFFEE WILT DISEASE (*Gibberella xylarioides* Heim and Saccas) IN ARABICA COFFEE (*Coffea arabica* L.) GENOTYPES

A Thesis Submitted to School of Graduate Studies,College of Agriculture and Veterinary Medicine
JIMMA UNIVERSITY

In Partial fulfillment of the requirements for the Degree of Master of Science in Plant Breeding

By

Admikew Getaneh

March, 2017
Jimma, Ethiopia

DEDICATION

This thesis is dedicated to the memories of my father **Getaneh Yigletu**,who did not live to see me get this far. Your support has always been great and this is your unreserved result. My father, I always remember your generosity and you lives in bottom of my heart, God keeps you in paradise.

STATEMENT OF THE AUTHOR

This is to certify that the research thesis entitled" Mode of Inheritance of Resistance to Coffee Wilt Disease (*Gibberella xylarioides* Heim and Saccas) in Arabica Coffee (*Coffea arabica* L.) Genotypes" submitted in partial fulfillment for the degree of Masterof ScienceinAgriculture (Plant Breeding) to Collage of Agriculture and Veterinary Medicine, Jimma University.I declare that this research thesisor any partthereofrepresents my own work and has notbeen submitted to any other institution elsewhere for the award of any degree, diploma or certificate. I have followed all ethical and technical principles for the proposition, data collection, data analysis and compilation of the thesis. Any scholarly matter that included in the thesis has been given recognition through citation.

Quotations from this thesis may be made without permission if accurate and complete acknowledgment of the source is provided. Request for permission for extended citations or copy of this thesis in whole or in part may be granted by the School of Graduate Study or the Department of Plant Science and Horticulture when in his judgment the proposed use of the material is in the interest of scholarship. In all other occasions, however, permission must be obtained from the author.

Name: Admikew Getaneh
Place:Jimma University
Date of Submission: _____
Signature: _____

BIOGRAPHICALSKETCH

The author, AdmikewGetaneh was born on November 17, 1987 inHagere-Mariam Woreda, North Showa, Amahara region, Ethiopia. He attended elementary education at Yenechoh and then,Hagere-Mariam primary school, and his secondary and preparatory education in Debre Berhan,Hailemariam Mamo Secondary and Preparatory School. Soon after the completion of his preparatory school in 2007, he joined in Debre Berhan University and completed undergraduate studies with a Bachelor ofPlant Science in 2010. He worked at Menschen für Menschennongovernmental organization and Kolfe Keranio Sub City Trade & Industry Development Bureau as Agricultural expert for about a year. Then, hewas employed in theEthiopian Institute of Agricultural Research, Jimma Agricultural Research Center, Gera Agricultural Research Sub Center as Junior Researcher in 2011. After three years of service, the author joinedthe College of Agriculture and Veterinary Medicine,Jimma University in October 2014to pursue hisMScDegree in Plant Breeding.

ACKNOWLEDGEMENT

First of all, my deep and heartfeltthanks to God for giving me strength to work this research thesis. I would like to thankthe Ethiopian Institute of Agricultural Research Center (EIAR) for financial support, College of Agriculture and Veterinary Medicine, Jimma Universityforproviding the necessary facilitiesand the learning environment and Jimma Agricultural Research Center to facilitate the research work providing me.

I would like to take this opportunity to thank those people who have had an impact on my research. First, I want to thank my advisors, Dr. Girma Adugna and Dr. Sentayehu Alamerew. My deep appreciation extended to Mr. Ashenafi Ayano, Director of Jimma Agricultural Research Center, for sharing his knowledge of coffee breeding.

I am highly indebted to my father Getaneh Yigletu, my mother Birke Laymekitu and my sisters Yetesha Getaneh and Elfinesh Getaneh for their moral supports and inspiration. I would also like to express my gratitude to my wife, Tigist Tesfaye for her support and patience. I am thankfulto Dr. Abush Tesfaye, Dr. Mandefro Nigussie, Dr. Leta Bedada, Afework Legese, Dereje Amare, Legese, Demelash Teferi, Kifle Belachew, Kelifa Nesro, Mebirate Kidane, Mamo Abeye, Abaynesh, Sisay Tesfaye, Desalegn, Wodmu andmany others, directly or indirectly participate in contributing their unique perspectives and support.I would also like to thank Niguse and Habtamu for their assistance in data collection, and also other employees for planting and maintaining the experiment. Lastly, I would like to thank my colleagues, Ermias Assefa, Gemechu Nadi, Lemi Beksisa, Mesfin H/Mriam, Tefera Abebe and Ahimed Getachew for their friendship and support.

ACRONYMS AND ABBREVIATIONS

ANOVA	Analysis of variance
BBC	Bacterial Blight of Coffee
CABI	CAB International
CBD	Coffee Berry Disease
CLR	Coffee Leaf Rust
CORI	Coffee Research Institute, Uganda, formerly Coffee Research Center (COREC)
CWD	Coffee Wilt Disease
DRC	Democratic Republic of Congo
FAO	Food and Agriculture Organization of the United Nations
JARC	Jimma Agricultural Research Center
GDP	Gross Domestic Product
GISH	Genomic in situ hybridization
ICO	International Coffee Organization
IPGRI	International Plant Genetic Resources Institute
MAFAP	Monitoring and analyzing food and agricultural policies
PWD	Pine Wilt Nematode
QTL	Quantitative Trait Loci
RFLP	Restriction fragment length polymorphism
SAS	Statistical Analysis System
SNA	Synthetic low nutrient agar
SNNPR	Southern Nations, Nationalities, and Peoples' Region

TABLE OF CONTENTS

Table of Contents **Page**

TABLE OF CONTENTS (Continued)

TABLE OF CONTENTS (Continued)

LIST OF TABLES

Tables **Page**

LIST OF FIGURES

Figure **Page**

LIST OF TABLES IN THE APPENDIX

Appendix TablePage

MODE OF INHERITANCE OF RESISTANCE TO COFFEE WILT DISEASE (*Gibberella xylarioides* Heim and Saccas) IN ARABICA COFFEE (*Coffea arabica* L.) GENOTYPES

ABSTRACT

Understanding the inheritance of resistance mechanism and estimation of combining ability, heterosis, heritability and association among traits provide valuable evidence in designing appropriate breeding programs andcoffee wilt disease (CWD) resistant variety development. However, there islackof such information and the study conducted to estimate combining ability, type of gene action, heterosis, heritability and correlation among characters. The experimentwasconducted in RCBD with three replications;eight parents, 28 F1 crosses (half diallel method) with one susceptible checkusing artificial seedling inoculation testin greenhouseat Jimma Agricultural Research Center (JARC). The analysis of variance showed highly significant differences ($p<0.01$)among the genotypes for wilted seedling percentage, incubation period, number of defoliated leaves and all seedling growth characters (height, stem diameter, average inter node length, petiole length, leaf area, number of nodes and leaves). The mean performance indicated that parents P2 (971), P5 (79233) and P7(974) exhibited lowmean wilted seedling percentage and small number of defoliated leaves with extended incubation period. Cross P7 x P8 revealed the lowest mean wilted seedlings percentage (20.56%) andelapsed extended incubation period (143 days), followed by crosses P2 x P7, P4 x P8 and P2 x P8. Percentage of better parent heterosis (BPH) for wilted seedling parentage and number of defoliated leaves showed non-significant and unappreciable in desirable direction. Only crosses P4 x P8 and P7 x P8 showed significant negative mid parent heterosis (MPH) for wilted seedling percentage. All growth characters exhibited low and inadequate BPH; while considerable MPH was noticed forincubation period,seedling height, average internode length, petiole length and leaf area in favorable effect.Both additive and non-additive gene actions were important in controlling the inheritance ofCWD resistance, incubation period and all growth characters, except stem diameter. Additive genetic variancebeing predominant for wilted seedling percentage, incubation period and leaf area.Parents P2, P7, P8 and P5 exhibited highly significant negative gcaeffects and good general combiners for CWD resistance. The sca effects of crosses P7 x P8 and P4 x P8 revealed good specific combiners with significant desirable MPH for low wilted seedling percentage and incubation period. In general, selection and hybridization could be an effective resistance breeding approach. Furthermore, wilted seedling percentage showed high broad sense heritability coupled with high genetic advance as percent of mean (GAM). Moreover, itrevealed highly significant negative correlation with incubation period, leaf area and stem diameter;while it showedpositive association with number of defoliated leaves. The study estimated the presence of little BPH and MPH, high heritability with high GAM and predominance of additive over non additive gene effects in controlling the inheritance of CWD resistance. Therefore, further study on F2, BC1 and BC2 generation both in greenhouse and multi-location (field condition),and QTL mapping study is needed. The resistant genotypes should be also evaluated for other major diseases, yield and important traits.

1. INTRODUCTION

Arabica coffee belongs to the botanical family Rubiaceae, genus Coffea. Itis the only known tetraploid chromosome number (2n=4x=44) and autogamous (self-fertile)in the genus. While, all other coffee species are diploid (2n=22) and self-incompatible(Krug and Carvalho, 1951; Carvalho, 1952; Charrier and Berthaud 1985). Allotetraploid nature of *Coffea arabica*also backed using GISH and RFLP analysis by Lashermes *et al*. (1999), suggested that it formed by hybridization between *Coffea canephora* and *Coffea eugenioides*. SouthwesternEthiopia is the primary center of origin and genetic diversity of Arabica Coffee (*Coffea arabica)* (Sylvian, 1955; Meyer, 1965). Anthony *et al*. (2001) and Anthony *et al*. (2002) using molecular markers also confirmed that Arabica coffee originated in southwestern Ethiopia.

From the genus Coffea, Arabica and Robusta coffee are the main cultivated species among more than 100 species (FAO, 2006). Arabica coffee,mainly produced in the Southern, South Western and Eastern parts of the country. The crop contributed about 31% of the foreign exchange earnings, 10% of the total agricultural production, 5% of GDP of the country and the country produced about 5% of world and 39% of the total Sub-Saharan coffee production (FAO, 2014). More than 90% of the production is from the garden, semi-forest and forest coffee systems of small-scale farmers; the remaining 10% comes from large-scale plantation. It is a source of livelihood for more than 25 million people (Kassahun and Getnet, 2008).

The total Arabica coffee production in Ethiopia (average of two consecutive years, 2014/15 to 2015/16) is about 4,172,883 quintalsfrom 607,836 hectare of land; 6.91quintals per hectare (CSA, 2016). However, the production is very low compared to world production and large coffee producing countries, such as Brazil, Vietnam and Colombia.This low coffee production is partly due to traditional coffee production systems, continued reliance on unproductive local coffeegenotypes, the widespread and prevalence of pests and diseases, the presence of abiotic stress and poor agronomic practices.These factors greatly reduce the productivity and quality of the product(Melaku, 1984; Girma *et al.*, 2009a). Coffee diseases such as, coffee berry disease (CBD), coffee leaf Rust (CLR) and CWD are the major diseases that reduce Arabica coffee production and quality(Eshetu, 1997; Eshetu *et al*., 2000).

CWD is a fungal disease that causes vascular wilt;caused by *Gibberella xylarioides* (Heim and Saccas, 1950; Geiser, *et al*., 2005). The fungus invades the coffee tree and colonizes the xylem system.Successive surveys by different scholars onthe occurrence and prevalence of*Gibberella xylarioides* in major coffee-growing regions ascertained the existence of the disease with varying intensity (Van der Graaff and Pieters, 1978; Merdassa, 1986; Girma, 1997; Eshetu *et al.*, 2000; Sihen *et al*., 2012). There were certain variations in the incidence of CWD between coffee fields at each locality that attributed to differences in their genetic makeup, age of coffee genotypes, cultural practices and environmental condition at a specific location.Generally, the prevalence and importance of CWD has been markedly increasing throughout coffee producing areas of the country (Girma *et al*., 2001; CABI, 2003; Girma, 2004). The national incidence and severity of CWD in Ethiopia is 28% and 5%, respectively. However, the incidence and severity varied from place to place in the range of 0-100% and 0-25%, respectively (CABI, 2003; Girma *et al*.,2009a).

A number of methods are used for CWD management. The common techniques used to protect the effect are uprooting and burning of infected coffee trees; prevention of tree wounding; use of protective fungicides in sealing wounds; use of disease free planting materials; disinfecting farm implement and use of biological control. However, these methods are difficult to implement; and use of resistant varieties is probably the most cost-effective, economical and eco-friendly method for the management of CWD, and is also relevant to smallholder coffee producers or farmers (Rutherford, 2006; Phiri and Baker, 2009; Girma *et al*., 2009a). According to Girma *et al.* (2005) there were highly significant differences between cultivars, isolates and cultivar-isolate interactions in seedling test using *Gibberella xylarioides*, suggesting the presence of certain qualitative (vertical) reaction with quantitative (horizontal) resistance. Van der Graaff and Pieters (1978) and Girma (2004) also reported the existence of Arabica coffee genotypes with variable levels of CWD resistance and identify resistant genotypes. Hence, breeding for resistance is important and should be the main objective.

To understand the inheritance control mechanism and improve disease resistance, agronomic and morphological characters,estimation of combining ability and heterosisis important parts of the breeding program.Exploitation of heterosis primarily depends on screening and selection of diverse genotypes for important characters. Combining ability helps to identify the best combining parents, to know the type of gene action and select appropriate breeding methods (Sprague and Tatum, 1942; Mathur and Mathur, 1983). Estimates of heritability along with genetic advance and the association between characters are also important selection parameters for plant breeder to select the required traits (Panwar *et al*., 2015).Musoli *et al*. (2013) investigated the inheritance of resistance to CWD in Robusta coffee and reported that the gene controlling resistance is polygenic; heritability of the disease is low to moderate. Therefore, selecting tolerant clones for CWD resistance improvement is possible, butdifficult to derive hybrid populations using parents to develop resistant varieties.

In Ethiopia,much effort has been made to improve coffee production, productivityand develop CBD resistant varieties through pure line selection and hybridization;about 36 improved varieties have been released (MOA, 2015).Despite, extensivework done to manage CWD, the genetic controlling mechanismof the disease is not initiated and implemented. Additionally, so far information has been lacking in combining ability, heterosis and heritabilityof CWD resistance in Arabica coffee, and its correlation with other CWD and seedling growth characters.Keeping the above views in mind, the study was conducted to meet the following objectives.

General Objective

To understand the mode ofinheritance controlling mechanism of Coffee Wilt Disease resistanceand growth characters.

Specific Objectives

1. To estimate combining ability,heterosis, heritabilityand identify the type of gene action controlling the inheritance of CWD and seedling growth characters
2. To determine the association of resistance to other CWD and growth characters

2. LITRATURE REVIEW

2.1.Biology of Arabica Coffee

Coffee is astimulant, woody perennial evergreen dicotyledonous plant. A mature coffee plant consists of a shoot and root system. Coffee flowers are white and fragrant; within each flower, there are five stamens with long anthers and short filaments inserted into the corolla tube and a pistil with a long, thin style having two branched stigma and an inferior ovary (Hadberg *et al*., 2003; Witengen, 2009). Flower buds generally open on sunny days early in the morning and pollen shedding starts soon afterwards. The stigma is receptive at the opening of the bud and found to remain receptive for three to four days, depending on weather condition (Carvalho *et al*., 1969). Walyaro and Van der Vossen (1977) later found that the stigma could remain receptive for at least nine days and recommended that the bags after pollination should not be removed for two weeks. Flowers start withering one or two days after pollination and fruits take 7 to 9 months from pollination to maturity. There are two or three relays of blossoms before all the buds reach maturity; the principal blossoms occur in late February to March under Ethiopian conditions.

Self-pollination is the transfer of pollen from anther to a stigma within the same flower or to a stigma of another flower on the same plant or within the same clone (Schlegel, 2003). From the breeding perspective, most crop species that reproduce by sexual means may be grouped according to their usual method of pollination as normally self-pollinated or normally cross-pollinated (Poehlman and Slepe, 1995). These groups are inclusive, because slight cross-pollination usually occurs in crops normally classified as self-pollinated, and some self-pollination usually occurs within the normally cross pollinated crops.Generally accepted that cultivated *Coffea arabica* is self-pollinating with 7% to 11% outcrossing rate (Carvalho *et al*., 1969; Charrier and Berthaud, 1985; Davis*et al*., 2010). Fertilization is takes place before or just at flower opening. However, Mayer (1965) observations and Gezahagn (2014) mating system analysis, about 40 % to 60% and 76% outcrossing were found in Arabica coffee, respectively.

2.2. Arabica Coffee Production Constraints

There are a number of production constraints that confront coffee producing farmers. Among these traditional agronomic practices, different biotic and abiotic factors are the most important constraints. Coffee diseases, such as coffee leaf rust, coffee berry disease, coffee wilt disease and bacterial blight of coffee are the major production problems(Mugiira *et al*., 2011). Theseconstraints can be improvedthrough good coffee management practices and using improved cultivars (Workafes and Kassu, 2000, Admasu and Klause, 2007).

2.3. Coffee Wilt Disease and its Importance

CWD is a vascular wilt disease syndrome; induced by *Gibberella xylarioides* Heim & Saccas (Kranz and Mogk, 1973).The pathogen enters into tree roots either through wounds or directly through root hairs and the epidermis of the small roots (Toole, 1941). Once a wilt pathogen has penetrated a suitable host through wounds, it moves to the vascular tissue. The pathogen then spread throughout the plant by means of mycelia growth or conidia, primarily micro conidia, produced in infected xylem vessel elements (Agrios, 2005). As the disease development progresses, the fungus invades tissues adjacent to the xylem tissues such as pith, cambium, phloem, and cortex. At this time, symptom expression is severe, and a portion of the plant or the entire plant may succumb to the disease (Nelson, 1981). The disease manifests itself after a prolonged incubation period, by expression of disease symptoms, including a rapid wilting and shedding of the foliage, and finally perithecia of the fungus is formed in the bark of the lower parts of the stem after complete tree death (Kranz and Mogk, 1973). Generally, coffee wilt symptoms progress from inward curling and wilting of leaves to bluish-black or brown-reddish stripes under the bark, die-back and death of affected trees (Girma *et al*., 2001).

CWD has been limiting coffee production and greatly distributed in many parts of Eastern, and Central African countries such as Ethiopia, Uganda, Democratic Republic of Congo, and Tanzania (Rutherford, 2006). Across the countries surveyed, a total of 1728 out of the 5505 farms (31%) were found to be infested with CWD; 1280 farms with Robusta coffee (75%) and 448 farms with Arabica coffee (12%). The CWD severity ranged from 0% in Rwanda to 45% in Uganda (Phiri and Baker, 2009). In Ethiopia, survey result showed that it decreased

yieldby 37%at the farm level and this led to a decline of 67% income. The annual national yield losses attributed to reached 3360 tons(CABI, 2003). This economic loss coupled with the difficult to manage the disease indicates that CWD is equally important toCBD (Girma *et al*., 2001; Girma *et al*., 2009a).

CWD was widespread in semi forest, garden and plantationcoffee production systems of Ethiopia and the incidence was significantly varied from 3.6 %to15.5%, 27.2% to 43.5% and 17.3% to 65.2%, respectively, indicating that the disease is more important inplantation followed by garden based production systems (Girma, 2004). Girma *et al*. (2001) also reported CWD incidence varied from 44% in Gera to about 69% in Bebeka and there were certain variations between Arabica coffee genotypes.Arega (2006) and Sihen *et al*. (2012)also assessed the presence of CWD in afro-mountain rainforest coffee areas; Harena (Bale), Berahane-Kontir (Sheko), Bonga and Yayu. They reported the prevalence of the diseases from 0% to 100 % incidence and the existence of high Arabica coffee genetic variability.

2.4. Coffee- *Gibberella xylarioides*Interaction

The study of host-pathogen associations involves a three-dimensional interaction between host varieties, pathogen strains and environmental variables that can affect disease expression. To be able to limit the effect of these factors on host-pathogen interactions, standard artificial screening protocols that discriminate between resistant and susceptible genotypes have been developed (Flood, 2006). Different inoculation procedures, such as stem nicking, root dipping and syringe injection have been used to screen andidentify genuinely CWD resistant genotypesby different countries (Pieters and Van der Graaff, 1980;Girma and Mengistu, 2000; Musoli, *et al*., 2001; Musoli, 2005). Stem nicking method of young coffee seedlings with inoculum suspension 2×10^6spore per milliliter of*Gibberella xylarioides* isolate at cotyledon stage (2 to 2.5 months old) using a scalpel has been adopted as the preferred standard practice on Arabica coffee.Thus**,** standardizing the inoculation protocols (methodologies), identifying proper growth stages of the host that show differential reactions, selection of aggressive strain/isolate and conditions that favor infection and wilt disease development are paramount importance in designing an effective screening and breeding program for CWD management (Girma *et al*., 2009b).

2.5. Breeding for Disease Resistance

Resistance is a relative term for the genetic based capacity of a host (plant genotype in which a pathogen or pest that produce an infection) to reduce the adverse effect of a pathogenic attack. On the other hand, the pathogen is a living organism that is capable of causing a distinct disease in another organism (Sharman, 2004). Therefore, breeding for disease resistance involves the manipulation of two genetic systems; one for plants (host) and the other for the organism (pathogen or pest) simultaneously (Acquaah, 2012).

The strategies adopted in breeding for disease resistance depends partly on what types of resistance are available, and also upon the type of disease. Plants are resistant to certain pathogens because they belong to outside taxonomicgroups (non-host resistance) or possess genes for resistance, or due to various reasons, escaped or tolerate infection. The major types of resistance are disease escape, tolerance and resistance to the pathogen (true or genetic resistance) (Russel,1978; Agrios,2005).Disease resistant cultivars are developed by identifying genes for resistance in the host species, or related wild species, and transferring the gene or genes into adapted cultivars and breeding lines, normally by hybridization or genetic engineering techniques. Resistance may be controlled by single geneor by polygenesdepending on the specialization of the pathogen and the nature of the resistance. There are two mechanism of host resistance; vertical and horizontal resistance.Vertical resistance or race specific genes are simply inherited and confer major resistance effects to particular racesor biotypes of the pathogen but not to other races. Horizontal resistance (non-race specificpolygenes) isinherited quantitatively, each contributing a small increment of control of the disease pathogen (Poehlman and Sleper, 1995). According toVan der Plank (1984), highly significant differences among the host and the pathogen (main effects) indicate the existence of horizontal resistance in the host and variation in aggressiveness in the fungus population, but a significant interaction between the cultivars and the isolates (differential effect) implies vertical resistance in the host and virulence in the pathogen.

2.6. Breeding for Coffee Wilt Disease Resistance

Resistance to wilt disease depends in part on genetic potential for virulence within the pathogen populations and the inoculum's concentration (Beckman, 1987). The resistance of a plant (or tissue) changes sequentially during growth and development; thus, certain growth stages are more favorable than others for comparison of resistant and susceptible cultivars.

Currently, attempts to control CWD are fundamentally based on the breeding of resistant plants, environmental management, and synthetic fungicide application (Strange, 1993). The high cost of pesticides, the emergence of fungicide-resistant pathogen bio-types and other social and health related impacts of conventional agriculture on the environment, recently led to an increased interest in agricultural sustainability, and biodiversity conservation (Cook *et al*., 1996; Van der Vossen, 2005). Thus, there is a need for sustainable solutions such as biological control agents and integrated disease management to reduce the problem that could provide effective control. These solutions minimize cost and chemical application for establishment of sustainable agricultural development and eco-friendly for human health and the environment. Now, CWD resistance breeding is an important reminder that conventional plant breeding still has a place in the armament of the modern plant breeder (Kangire, 2014).The presence of two pathogenic forms within coffee wilt disease populations (Arabica and Robusta strains) also suggested that the ability to design effective CWD management strategies, develop resistant cultivars or lines and formulate appropriate breeding programs towards each population group (Girma *et al.*, 2005).

2.6.1. Sources of resistance

The source of genes for resistance is the same gene pool of the crop that provides genes for inherited resistant characteristic; namely, older varieties, abandoned, earlier or discarded breeders' stock, wild plant relatives, other native or foreign commercial varieties, and sometimes, induced mutations (Agrios, 2005). Intraand inter specific differences of coffee speciesprovidea potential source to exploited CWD resistance. Intra specific variability is the best and easiest to exploit since resistant individuals are easily released as new varieties without undergoing hybridization;if itpossessesgood other agronomic traits (Musoli, *et al*., 2009).

There were evidences indicating variations in resistance or tolerance levels in Arabica coffeecultivars under field conditions (Girma *et al.*, 2001; Girma, 2004). Seedling and conidium germination test on six host genotypes with four*Gibberella xylarioides* isolates support the conclusion that the resistance is of a horizontal nature (Pieters and Van derGraaff, 1980).There were also highly significant differences among cultivars, isolates and cultivar-isolateinteractions in seedling test;suggesting the presence of certain qualitativevertical reaction with quantitative horizontal resistance (Girma *et al*., 2009b). In Arabica coffee different investigators also reported that the available genetic diversity is high enough to be exploited for resistance against CWD (Van der Graaff and Pieters, 1978). Based on standard screening procedures varieties Catimor J-19, Catimor J-21, 7440 and 8136 showed resistant to moderately resistant reaction to CWD (Girma, 2004). Among the later, released group, Sidama / Yirgachefe varieties 971 (*Fayate*) and 974 (*Odicha*) show resistant reaction to CWD (Chala *et al*, 2012).But, Demelash and Kifle (2015) reported that most of the released Arabica coffee varieties are susceptible to CWD under greenhouse condition. According to Demelash (2013) work, 370 and 279/71 genotypes indicated CWD resistant as compared to standard resistant check (catimor J-19).However, Phiri and Baker (2009) are uncertain for the resistant coffee genotypes in Ethiopia and thought, there is no a clear-cut information about which Arabica coffee genotypes markedly showed CWD resistant.

Robusta coffeehashigh genetic variability and the genetic diversity among *Coffea canephora* populations at molecular level was attributed to variations (heterozygosity) within individuals (Musoli, 2007). A breeding program in Uganda has screened 1500 CWD resistantRobusta lines. Further screening and evaluation for a range of agronomic traits, it reduced to seven lines that fulfilled the overall qualities (Phiri and Baker, 2009).Furthermore, in Tanzania, 875 Robusta lines werescreened and six CWD resistant Robusta lines were selected(Kilambo *et al*., 2012).

2.6.2. Inheritance of resistance

Inheritance is the transmission of genetic information from parents to progeny(Schlegel, 2003). This transmission mechanism of genetic information is paramount and important to design effective breeding methods to any crops. The most important considerations to know about traits inheritance are whether dominant or recessive gene controlled resistance or it controlled by monogenic, oligogenic or polygenic and whether or not cytoplasmic inheritance involved. Furthermore, the relative importance of additive and non-additive gene actions (dominance and epistasis) in controlling traits also useful to decide appropriate breeding method. Understanding the inheritance of resistance mechanism and the type of resistance are necessary to determine appropriate breeding program to develop CWD resistant varieties (Musoli *et al*., 2009).

Musoli *et al*. (2013) studied the inheritance of resistance to*Fussarium xylarioides*in crosses of *Coffeacanephora*usingpartial diallel progeny and a half-sib progeny test and suggesting polygenic control of the resistance.Based on breeding for fusarium wilt race 4 resistance in Pima cotton, resistance differences detected between F1 hybrids and the mean of the parents in foliar symptoms and vascular discoloration; the bimodal distribution observed on the recombinant inbred line(RIL) population provide strong evidence for a dominant gene effect (Ulloa *et al*., 2006).

Based on Bayetta (2001) result from genetic studies together with van der Vossen and Walyaro (2009) study on identification of molecular markers linked to a gene conferring resistanceto CBD, providedan evidence for oligogenic inheritance of CBD resistance. Changaya *et al*. (2012) conducted an experiment on inheritance of resistance to Fusarium wilt and yield traits in pigeon pea using Line x tester mating design. They conclude One to two genes governing fusarium wilt resistance in pigeon pea.Mert *et al*. (2005) reviewed on the inheritance of resistance to Verticillium wilt (caused by both the defoliating (D) and non-defoliating (ND) pathotypes) in cotton. Tests of F2:3 families inoculated with defoliating pathotype confirmed that resistance was controlled by a single dominant gene;while resistance to the non-defoliating pathotype is governed by dominant alleles at two loci.

2.6.3. Hybridization for CWD resistance

Hybridization is a method of breeding, new cultivars that utilizes crossing to obtain genetic recombination or it is the crossing of individuals of unlike genetic constitution. Selfing and crossing are essential procedures in the hybridization program. The exact procedures employed depend upon the crop species, the structure of the floral organs, and the type of pollination, i.e., cross pollination or self-pollination (Poehlman and Sleper, 1995).

Hybrids have been generated through artificial pollination to combine CWD resistant clonesor genotypes and the complementary traits found in the commercial clones or varieties (Musoli*et al*., 2009). The hybrid progenies generated in such crosses are evaluated as individual trees for CWD resistant and agronomic traits starting at the screen house through field trial testing. Then, bestperforming individuals (genotypes) can be selected, cloned and planted in multi-location trials for adaptation and adoption tests and released to farmers. It is expected that progenies involving parents from different population anddistant geographical locations intend to benefit from hybrid vigor; derived from double heterozygosity of the parents. Due to the specificity of the pathogen populations affecting the different commercial coffee species, resistant varieties can be also derived through inter-specific hybridizations; even though it is complicated and difficult to derive a variety of desired quality (Phiri and Baker, 2009).

2.7. Diallel and Combining Ability Analysis

A diallel mating design is a type of mating design that requires the making of all possible crosses among a given number of parental genotypes to study the inheritance of quantitative traits(Gardner and Eberhart, 1966).It is popular because it can yield more information on general combining ability (GCA) of parents, specific combining ability (SCA) of crosses, genetic variance components and heritability. It has also been used to estimate gene action, heterosis, and inbreeding depression involved in determining quantitative traits.

The genetic material evaluated in the diallel mating design includes parents and the progenies obtained by crossing; those individuals in all combinations.The number of entries that are evaluated for the diallel mating design is determined by the number of genotypes (parents) used for crossing. If the number of parents is designated as p, the number of pairwise matings

among them is equal to P2. In order to make a diallel analysis and to generate the above information, the Griffing's and Hayman's approach are the two main stratagy being followed (Singh and Chadhaury, 1985). Griffing's diallel analysis has been widely used by plant breeders and no genetic assumptions requires. It has been shown reliable information on the combining ability potential of parents. According to Griffing (1956) there are four combinations of crosses and the parents that can be included for evaluation in the diallel mating design: Method 1 includes parents, F1's and reciprocal crosses p (p+1); Method 2 embrance F1'scrosses and parents (p (p+1) /2);Method 3 consistest of F1's and reciprocal crosses p (p-1), and Method 4 usedtoF1 crosses (p (p-1) /2) only. Moreover, half or partial diallel mating designs is widely used in crop and tree breeding program (Huber, 1992; Yanchuk, 1996).Two models; Model 1 (Fixed effects model) and Model 2 (Random effects model) can be used depending on parental lines selection to analysis of combining ability. Fixed effects model implies that parents were not randomly chosen and can not regarded as a random sample from a population.While, the random effects model implies that parents were randomly chosen from a given population.

Combining ability is defined as the performance of a parent in hybrid combination (Kambal and Webster, 1965). It can be partitioned into two components; variance due to general combining ability (GCA) and variance due to specific combining ability (SCA). General combining ability is the average performance of a parent in hybrid combination and is recognized primarily as a measure of additive gene action. SCA describes certain hybrid combinations do relatively better or worse than what would be expected on the basis of the average performance of the parents, and is regarded as an estimate of non-additive gene action (dominance and epistasis) (Sprague and Tatum,1942; Hallauer and Miranda, 1988). Generally, estimation of GCA and SCA helps to identify superior parents, which can be utilized in hybrid and cultivar development. The best performing progeny may be produced by crossing the two parents having the largest GCA effects. If SCA effects are significant, then the relative size of mean squares for GCA and SCA can be used to assess the relative importance of GCA and SCA. A relatively large GCA to SCA variance ratio suggests the importance of additive gene effects, and a low ratio indicates the presence of dominant and / or epistasis gene effects (Griffing,1956).

Gene effects do not always fall into clear-cut categories, and quantitative traits are governed by genes with small individual effects; which are often described by their gene action rather than the number of genes, which encoded them. There are four types of gene action; additive, dominance, epistasis, and overdominance.The effect of a gene is said to be additive when each additional gene enhances the expression of the trait by equal increments.This means, the phenotype reflects the genotype in additive action, assuming the absence of environmental effect. whereas, dominace gene action isthe heterozygote is more like one parent than the other, and epistasis gene action is the interaction of alleles at different loci.Overdominance effects occur when each allele contributes a separate effect, and the combined alleles contribute an effect greater than that of either allele separately (Poehlman and Sleper, 1995). Understanding the types of gene action is critical to the success of plant breeding. It is used for selection of parents used in crosses to create segregating populations, choice of the method of breeding used in crop improvement and to gain understanding of the breeding material by estimating genetic parameters. When additive gene action predominates in a self-pollinated species, breeders should consider using selection methods such as pure line selection, mass selection, progeny selection and hybridization. However, when non-additive gene action predominates, effective methods of breeding are the exploitation of heterosis in breeding hybrid cultivars (Acquaah, 2012).

In Ethiopia, different scholar estimate combining ability using half diallel crosses and studied the inheritance of Arabica coffee yield, disease and growth characters. Mesfin (1982) studied a five parent diallel cross (involved CBD resistant cultivars), both GCA and SCA variances were significant for stem girth and number of primary nodes characters. The study in six parents using half diallel at seedling stage in the nurseryrevealed highly significant GCA and SCA mean squares for 18 and 16 seedling characters, respectively, including seedling height, inter node length, stem girth, node number, leaf area, number of leaves and tap root length traits (Bayetta, 1991). The variance ratios computed indicated the predominance of non-additive genetic variance for most of growth traits, except number of true leaves, taproot and lateral root length. According to Bayetta (2001) study in five coffee parents using half diallel crosses, both additive and non-additive gene actions were important for the expression of plant height, stem diameter and number of main stem nodes, but for inter node length of stem and leaf area only additive gene action was important. He also reported

that parents with good GCA effects did not produce good specific combinations and hybrids with highest SCA effects could be rise from any possible combination of parents. Similarly, other workers stated that analysis of variancedue to GCA and SCA was significant for growth and leaf characters, including plant height, average inter node length, number of nodes on the stem and stem girth traits(Wassu, 2004; Ashenafi, 2013). These results indicate both additive and non-additive gene actions were involved in the inheritance of these traits.

In Kenya, Walyaro (1983) evaluated 11 parent using diallel cross for selected growth characters, including height, girth, inter node length and yield characters. He reported the importance of both additive and non-additive gene action for growth characters studied and concluded the predominance of additive genetic variance over non-additive genetic variance. Reciprocal differences for almost all the characters studied were insignificant. Musoli *et al*. (2013) studied on Robusta coffee wilt disease and reported that estimated of general combining ability was significant for seedling mortality percentage. However, SCA was non-significant, except the first assessment and additive and dominance variances were low for CWD compared to the environmental variance.

There are also many research studies about combining ability and genetic variability for disease and quantitative growth traits for different crops. According to Changaya *et al*. (2012) stated that bothadditive and non-additive gene actions were significant and responsible for fussarium wilt resistance in pigeon pea. Viands (1985) studied genetics of resistance to verticillium wilt in two alfalfa cultivars. His quantitative genetic analyses indicated that resistance in each cultivar was controlled by additive genes. According to Acharyaand Huang (2003) genetic analyses on the resistance mechanism in alfalfa plants have indicated that verticillium wilt resistance is controlled by multiple genes.

The genetics of resistance to downy mildew (*Pseudoperonospora cubensis*) in the muskmelon (*Cucumis melo*) was studiedby means of a half diallel between 8 inbred lines (Epinat and Pitrat, 1994). Analysis of variancedetected highly significant general combining ability effects. Although SCA effect was significant, it had a low impact on total variation and therefore,the presence of heterosis for resistance was not revealed. Decomposition of dominance variation indicated that the dominance effect was not unidirectional and

dominantgenes were not uniformly distributed among the parents. Based on Mwanga *et al*. (2002), both GCA and SCA for resistance to sweat potato virus disease (SPVD) showed significant effects. But, GCA to SCA variance component ratios was large and indicating that additive gene effects were predominant in the inheritance of resistance to SPVD.

Azad *et al*. (2014) studied on six diverse maize inbred lines using a half diallel mating design and they observed significant general combining ability variances only for cob height,but specific combining ability variances was observed for plant height, cob height, cob length, cob girth, number of kernels per cob, cob weight and hundred grain weight. The GCA to SCA ratio was less than unity for all studied traits except shelling percentage.

2.8. Heterosis

Heterosis is synonymous with hybrid vigor, which is defined as the superiority in size, vigor, or productivity of a hybrid or F1 over its parents (Ghaderi *et al.*, 1984). One of the most important factors determining the feasibility of producing a hybrid is the nature and extent of heterosis that exist in the population under improvement. An alternative term, heterosis, was proposed by Shull (1952) to denote the stimulation in size and vigor in a hybrid as an expression of hybrid vigor. To be useful, the hybrid plant needs to exceed the best parent in yield and productivity. Unless a hybrid is superior to its best parent line, it has no advantage for the breeder or the farmer. According to Rood *et al*. (1986), the effects of hybrid vigor in plants are manifested by reflecting in cell size, increased rates of metabolic reaction or more efficiently organized metabolic system. Furthermore, hybrid vigor increased vegetative growth and yield of the harvested product. It is generally greatest following crosses among diverse genotypes of a cross-pollinated species, but it may be also expressed following crosses among diverse genotypes in a self-pollinated species (Poehlman and Sleper, 1995).

There are four basis of heterosis explanation; which are genetic, physiological, biochemical and molecular, but the specific genetic basis has not been fully determined. However, two schools of thought have been advanced to explain the genetic basis for heterosis. These are the dominance theory, proposed by Davenport (1908) and the over dominance theory, proposed by Shull (1908).According to the dominant hypothesis, heterosis results from the masking of harmful effects of recessive alleles by dominant alleles and it is not the result of

heterozygosityto improve a trait. It suggests that at each locus the dominant allele has a favorable effect, while the recessive allele has an unfavorable or neutral effect (Singh, 1993).Furthermore, a genotype with more dominant alleles will be more vigorous than one with few dominant alleles. The possibility to accumulate all the favorable dominant alleles into one homozygous strain by selection and obtain inbreeds that is as vigorous as hybrids and the F2 distribution skewed because of the ¾ dominants to ¼ recessives segregation are the two difficulties of dominant hypothesis (Lamkey and Edwards, 1999).

On the other hand, the over dominance hypothesis states that the hybrid is better than either of the parents because the heterozygote is phenotypically different from and superior to both homozygote parents. This could happen if the two alleles specified different products. The most vigorous hybrid plant being the one with the greatest number of heterozygous loci (Poehlman and Sleper, 1995).In this theory the objection is the absence of clear-cut circumstance where the heterozygote is superior to the two homozygotes. But, there is no doubt that in the case of some genes, heterozygotes are superior to the homozygotes (Singh, 1993).Using molecular markers, Stuber *et al*. (1992) were able to detect quantitative trait loci (QTL) contributing to hybrid vigor in maize. The results from their findings showed that the heterozygotes of most QTLs detected for grain yield had higherthan homozygotes, suggesting that over dominance is the principal factor controlling heterosis in the study. Generally, the dominance theory is the more favored of the two theories by most scientists, even though neither is completely satisfactory (Acquaah, 2012).

The measurements of heterosis in the crosses were expressed as on the basis of the mid parents, better parents and economical (standard) parent. The selection of appropriate measurement is based on the objectives of the breeders and the estimate of each heterosis is according to Falconer and Mackay (1996). Heterosisis studied for Arabica coffee for yield,yield related and disease resistance (such as CBD)traits by several investigators. Significant heterosis relative to mid parent (MPH %) and better parent (BPH %) were observed in yield, plant height, stem girth and average inter node lengtheither in the positive or negative direction (Wassu, 2004; Ashenafi, 2013).As stated by Mesfin (1982), hybrid and parental mean susceptibilities to CBD and percent susceptibilities of the hydrides over the mid parent and susceptible parent values were +2% to +51% and -15.5% to +29.5%, respectively

and hybrids expressed up to 12% heterosis over the better parent for stem girth.The hybrids resistance x susceptible were significantly susceptible to their mid parent values.Bayetta (1991, 2001) also reportedthat positive and significant better parent heterosis forstem diameter and yield. But, he observed unimpressive amount of heterosis for most growth characters. Moreover, he concludes that there is an opportunity to develop and exploit the maximum potential of heterosis through hybridization of different origin, morphology, biotic and abiotic stress genotypes for the required characters.

2.9. Heritability

The concept of the reliability of the phenotypic value of a plant as a guide to the breeding value (additive genotype) is called the heritability of the metrical trait (Allard, 1999; Acquaah, 2012). Heritability is the proportion of observed variability, which is due to heredity, the remainder being due to environmental causes. The effectiveness of selection for a trait depends on the relative importance of genetic and non-genetic factors in the expression of phenotypic differences among genotypes in a population (Fehr, 1987). Heritability can be expressed in a broador narrowsense (Hurtl, 1994). Broad-sense heritability is the ratio of the total genotypic variance to phenotypic variance. Heritability in the narrow-sense is the ratio of the additive genetic variance to phenotypic variance and depends on the proportion it affects genetic gain (Falconer and Mackay, 1996). The main purpose of estimating heritability and genetic parameters is to estimate the expected genetic gains from selection based on alternate strategies (Holland *et al*., 2003).

Musoli *et al*. (2013) reported the genetic variance components drastically decreased, corresponding to increasing tree mortality. Resulting broad (h2b) and narrow (h2n) sense heritability estimates drastically reduced with successive assessments. The broad sense heritability was moderate (0.329), and the narrow sense heritability was low (0.112). This shows that CWD resistance is heritable, but its transmission from parents to progenies is only about 33%; therefore, low genetic gains of choosing a progeny of resistant parents as source of planting materials for a garden production. Commercial CWD-resistant Robusta coffee varieties should therefore be propagated vegetatively to retain the resistance.Epinat and Pitrat (1994) reported that high narrow sense heritability (0.82-0.88) and low non-additive gene

effect indicated that downy mildew resistance in muskmelon can be effectively handled through recurrent selection methods. Furthermore, Mwanga *et al*. (2002)estimated moderate narrow-sense heritability (31–41%) and high broad-sense heritability (73–98%); indicating that rapid genetic gains for SPVD resistance could be accomplished by mass selection breeding technique.

2.10. Correlation amongCharacters

Correlation is a measure of the degree of association between traits and the association may be on the basis of genetics or may be non-genetic; between two or more traits (Hallauer and Miranda, 1988; Acquaah, 2012). If a genetic association exists, selection for one trait will cause the changes on other traits. This response to change by genetic association is called correlated response; it may be caused by pleotropism or linkage disequilibrium. Pleiotropism is the multiple effect of a single gene (i.e., a single gene simultaneously affects several physiological pathways). In a random mating population, the role of linkage disequilibrium in correlated response is only important if the traits of interest are closely linked.

Several studies have suggested that morphological traits (height, basal diameter of the axis, and the number of branches) of Japanese pines are associated with resistance to PWN (Toda *et al*., 1986; Toda and Fujimoto, 1987; Kuroda, 2004).These morphological traits showed different levels of relevance among individuals. Yamanobe (2009) supposed thata thicker basal diameter was better survived. Pine trees can survive as long as there is a partial passage for xylem and phloem transport, even if almost no transport occurs (Kuroda, 1999). Trees with a wider diameter at the base may have a greater potential to retain functional passages than thinner trees. With respect to the number of branches, subjects with morebranches would survive better as long as there are more branches below the inoculation position. For Upland cottons, significantnegative correlations were observed between foliar damage or vascular discoloration with number of nodes and plant height. These significant correlations indicated that the reduction in plant growth related to symptoms (Ulloa *et al*., 2006).

Agrios (2005) stated inoculation of fussarium wilt resulted in clogging of xylem vessels by mycelia, spores and tyloses. Crushing of vessels by proliferating adjacent parenchyma cells was also observed, which hamper the translocation of water on the infected plants. The leaves of infected plants transpire more water than the roots and stem can transport, resulting in wilting symptoms. That is why growth and transpiration were reduced in fussarium wilt infected plants. Study wasconducted to assess the effects of wilt fungus on growth and transpiration of chickpea. Results showed inoculation of *Fussarium oxysporum* reduced plant growth, transpiration and caused severe wilting (Siddiqui and Singh, 2004).Walyaro and Van der Vossen (1979) also studied on 16 Arabica coffee varieties and they reported phenotypic correlation is generally much lower than the genotypic ones, indicating that the inherent association between characters is strongly influenced by environmental causes. The girth at the base of the main stem is genotypically correlated with height. Accordingto El-bramawy *et al*. (2009), the regression analysis of branch number and seed color in sesame were significantly correlated with fusarium wilt and charcoal rot diseases infection percentages. Therefore, these traits may be used for direct selection of sesame accessions that are resistant to fusarium wilt and charcoal rot disease.

3. MATERIALS AND METHODS

3.1. Description of the Study Area

The study was conducted at Jimma Agricultural Research Center (in greenhouse), Southwest Ethiopia. Jimma is found 7^{o}46'N latitude and 36^{o} E longitude coordinate and at an elevation of 1753 meter above sea level in Jimma Zone, Oromia Regional State. The study is located 358 kilometers away from Addis Ababa and 12 kilometer from Jimma town in the west direction.

3.2.Coffee Genotypes and Experimental Design

SeventeenArabica coffee genotypeswith different CWD resistance reactions(resistant, moderately resistant and susceptible) were testedbased on artificial inoculation test or natural CWD infested soils.The genotypes were obtained from Jimma, Gera and Tepi Agricultural Research Centers. Then, eight promising coffee parents (from CWD resistance verification trail), namely 75227 (P1), 971 (P2), 74110 (P3), 8136 (P4), 79233 (P5), 74144A (P6), 974 (P7), 370 (P8) and one susceptible check (Geisha) were selectedbased on the results of verification, yield and some other agronomic traits. These parental lines were generated from different CWD reaction groups; parental lines 971 (P2), 79233 (P5) and 974 (P7) relatively resistant; 370 (P8), 8136 (P4), 74144A (P6) moderately resistant; 75227 (P1) and 74110 (P3) susceptible parents (Table 1). Then, eight parents were crossed in 8 x 8 half diallel mating design using Griffing (1956) method 2 and model I at Gera, southwest of Ethiopia in 2014.The resulting 28 F1 crosses along with eight parents and one susceptible check were studied from 2015 to 2016. The above symbols and designation of the parental lines are the same throughout this thesis.

The experiment was laid out using randomized complete block design (RCBD) with three replications in the greenhouse using heat sterilized and moistened sandy soil in the disinfected plastic pots (each has 5652 cm^3 capacity). Five seedlings from each pot were sampled and the growth traits were recorded for each genotype.

Table1.Description of Arabica coffeegenotypes used for the study

Serial No.	Coffee genotypes	Origin	Breeding method	Released or collection year	Some characters description
1	75227	Gera, /Jimma	Selection from local landraces	1980/81	Open growth habit, good yielder, green tip leaf color, CBD resistant, susceptible to CWD (Demelash andKifle., 2015)
2	971	Gelana Abaya/ Borena	Selection from local landraces	2010	Resistant to CWD(Chala *et al*., 2012)
3	74110	Metu / Illubabor	Selection from local landraces	1978/79	Resistant to CBD, susceptible to CWD, good yielder, compact growth habit, green tip leaf color(Demelash andKifle., 2015)
4	8136	Gera/ Jimma	Selection from local landraces	2006	High yielding potential with consistence bearing habits, resistant to CBD &CLR, vigorous with intermediate growth habit, stiff stem, manageable height, moderately resistant to CWD (Girma, 2004)
5	79233	Introduce from France	International collection	1979	Green tip leaf color, CWD resistant under natural infested soil (no tree death)(personal observation)
6	74144A	Balle/ Oromia	Under collection breeding program	1978/79	Moderately resistant to CWD under natural infested soil, susceptible to CBD, high quality, compact growth habit, broth leaf color (personal observation)
7	974	Gelana Abaya/ Borena	Selection from local landraces	2010	Broth tip leaf color, compact growth habit resistant to CWD (Chala *et al*., 2012)
8	370	Seka-Chekorsa/ Jimma	Selection from local landraces		Resistant to CWD, broth tip leaf color, susceptible to CBD (Demelash, 2013)
9	Geisha*	Introduced from India	International collection	2002	Green tip leaf color, highly susceptible to CWD (Demelash, 2013)

*= susceptible check, Source: JARC / Coffee Breeding and Genetics division database for genotypes origin, breeding method and some characters description

3.2.1. Selfing and crossingtechniques

Two to three uniform trees were designated from each genotype and healthy branches with sufficient flower buds were selected from each tree, marked and coveredbefore flowering in February, 2014. Any flowers that had already opened and young developing buds were carefully removed without damaging the rest using hand, leaving only vigorous and yellow to whitish buds that were uniformly ready to open. Each selected branch was covered before flower opening using waterproof paper bag until complete shedding of the petals.

Crossing:before coffee flowering (blooming stage),two to three uniform trees were designated from each genotype and branches were selected and prepared similarly to selfing. Then, antherswere removed with whole corolla, together with the attached stamens, above the middle of the corolla tube by hand without damaging the pistil. The emasculated flowers or section of the branches was covered with waterproof paper bags at both ends, to protect them from stray pollen. On the same day of emasculation, mature, whitish and ready to open flower buds from male parent trees were collected and taken into the laboratory. The collected flowers were put under full light condition. In the following morning or one to three days following emasculation, open flowers were collected using labeled petri dishes and transported to the site of the female parent in the field. The covered emasculated flowers were opened at the upper end and the male flowers gently dusted or rubbed carefully against the stigmas to make fertilization. Immediately after pollination, the paper bags were closed and the branches labeled with cross number. Then, the bags were removed 10-15 days after pollination (after complete shedding of petals from flowers of the surrounding area). Frequent visit and follow up were made to remove any emerged new flower buds and proper maintenance of labels on the branches until harvesting.

3.2.2. Coffee seedling raising

Each genotype seeds were soaked in distilled sterile water separately for about 48 hours after removing the parchment and forty seeds of each genotype were sown in heat sterilized and moistened sandy soil. (Girma and Mengistu, 2000).Sterile water applied every one day interval to maintain adequate moisture for seed germination, emergence and growth of the plants. After germination, the seedlings thinned into twenty five seedlings per pot (20 seedlings were used for

artificial inoculation test and five seedlings for control). Then, five inoculatedsample seedlingswere selected from each pot and labeled yellow thread in order to measure growth characters.

3.2.3. Inoculum preparation

A representative Gera isolates of *Gibberella xylarioides* were taken and multiplied for inoculation using the method of Pieters and Vander Graaff (1980) with some amendments of Girma and Mengistu (2000). The stock culture of the representative isolate was used to initiate colony growth by sprinkling grains of sand onto Petri-dishes with SNA followed by further sub-culturing on the same medium and simultaneouslycolony growth using fresh coffee branches;collected from healthy trees, cut into small pieces of 15 cm and the bark was slightly scratched off to expose the wood. Then, the branches were placed in a test tube (3.75 cm^3) having a small roll of well-moistened cotton wool underneath and sterilized in the autoclave. Each of a batch of 10 twigs was inoculated with 2-3 ml of conidia suspension of the isolate and incubated for 14 days.The conidia used for seedling inoculation were obtained by thoroughly rinsing off the branch's good colony growth into sterile water in a sterile beaker. The suspension of the isolate was stirred up with a magnetic stirrer and filtered through double layers of cheese cloths. The concentration of spore suspension was counted with haemo-cytometerand adjusted to about 2 x 10^6 conidia/ml (Girma *et al*., 2009b).

3.2.4. Coffee seedlings inoculation and management

Twenty seedlingsper pot for each genotype were inoculated at fully opened cotyledon stage (10 weeks old) with viable conidial suspension of *Gibberellaxylarioid*es by stem nicking technique (Pieters and Van der Graaff, 1980; Girma and Mengistu, 2000). Sterile scalpels were first immersed into the suspension, and then each seedling was nicked at 2 cm above the soil level and dropped of nearly 1 milliliter in the notch. The treated plants were immediately kept in an air conditioned growth room with high relative humidity (>95%) and optimum temperature (23-25^oC) for infection. After 10 days, the inoculated seedlings were transferred to greenhouse with a temperature of 15-30^oC and relative humidity of 60-80% (Girma *et al.,* 2009b). After six months

of inoculation re-isolation of the pathogen from wilted coffee seedlings was carried out and confirmed the existence of *Gibberella xylarioid*es.

3.2.5. Coffee wilt disease assessment

An effective and reliable method of quantifying resistance wasapplied for comparison of results and selection of genuinely resistant genotypes. Wilted seedling percentage wasassessed as numbers of infected plants compared to non-infected to determine relative resistance between genotypes. The numbers were expressed as percentage infection (Girma and Mengistu, 2000; Girma *et al.*, 2009b; Musoli *et al.*, 2009).

3.3.Data Collected

Coffee wilt disease parameters

Wilted seedling Percentage: thenumber of wilted or dead seedlings were countedat fourteen days interval for six months (12 times),starting a month after inoculation

Incubation periods (days):recorded the number of days frominoculation to the first disease symptom appearancewithin two days interval

Number of defoliated leaves per seedling: counted the number of defoliatedleaves (true leaves) per seedling fromfive selected seedlings at 4 months old seedling stage

Number of yellow leaves per seedling: counted the number of true leaves changed to yellow and expressed wilting symptoms per seedlingat 4 months old

Growth characters

Seedling stem height (cm):measured seedling from the ground level to the tip using centimeter from five sampled seedlings

Seedling stem diameter (mm): measured at 2 cm above the ground level using caliper

Average internode length on stem (AINL): computed per tree as (Walyero, 1983):

(TH-HFTL)/NN-1

Where, TH = total seedling height,

HFTL = height up to the first true leaves,

NN = number of main stem nodes

Numbers of stem nodes per seedling (NN): counted the number of main stem nodes above and including the first true leaves from five sampled coffee seedlings

Number of leaves per seedling:counted the number of emerging true leaves in each seeding from average of five randomly selected seedlings per pot

Leaf petiole length (cm): measured average of five leaf petiole from the base (stem and leaf joining point) to the insertion of leaf blade

Leaf area (cm^2): measured average of five mature leaves per pot (length x width in broadest portion x 0.88 (Walyero, 1983).

All growth characters data were measured at 4 months old seedling stage based on IPGR (1996) coffee descriptors.

3.4.Statistical Analysis

CWD and growth characters mean values of the five randomly taken seedlings from 28 F1 crosses, including eightselfed parents either with or without checkwere subjected to analyses of variance (ANOVA) using SAS program version 9.2 (SAS, 2008). Fisher's least significant different mean separation tests were performed to identify and comparison of genotypes means that are significantly different from each other. The analysiswas carried out according to the following model.

$$Y = \mu + bi + g_j + e_{ijk}$$

Where: Y= the response variable corresponding to treatmenti^{th} measure on block j^{th}

b_i= the effect of i^{th} replication

g_j= the effect of j^{th}genotype and

e_{ijk} = the residual term

3.4.1. Wilted seedling percentage

Wilted seedlingpercentage was calculated from the cumulative number of dead over total number of seedlings (dead plus healthy) for a total period of six months (based on external symptoms).

Wilted seedling Percentage = $\frac{\textbf{cumulative number of dead seedlings}}{\textbf{total number of seedlings (dead plus healthy)}}$ x 100

Wilted seedling percentage at 6 months after inoculation and number of yellow leaves per seedling, number of defoliated leaves per seedlingand all growth characters at four months post inoculation data were used for analysis. Additionally,estimation of variance components (phenotypic, genotypic and environmental variance), broad sense heritability, genetic advance as percent of mean and correlation among the characters were computed in similar manner.Most results of genetic variance components, heritability and other traits software outputs were counter checked using excel spreadsheets and confirmed the output.

3.4.2. Estimationof variance components

The phenotypic, genotypic and environmental variances were estimated based on the method suggested by Singh and Chaudhury (1985).

$$\text{Environmental variance}(\sigma^2\mathbf{e}) = \mathbf{mse}$$

$$\text{Genotypic variance } (\sigma^2 g) = \frac{\mathbf{Msg - Mse}}{\mathbf{r}}$$

$$\text{Phenotypic variance } (\sigma^2 p) = \sigma^2 g + \sigma^2 e = (\ \boldsymbol{\delta^2 A + \delta^2 D\ +\ \delta^2 I})\ +\ \boldsymbol{\delta^2 e}$$

Where, r = number replication

MSg, MSe = mean square due to genotypes andmean square of error, respectively

$\sigma^2 A$, $\sigma^2 D$, $\sigma^2 I$ = additive variance, dominant variance and epistatic, respectively

3.4.3. Heritability

Broad sense heritability for CWD and seedling growth characters was computed using the formula suggested by (Allard, 1999) as:

$$h^2_B = \frac{\sigma^2 \mathbf{g}}{\sigma^2 \mathbf{p}} * \mathbf{100}$$

Where, h^2_B= broad senseheritability

$\sigma^2 g$ = genotypic variance and

σ^2P = phenotypic variance

3.4.4. Genetic advance (GA)

The genetic advance expected under selection assuming the selection intensity of the superior 5% of the plants for a character was estimated according toAllard (1999) method as follow;

$$\mathbf{GA = K\ \sigma p\ h^2_B}$$

The Genetic advance as % of mean (GAM) was computed as:

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

Where, h^2_B = heritability in the broad sense

K = the selection differential and

σp=phenotypic standard deviation on mean basis

GA = genetic advance under selection and

$\bar{X}$ = mean of the population in which selection was employed

3.4.5. Heterosis

Heterosis of different traits was estimated following the formulae suggested by Falconer and Mackay (1996) as follows:

Relative heterosis (mid parent heterosis) = $\left[\frac{F1-MP}{MP}\right] * 100$

Heterobeltisois (better parent heterosis) = $\left[\frac{F1-BP}{BP}\right] * 100$

Standard heterosis (susceptible check heterosis) = $\left[\frac{F1-SC}{SC}\right] * 100$

Susceptible parent heterosis = $\left[\frac{F1-SP}{SP}\right] * 100$

Where, F1 is the mean value of the hybrid

MP denotes the mean value of the two parents involved in producing the F1, and

BP denotes the better parent mean value for CWD and growth trait

The standard error of the difference for heterosis was calculated as follows:

SE (m) for MP = $\pm \sqrt{\frac{3Me}{2r}}$

SE (m) for BP, SP and SC = $\pm \sqrt{\frac{2Me}{r}}$

SE (d) for MP = SE (m) for MP x t at error degree of freedom

SE (d) for BP = SE (m) for BP x t at error degree of freedom

SE (d) for BP = SE (m) for SP x t at error degree of freedom

SE (d) for BP = SE (m) for SC x t at error degree of freedom

Test of significance for heterosis was done by comparing (F1-MP) with SE (d) for mid parent, (F1 -BP) with SE (d) for better parent heterosis, (F1 -SP) with SE (d) for susceptible parent heterosis and (F1 -SC) with SE (d) for susceptible check heterosis.

Where, SE (m) is standard error of the mean, SE (d) is standard error of the difference, Me is error mean square and r is the number of replications.

The minimum values were considered as the better parent in the case of wilted seedling percentage and number of defoliated leaves per seedling.

3.4.6. Combining ability

Analysis of variancefor combining ability

F1 generations and selfed parental lines CWD and growth characters data were subjected to combining ability analysis using both plant breeding tools (PBTools) software version 1.4 (PBTools, 2014) and SAS program version 9.2 (SAS, 2008) to cross-checked the result (Table 2). Combining ability analysis computed using the following mathematical model;

$$Y_{ij} = \mu + g_i + g_j + s_{ij} + \frac{1}{bc}\sum_{k\,l}^{\Sigma\Sigma} e_{ijkl} \begin{cases} i, j = 1,2,3\ldots,n \\ k = 1,2,3\ldots,b \\ l = 1,2,3\ldots,c \end{cases}$$

Where, Y_{ij} = the value of a character measured on cross of i^{th} and j^{th} parents, μ = overall mean

g_i, g_j = the general combing ability effect of the i^{th} and j^{th} parents, respectively

S_{ij} = the specific combing ability effect of the cross i x j

$\frac{1}{bc}\sum_{k\,l}^{\Sigma\Sigma} e_{ijkl}$ = the mean error effect of the $ijkl^{th}$ observation

n, b and c = number of parents, blocks and sampled plants, respectively

Table 2.Analysis of variance for combining ability based on Griffing (1956) methods 2 and model I

Source	Degree of freedom	Sumof square	Mean square	EMS (expected of mean square) Fixed Model
GCA	n-1	Sg	Mg	$\sigma^2_e + \frac{n+2}{(n-1)} \Sigma\, gi^2$
SCA	n(n-1)/2	Ss	Ms	$\sigma^2_e + \frac{2}{n(n-1)} \Sigma\Sigma\, sij^2$
Error	(r-1) ((n(n+1)/2)-1)	Se	Me’	σ^2_e

Source: Griffing’s (1956)

GCA= general combining ability, Mg, Ms and Me’ are mean squares of GCA, SCA and error, respectively, n= number of parents, SCA = specific combining ability, Se = error sum square, Sg = GCA sum square, Ss= SCA sum square, σ2e= error variance, gi and sijGCA effects of parent $i^{th}$and SCA effect of the cross i x j, respectively

GCA and SCAsum squares, mean squares, effectsof genotypes andgenetic variance components were calculated using Griffing (1956) formula:

$$Sg = \frac{1}{n+2}[\sum(Yi. + Yii)^2 - \frac{4}{n}Y..^2)]$$

$$Ss = \sum\sum Yij^2 - \frac{1}{n+2}\sum(Yi. + Yii)^2 + \frac{2}{(n+1)(n+2)}Y..^2)$$

Se = error sum of squares from all genotypes

Where, Sg, Ss= sum of squares due to GCA and SCA, respectively.

Yi. = total of the crosses involving the i^{th} parent

Y.. = grand total for all observations

Y_{ii}, Y_{ij} = parent and cross mean value in diallel, respectively

n = number of parents

Then, GCA and SCA mean squares were calculated as follows:

$$Mg = \frac{Sg}{n-1}$$

$$Ms = \frac{Ss}{n(n-1)/2}$$

$$M'e = \frac{MSe}{bc}$$

Tests for significance of mean squares were made using F-test at(n-1) and (r-1) [(n (n+1) / 2)-1] for GCA andn (n-1) / 2 and (r-1) [(n (n+1) / 2)-1] for SCA degree of freedom as follows:

$$Fg = \frac{Mg}{Me'}$$

$$Fs = \frac{Ms}{Me'}$$

Where, Mg, Ms = GCA mean square, SCA mean square, respectively

M'e, Mse = error mean square of combining ability and error mean square of ANOVA from all genotypes (parents and crosses), respectively

n, b,c = number of parents, blocks and sampled plants in each observation

Fg, Fs = Calculated F value of GCA and SCA, respectively

Estimates of GCA and SCA effects

The general combining ability effect (g_i) andspecific combining ability effect (s_{ij}) were estimated using Griffing (1956) equation:

$$g_i=\frac{1}{n+2}[Yi. +Yii- \frac{2}{n}Y..]$$

$$Sij = Yij - \frac{1}{n+2}((Yi. +Yii + Y.j + Yjj)) + \frac{2}{(n+1)(n+2)}Y..$$

Where,Yi., Y.j = means of the i^{th} and j^{th} parents, respectively

Y.. = grand mean, n = number of parent lines

Standard errors were calculated using the formula:

$$SE(g_i)= [\frac{n-1}{n(n+2)}\delta_e^2]^{1/2}$$

$$SE(g_i-g_j)= [\frac{2}{n+2}\delta_e^2]^{1/2} \qquad (i \neq j)$$

$$SE(S_{ii})= [\frac{n(n-1)}{(n+1)(n-2)}\delta_e^2]^{1/2}$$

$$SE(S_{ij})= [\frac{n^2+n+2}{(n+1)(n+2)}\delta_e^2]^{1/2} \qquad (i \neq j)$$

$$SE(S_{ii}- S_{jj}) = [\frac{2(n-2)}{n+2}\delta_e^2]^{1/2} \qquad (i \neq j)$$

$$SE(S_{ij-}S_{ik}) = \left[\frac{2(n+1)}{n+2}\delta_e^2\right]^{1/2} \qquad (i \neq j, k; j \neq k)$$

$$SE(S_{ij-}S_{kl}) = \left[\frac{2n}{n+2}\delta_e^2\right]^{1/2} \qquad (i \neq j, k, l; j \neq k, l; k \neq l)$$

Where, δe, SE =error variance and standard error, respectively

SE (g_i)= standard error forthe i^{th}parent gcaeffect

SE (g_i-g_j) = standard error for the i^{th} and j^{th} parents gca difference

SE (S_{ii}) = standard error of the i^{th} selfed parent sca effect

SE (S_{ij}) = standard error of the i^{th} and j^{th} parent sca effects

SE (S_{ii}-S_{jj}) = standard error of the i^{th} and j^{th} parent sca effects difference

SE (S_{ij}-S_{ik}) = standard error of two sca effects difference having one parent in common

SE (S_{ij}-S_{kl}) = standard error of the differences between two sca effects without having common parent

The significance of GCA and SCA effects were tested by comparing the calculated GCA and SCA effectswith thetabular value(5% and 1% probability level of significance) at error degree of freedom multiplied by standard errors.

Variance components and gene actions

The estimates of genetic components computed as the expectation for model I bySingh and Chaudhury (1985) formula:

Component due to GCA

$$\frac{\mathbf{1}}{\mathbf{n-1}}\sum \mathbf{gi^2} = \frac{\mathbf{Mg - Me'}}{\mathbf{n+2}}$$

Component due to SCA

$$\frac{\mathbf{1}}{\mathbf{n(n-1)}}\sum\sum \mathbf{Sij^2} = \mathbf{Ms - Me'}$$

The relative size of variances due to GCA and SCA in progeny performance was estimated as;

$$\text{GCA to SCA ratio} = \frac{\frac{1}{n-1}\sum g_i^2}{\frac{1}{n(n-1)}\sum\sum s_{ij}^2} = \frac{1}{n+2}\left[\frac{Mg - m'e}{Ms - M'e}\right]$$

3.4.7. Estimates of correlation among characters

The associations between traits were calculated asSingh and Chaudhary (1985):

$$\mathbf{r_p} = \frac{\mathbf{pcovx.y}}{\sqrt{\delta^2\mathbf{px}.\delta^2\mathbf{py}}}$$

$$\mathbf{r_g} = \frac{g\mathbf{covxy}}{\sqrt{\delta^2\mathbf{gx}.\delta^2\mathbf{gy}}}$$

Where,rp and rgare phenotypic and genotypic correlation coefficients, respectively

pcovx.y and gcovx.yare phenotypic and genotypic covariance between variables x and y, respectively

δ^2pxand δ^2gxare phenotypic and genotypic variances for variable x, and

δ^2pyand δ^2gyare phenotypic and genotypic variances for the variable y, respectively

The significance of phenotypic andgenotypiccorrelation coefficient was testedat 5 % and 1 % significance level by comparing the computed '**r**′ value to the tabular 'r′value at n-2degree of freedom.

4. RESULTS AND DISCUSSION

Eight selfed parents, 28 F1 crosses and one susceptible check were used to measure four CWD and seven seedling morphological characters for further genetic analysis and interpretation.

4.1. Analysis of Variance

The analysis of variance mean squares were highly significant ($p<0.01$) for wilted seedling percentage, incubation period, seedling height, average internode length, nodes number, petiole length, leaf area andleavesnumber when compared F1 crosses with parents (Appendix Table 1).Coffee seedling stemdiameter (girth)showed significant ($p<0.05$)differences between F1 crosses and their parents;but it revealed non-significant without its parents.When compared F1 crosses versus parents, the number of defoliated and yellowleaves exhibited highlysignificant at $p<0.01$ andnon-significant differences, respectively.While,F1 crosses alone, both number of defoliated and yellow leaves per seedlingrevealed significant differences ($p<0.05$). All disease and growth characters (except stem diameter and number of yellow leaves) showed significant differences among parental lines. Theseresults confirmed the existence of genetic diversity between the parental lines and 28F1 crosses formost CWDand growth characters and meetsthe prerequisites fordetail genetic analysis given by Griffing(1956).

Screening of different coffee genotypes by different investigators also reported the existence of Arabica coffee genetic variation to CWD; both at seedling inoculation test and mature coffee plants (Van der Graaff and Pieters, 1978; Merdassa, 1986; Girma and Hindorf, 2001; Girma, 2004; Girma *et al*., 2005 Arega, 2006; Chala, *et al*., 2012; Sihen *et al*., 2012; Demelash, 2013; Kifle, *et al*., 2015; Demelash and Kifle, 2015). Alike, Musoli, *et al*. (2013) also found highly significant genetic differences for CWD severity on Robusta coffee. The present study supported those investigator findings and confirmed that there is a perceptible Arabica coffee genetic variation to the disease.

4.1. Mean Performance of Parents and F1Crosses

4.1.1.Coffee WiltDisease characters

The mean performance of CWDparameters, stem and leaf characters of the crosses, parental lines and susceptible check are summarized in Table 3. Mean wilted seedling percentage ranged from25.1% for resistant or tolerant parent P2 to 91.4% for susceptible parent P3;and from 20.6% for resistant cross P7 x P8to 90.7% for susceptible crossesP1 x P6 andP1 x P8. From the result, the crosses appeared relatively a wide range of percentage compared to the parents. But, only one cross recorded low wilted seedling percentage than resistant parents.From the mean performance of parents, P2 (971)exhibited lowwilted seedlingpercentage or higher survival percentage (relatively CWD resistant), followed by ParentsP5(79233), P7 (974) and P8 (370).In contrast, parental lines P3 (91.4%), P6 (87.5%) and P1 (86.7%) showedhighwilted seedling percentage; it suggests that these parents were highly susceptible under greenhousecondition.

Moreover, crosses P7 x P8 (20.6%), P2 x P7 (26.2%), P4 x P8 (28.2%),P2 x P8 (28.5%), P2 x P5 (29.8%),P5 x P8 (37.0%), P4 x P7 (39.6%)and P5 x P7 (42.7%) exhibited relatively lowmean wilted seedling percentage.In contrary, cross P1 x P6 and P1 x P8 (equally) recorded the highest percentage (90.7%), followed by P1 x P3, P1 x P5 and P3 x P6. This result indicates that therewere clear genetic variations between parents and crosses for CWD resistance. Generally, mean resultshowed that when resistant crossed with resistant or moderately resistant parents, the progenies became relatively resistant or moderately resistant; while susceptible parents crossed with any CWD reaction groups (resistant, moderately resistant or susceptible parents), the progenies became susceptible. Therefore, susceptible genes might be dominant (partially or completely)over the resistant genes in the inheritance of CWD resistance.High mean wilted seedling percentage was observed in the crosses (65.0%) compared to parents(59.9%).It indicatedthat there was nooverallgenetic improvement in the favorable directionthrough crossing;even though some individual crosses showed lower wilted seedling percentage than the corresponding parents. Generally, neither parents nor crosses showed exemptto the disease.

Mean incubation period of parents and crosses showed significant differences. It elapsed from 89.7 to 133.0 days for parents and 96.3 to 143.0 days for F1 crosses. Parental lines P5, P8, P2, P7, P4, P3, P1 and P6 required 133.0, 126.7, 118.3, 115.3, 107.7, 91.7, 91.3, 89.7 days, respectively. P5 (133 days) required longest incubation period compared to other parental lines and found in the fourth rank from the total genotypes. The top three crosses that showed relatively extended incubation period were crosses P7 x P8 (143.0 days), P2 x P4 (137.7 days), and P4 x p8 (136.7days). Conversely, crosses P1 x P3 (96.3 days), P3 x P8 (97.0 days), P1 x P3 (98.3 days) and P1 x P2 (99.0 days) manifested early disease symptoms. Therefore, the significant differences of incubation period between genotypes showed that the existence of variable Arabica coffee genotypes to *Gibberella xylarioides* interaction. This might be due to the differences of host (coffee genotypes) defensive ability to the disease.

Number of defoliated leaves per seedlingalso showed significant differences;whilenumber of yellowleaves revealed non-significant differences between parents and F1 crosses. Parental lines P2, P8, P7 and P5 recorded small number;whileP6, P3 and P4 recorded manynumbers of defoliated leaves. When overall mean of the F1crosses compared with parents, the former recorded many number of defoliated leaves. The four top favorable crosses that recorded low number of defoliated leaves were P5 x P8, P2 x P8, P7 x P8 and P2 x P7; whereasP1 x P6, P1 x P3, P1 x P8 and P3 x P6 recorded many number. Therefore, P2, P8 and P7 probably inherited their favorable genes towards most of their cross combinations; while Parents P1, P3 and P6 might be contributed their unfavorable genes for high defoliated leaves towards their corresponding progenies.

In general, parents P2, P5, P7 and P8 manifested relatively low wilted seedling percentage, extended incubation period and minimumdefoliated leaves; expressed favorable effects for the resulting crosses. It probably the potential of the parents inherited their favorable genes to their progenies; while P1, P3 and P6 contributed unfavorable genes for these characters to their progenies; the reason why most of their progenies weak in performance.Similarly, Chala *et al*. (2012) reported that parents P2 and P7 exhibited low wilted seedling percentage and considered as CWD resistant.In contrary to the present finding, Demelash (2013)reported that parent P8 showed CWD resistant.In this study,genotypes with resistant reaction had longer incubation period; while susceptible reaction expressed early wilting symptom

development.The positive relationship ofCWD resistance with incubation period also reportedby Girma and Chala (2008) and Kifle *et al.*(2015).

4.1.2. Seedling stem characters

The result indicated that meanseedlingheight and stem diameter revealed significant differences, respectively. Mean height rangedfrom 10.3cm (P6) to 14.2cm (P8)for parental lines and 12.1cm (P5 x P7) to 15.2cm (P2 xP8)for F1 crosses (Table 3).For stem diameter, parent P2 recorded the widest(2.25mm); while P6recorded the thinnest (2.03 mm)compared to parents. However, crossesP1 xP4, P4 xP5, P4 x P6 and P4xP7measured the wider stem diameter (2.28 mm) compared to overall genotypes. Furthermore, it also wide enough for crosses P2x P5, P2 x P6, P4x P8, P2xP3 and P2x P8. On the other hand, Cross P6 X P7 (1.99mm)recorded the thinnest or the slimmest diameter, followed by Crosses P6xP8 (2.01 mm), P3xP8 (2.04 mm), P5xP6 (2.07 mm), P1 X P3 (2.08mm), P3xP7 (2.08 mm) and P1xP8 (2.09 mm). From the result, it seems like that P4 and P2 inherited the genes for wide stem diameter; while P6 and P3contributed genes for thin diameter to their crosses. Generally, F1 crossesmean seedling height and stem diameter were greater than the parents mean; but not statistically different. These results expected that during mitosis and meiosis cell division andcrossing over, some favorable genes transferred from parents to their progenies and certain improvement had been taking place for both characters.

Mean number of nodes ranged from 1.90 (P7) to 2.90 (P6) for parents and from1.97 (P1 x P7) to 3.00 (P3 x P5) for crosses.ParentsP6, P3and P5hadmany number of nodes in that decreasing order, whereas P7, P1 and P4 recordedfew numbers.Parentsmean number of nodes expressed greater than F1 crosses; while it reversed for average inter node length. All crosses mean average inter node length found within parents mean value (1.28 to 3.80 cm), except crosses P2 x P8 and P4 x P8 (greater than P8). As well, most crosses showed greater than the individual parentsmean and a significant differences were detected; when two parents combined together, it boosts the crosses average inter node length.So, the genetic variations between parents and the possibility to inherit their genes for average inter node length perhaps leads to significantF1 crosses improvement.

4.1.3. Seedling leaf characters

Most of the F1 crosses were higher mean petiole length when compared to theircorresponding parental lines and greaterthan the smallest parent P6 (0.41 cm). The greatest mean recorded in the cross P2 x P8 (0.58 cm).The overallmean leaf area of the genotypes showedbetween8.79 cm^2 to 18.98 $cm^2$and cross P4 x P8 recorded the maximum mean value.For parental lines, better leaf characters were noted in the order given that parents P2, P8 and P7 for petiole length; P8 and P2 for leaf area; and P5, P3 and P2 for number of leaves.Parent P6 for petiole length and leaf area, and P7 for number of leaves showed inferior for the characters. Surprisingly, the mean value of petiole length and number ofleaves ofparents and F1 crosses were equal, 0.49 cm and 6.35 mean numbers of leaves, respectively. Generally, crosses mean leaf area revealed greater than their parents. This indicates that through crossing some favorable genes, which is important to boost leaf area, possibly transferred from parents to their progenies.

Besides, F1 crosses and parental lines, the mean performance of the susceptible check (Geisha) expressed 81% wilted seedlings percentage and elapsed 85 days incubation period (shortest or earliest genotype to express symptom). It also showedhigher mean number of defoliated leaves, thinner stem diameter,shorter height, smaller inter node length, lesser mean leaf petiole length and leaf area compared to crosses and parents mean. However, number of nodes showed comparable result and in the case of number of leaves, it was the second, next to parent P5 and equal to the highest crosses(P3 x P5).

The significant differences in mean performance observed in disease and seedling growth characters are encouraged for some circumstance. The overall mean of the F1 crosses showed higher than their parents for all disease and most seedling growth characters.In addition, some F1 crosses recorded low mean wilted seedling percentage (desirable) than the resistant parents. However, the mean differences between parents and crosses were not statistically significant. These results evidently suggest that the possibility to further improve wilted seedling percentage (CWD resistance), incubation period, number of defoliated leaves and mostseedling growth characters through selection and hybridization. In this study, significant increase in seedling stem diameter of most CWD resistant parents and crosses (such as

crosses P4 x P7, P4 x P8 and P2 x P8) indicating that the possibility of improving water and food translocation in seedlings. Relatively high amount of water can be move through the stem to the upper parts of the seedling (leaves) and food from leaves to the root and stem parts. Hence, it showed that better survival than thinner or smaller stem diameter, which is susceptible to CWD. According to Agrios (2005), fussarium wilt infected plants leaves transpire more water than the roots and stem can transport and reduce growth and transpiration, resulting wilting symptoms. So, the reason why the present most seedling growth characters stunted when CWD infection enhanced.Therefore, consideringgrowth characters are important in the evaluation of Arabica coffee genotypes for CWD resistant.

Similar study in Japanese pines associate with resistance to Pine wilt nematode was reported with respect to basal diameter; a thicker diameter predicted to survive better and also trees can survive as long as there is a partial passage for xylem and phloem transport (Yamanobe, 2009; Kuroda, 1999). Siddiqui and Singh (2004) alsoconducted glass-house experiments to assess the effects of wilt fungus on the growth and transpiration of chickpea. Results showed that inoculation of *Fussarium oxysporum* reduce plant growth, transpiration and caused severe wilting. The presentmost seedling growth characters resultsarein agreement with the works of Mesfin (1982), Mesfin and Bayetta (1983), Bayetta (2001), and Wassu (2004).However,the analyses of variance for mean leaf area contradictto the work of last investigator. This differenceprobably due to the stage of coffee plants to measure characters, genotypes variation and environmental variation.

Table3.Mean performance of parents, F1 crosses and susceptible check for CWD and seedling growth characters

Genotypes	WS (%)	IP (Days)	NDL (no.)	SH (cm)	SSD (mm)	AINL (cm)	NN (no.)	PL (cm)	LA (cm^2)	NL (no.)
Parents										
P1	86.7a	91.3ij	1.96b-i	13.1f-n	2.17a-g	3.01g-l	2.27j-m	0.49d-j	10.79klm	6.18c-g
P2	25.1i	118.3de	0.78i	13.3c-l	2.25a-d	3.09e-k	2.57c-f	0.53b	16.24bcd	6.59a-f
P3	91.4a	91.7hij	2.69a-d	12.0o	2.20a-f	2.30nop	2.73b	0.49d-j	10.45lm	6.76a-d
P4	72.0a-e	107.7efg	2.18b-h	13.2e-m	2.24a-d	3.04f-l	2.25j-m	0.47ijk	13.36e-i	6.13d-g
P5	32.2hi	133.0abc	0.96hi	12.4l-o	2.16a-g	1.85pq	2.71bc	0.47ijk	10.28lm	7.16a
P6	87.5a	89.7j	3.58a	10.3p	2.03efg	1.28q	2.90a	0.41m	8.79m	6.31b-g
P7	35.2ghi	115.3def	0.93hi	13.2e-m	2.06d-g	3.11d-k	1.90p	0.50b-i	12.45g-l	5.14i
P8	49.0e-h	126.7bcd	0.80i	14.2a-e	2.13a-g	3.82abc	2.44f-i	0.52bcd	16.37bcd	6.50a-f
Mean	**59.9**	**109.2**	**1.73**	**12.7**	**2.15**	**2.69**	**2.47**	**0.49**	**12.34**	**6.35**
Crosses										
P1 x P2	81.3abc	99.0g-j	1.89b-i	13.4c-l	2.23a-d	3.28b-j	2.38h-k	0.49d-j	11.94i-l	5.73ghi
P1 x P3	89.3a	98.3g-j	2.91abc	13.7c-j	2.08c-g	3.62a-g	2.20lmn	0.48g-j	10.95klm	5.33hi
P1 x P4	74.7a-d	96.3g-j	2.20b-h	14.4abc	2.28ab	3.74a-d	2.30i-m	0.51b-h	13.54e-i	6.18c-g
P1 x P5	88.0a	102.0ghi	2.28a-g	14.8ab	2.21a-e	3.61a-g	2.32i-m	0.50b-i	13.22e-j	6.58a-f
P1 x P6	90.7a	101.7ghi	2.98ab	13.5c-k	2.13a-g	2.69j-o	2.67bcd	0.46jkl	11.16jkl	6.58a-f
P1 x P7	62.7b-f	108.0efg	1.16e-i	14.1b-f	2.19a-f	3.72a-e	1.97op	0.49e-j	13.69e-i	6.13d-g
P1 x P8	90.7a	100.0g-j	2.87abc	14.1b-f	2.09b-g	3.48b-h	2.33i-l	0.50b-i	14.20d-h	6.44b-f

Table 3. (Continued)

Crosses	WS (%)	IP (Days)	NDL (no.)	SH (cm)	SSD (mm)	AINL (cm)	NN (no.)	PL (cm)	LA (cm^2)	NL (no.)
P2 x P3	69.5a-e	117.3def	1.98b-i	13.0h-o	2.25a-d	2.77i-o	2.60b-e	0.51b-h	14.54d-g	6.85abc
P2 x P4	56.9d-g	137.7ab	1.98b-i	12.4k-o	2.20a-f	2.50k-o	2.63b-e	0.50b-i	16.06cd	6.80a-d
P2 x P5	29.8hi	120.0d	1.27e-i	13.0h-o	2.27abc	2.80i-o	2.63b-e	0.52bcd	13.76e-i	6.71a-e
P2 x P6	61.3c-f	118.0de	2.62a-d	13.4c-l	2.27abc	3.48a-h	2.30i-m	0.49d-j	16.03cd	6.58a-f
P2 x P7	26.2hi	123.0cd	0.93hi	13.5c-j	2.20a-f	2.95h-n	2.09no	0.49d-j	14.77def	5.73ghi
P2 x P8	28.5hi	123.0cd	0.89hi	15.2a	2.25a-d	4.07a	2.23k-n	0.58a	17.60abc	6.62a-f
P3 x P4	81.0abc	101.7ghi	2.42a-f	12.3l-o	2.15a-g	2.36m-p	2.20lmn	0.48f-j	12.03h-l	6.31b-g
P3 x P5	81.3abc	105.7fg	2.09b-i	13.3d-l	2.12a-g	2.28op	3.00a	0.53bc	12.05h-l	6.98ab
P3 x P6	86.7a	101.7ghi	2.82abc	12.8i-o	2.18a-f	2.40l-p	2.40g-j	0.51b-h	11.69i-l	6.93ab
P3 x P7	85.3ab	103.3gh	1.98b-i	14.0b-h	2.08c-g	3.20c-j	2.17mn	0.51b-h	13.40e-i	6.05fg
P3 x P8	86.6a	97.0g-j	2.47a-e	12.7j-o	2.04e-g	3.10d-k	2.29i-m	0.51b-h	13.32e-j	6.22c-g
P4 x P5	70.8a-e	116.0def	1.64c-i	14.1b-f	2.28ab	3.36b-i	2.33i-l	0.50b-i	15.23de	6.18c-g
P4 x P6	74.3a-d	101.0g-j	2.04b-i	14.3a-d	2.28ab	3.28b-j	2.57c-f	0.48f-j	13.43e-i	6.18c-g
P4 x P7	39.6f-i	132.3abc	1.40d-i	13.6c-j	2.28a	3.08e-k	2.50e-h	0.42m	18.30ab	6.40b-g
P4 x P8	28.2hi	136.7ab	1.11f-i	14.1b-f	2.27abc	3.87ab	2.33i-l	0.52bcd	18.98a	6.67a-e

Table 3.(Continued)

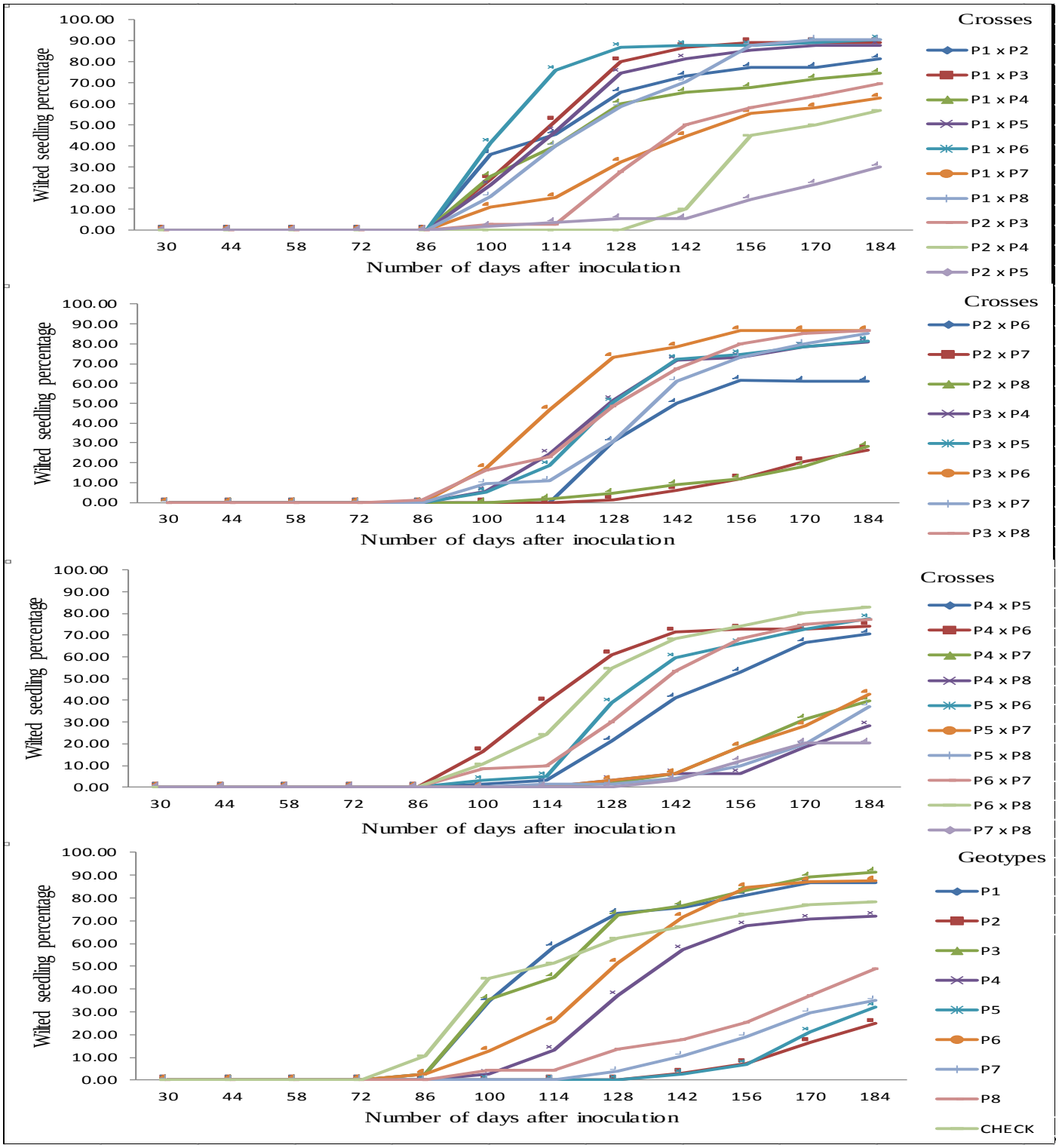
Crosses	WS (%)	IP (Days)	NDL (no.)	SH (cm)	SSD (mm)	AINL (cm)	NN (no.)	PL (cm)	LA (cm^2)	NL (no.)
P5 x P6	77.7a-d	108.0efg	1.82b-i	12.3l-o	2.07d-g	2.26op	2.53d-g	0.48f-j	11.91i-l	6.49a-f
P5 x P7	42.7f-i	123.0cd	1.02ghi	12.1on	2.14a-g	2.35nop	2.40g-j	0.43lm	11.02kl	5.95fgh
P5 x P8	37.0ghi	126.7bcd	0.84i	12.9h-o	2.14a-g	3.09e-k	2.36h-k	0.48f-j	12.68f-k	6.49a-f
P6 x P7	76.9a-d	108.0efg	2.40a-f	12.2mno	1.99g	2.40l-p	2.23k-n	0.44klm	11.86i-l	6.04efg
P6 x P8	82.9abc	107.3efg	2.00b-i	13.0h-o	2.01fg	3.06f-k	2.20lmn	0.52bcd	12.30h-l	6.04efg
P7 x P8	20.6i	143.0a	0.89hi	13.9b-i	2.14a-g	3.66a-f	2.43f-i	0.52bcd	17.93abc	6.62a-f
Mean	**65.0**	**112.7**	**1.89**	**13.4**	**2.17**	**3.09**	**2.38**	**0.49**	**13.84**	**6.35**
Check	78.33	85.00	2.00	10.07	2.07	1.62	2.34	0.48	12.32	6.98
LSD (0.05)	[a]23.21(17.65)	11.72	1.30	1.08	0.18	0.64	0.15	0.03	2.16	0.70
	[b]**23.29 (17.69)**	**11.82**	**1.32**	**1.08**	**0.19**	**0.65**	**0.15**	**0.03**	**2.18**	**0.69**
	[c]24.78 (18.38**)**	11.64	1.42	1.09	0.20	0.66	0.16	0.04	2.23	0.73
CV (%)	[a']22.18 (19.73)	6.48	43.07	5.02	5.24	13.38	3.94	4.25	9.83	6.74
	[b']**22.38 (19.84)**	**6.49**	**43.64**	**5.01**	**5.30**	**13.25**	**3.95**	**4.25**	**9.93**	**6.65**
	[c']23.27 (20.36)	6.31	45.95	4.97	5.52	13.15	4.01	4.57	9.84	7.05

P1 = 75227, P2 = 971, P3 = 74110, P4 =8136, P5 = 79233, P6 = 74144A, P7 = 974 and P8 = 370, a= least significant difference (LSD) and a'= coefficient of variation (CV %) of parents, F1 crosses and susceptible checks, b= LSD and b'= CV% of parents and F1 crosses and c= LSD and c'= Cv% of F1 crosses only, cm= cent meter, cm^2= cent meter square, mm= mill meter, no. = number AINL= average inter node length, IP = incubation period, LA= leaf area, NL= number of leaves, NDL= number of defoliated leaves per seedling, NN= number of nodes,NYL= number of yellow leaves per seedling, PL= petiole length, SH= seedling height, SSD= seedling stem diameter, WS%= Wilted coffee seedlings percentage

All growth characters measured at 4 months and disease severity data (wilted seedling percentage) recordedat 6 months after artificial inoculation used for statistical analysis.CV and LSD value in brackets is arcsine transformed value of wilted seedlings percentage

4.2. Wilted Seedling Percentage Progress on Time Trend

Mean wilted seedling percentage progress on 37 Arabica coffee genotypes (28 F1 crosses, 8 parental lines and one check) at greenhouse revealed variable responses to CWD infection (Figure 1). Different coffee genotypes seedlings were infected at varying intensities when disease assessment started and it progressed at varying rates until the final disease severity time (6 months after inoculation). Most of the genotypes showed a constant trend of disease progress after five months of data assessment. This figure showed that Arabica coffee genotypes had variable levels of resistance to CWD infection and progressed at varying rate after infection.Coffee parental lines P2, P5 and P7 and crosses P7xP8, P2 x P7, P4xP8, P2xP5, P2x P8, P4xP7, P5xP8and P5xP7manifestedlate disease infection and recorded low percent of disease progress for 6 months disease assessment period (12 times recorded within 14 days interval).The above mentioned parents and crosses appeared to be exempt to the disease until 4 months when high numbers of coffee seedlings started being wilting. Most genotypes showedhigh and increasing disease severity (wilted seedling percentage) starting 3 to 4 months after inoculation. This may be due to well-established by the pathogen and production of micro and macro conidia, mycelium and spores to colonize the host tissue and hinder the normal physiological processes. Agrios (2005) also explained it in detailed for tomato fussarium wilt disease.Therefore, those parents and crosses (mentioned above) that showed late symptoms expression and low wilted seedling percentage is important for further crossing or breeding programs in order to manage CWD impact through wilt resistance varietydevelopment.



P1 = 75227, P2 = 971, P3 = 74110, P4 =8136, P5 = 79233, P6 = 74144A, P7 = 974 and P8 = 370

Figure 1. Wilted seedling percentage progress on time trend for coffee parental lines and their F1 cross

4.3. Heterosis

Percentage of heterosis over the better parent (BPH), mid parents (MPH), susceptible parents (SPH) and susceptible check (SCH) were estimated for three CWD and seven growth characters (Tables4, 5and6).

4.3.1. CWD characters

Percentage of BPH, MPH, SPH and SCHfor wilted seedling percentage, number of defoliated leaves and incubation period are presented in Table 4. BPH ranged from -42.49% to 224.17% for wilted seedling percentage with +66.70% overall mean value. It revealed that 14 crosses expressed positive and significant undesirable heterosis.Although, no cross showed negatively significant BPH, crosses P4 x P8 and P7 x P8 manifested negative desirable effects. The maximum positive observed in cross P1 x P2.The top three most undesirable crosses were P1 x P2, P2 x P3 and P1 x P5. Heterosis for negative traits like disease, smaller values (negative values) are desirable for resistance. However, about 50%of the crosses exhibited positive and significant BPH for wilted seedling percentage; probably due to the result of lacking dominance of resistance or it might be present, but masked by the harmful effect of susceptible genes in controlling the inheritance of CWD resistance.

MPHfor wilted seedling percentageranged from -53.42% (P4 x P8) to + 48.08% (P1 x P5). Out of 10 negative heterosis, only two crosses (P4 x P8 andP7 x P8)showed negative and significant ($p<0.01$and / or $p<0.05$); while fourcrosses (P1 x P5, P1 x P2, P3 x P7 and P1 x P8)exhibitedpositively significant. For SPHand SCHranged from -60.86% (P4 x P8) to+21.31% (P5 x P7) and from -73.52% (P1 x P6 and P1x P8) to+15.75%(P7 x P8), respectively.Five crosses showed negatively significant and P4 x P8, P7 x P8 and P4 x P7 were the maximum desirable crosses for SPH. While, eight crosses observed negatively significant and P7x P8, P2x P7 and P4x P8 were the top three crossesthat expressedminimum heterosis (favorable effect)for SCH.This result suggests that crosses that observed only the negative mid parent, susceptible parent and susceptible check heterosis were in a desirable direction to CWD resistance.

The value of heterosis fornumber of defoliated leaves ranged from -6.13%to+258.75% for BPH andfrom -28.97% to +108.22% for MPH. In both cases, cross P4 x P6 and P1 x P8 expressed the minimum and maximum heterosis, respectively. It also ranged from -49.16% (P4 x P8) to +46.51% (P1 x P8) for SPH and from -57.84% (P5 x P8) to +49.00% (P1 x P6) for SCH. All crosses (except cross P4 x P6) showed positive BPH (unfavorable effects) and four crosses exhibited positivelysignificant. MPH of all crosses (except cross P1 x P8, positively significant)expressednon-significant; ninecrossesmanifestednegative and 19 crosses were positive. On the other hand, 20 crosses expressed negative,but only three crosses detected significant for SPH. Furthermore, estimateof SCH revealed non-significant; 15 crosses negative (desirable direction), while 13 crosses expressed positive(undesirable direction).Generally, crossP4 x P6 showed desirable effects; while crosses P1 x P8, P2 x P6 and P3 x P8 exhibited undesirablecrossesfor BPH. Moreover, crosses P4 x P6, P4 x P8 and P5 x P6 were the uppermost favorable crosses than the mid parent; whereas crosses P1 x P6, P1 x P5 and P2 x P5 were the most three inferior crosses.Generally, about 96 % of the crosses for BPH and one third of the crosses for MPH expressed undesirable effects for number of defoliated leaves. This inferiority of crosses for the character probably due to gene(s) responsible for attached leaves to the stem firmly wasinefficient to persist and protect *Gibberella xylarioides*virulence genes duringhost pathogen interaction.

For incubation period,BPH ranged from -23.42 % (P3 x P8) to +16.34 % (P2 x P4) with -4.92% overall mean. Crosses P2 x P4, P4 x P7 and P7 x P8 the only crosses that exhibited positively significant BPH.But,10 crosses showed negatively significant heterosis.Positive and significant MPH observed in eight crosses(desirable direction), while only cross P3 x P8 exhibited negatively significant heterosis. Crosses P2 x P4, P4 x P7, P7 x P8and P4 x P8 were the supreme crosses, in that order of desirable magnitude. This result indicates that29% of the crossesrequired significantly longer incubation period than their average parents.Moreover, all crosses exhibited Positive SPH and SCH; 13 and 27 crosses showed significant heterosis, respectively.Crosses P5 x P8 and P2 x P6 for SPH andcrosses P1 x P4and P7 x P8 for SCH showed minimum and maximum heterosis for incubation period, respectively.

In summary,most crosses showed undesirable and insignificantBPH and MPH for wilted seedling parentage and number of defoliated leaves (no cross exhibited significant desirable heterosis). Moreover,no cross showed significant BPH and only two crosses expressed negatively significant MPH for wilted seedling percentage (desirable direction). Mostly, when resistant crossed with susceptible parentsthe resulting offspring (F1 generations) mean foundbetween the two parents; towards the susceptible parent. This result suggests that probably partial to complete dominance of susceptible genes over the resistancegenes controlling the inheritance of CWD resistance. Alternatively, resistance genes might be lacking dominance. However, for incubation period three and eight crosses observed significant positive (desirable direction) BPH and MPH, respectively. When resistant parents crossed with susceptible parents, about one fourth of the crosses showed significant MPH. Some crosses mean also expressed longer period than any one of the parents. Therefore, probably the existence of partial to over dominance for incubation period in favorable direction. For number of defoliated leaves dominance was possibly lacking in desirable direction or it might be present, but interacting in an unfavorable direction. Otherwise, it perhaps the existence of the masked effects of epistasisgenes (alleles).The presence or absence of dominance gene further described and discussed in combining ability session.

As described earlier, relatively small or negative MPH (favorable effect)was detected for crosses that had less mean wilted seedling percentage. Conversely, crosses that expressedheterosis in favorable direction are not always advantageous.Because, some crosses,such asP3 x P6 for wilted seedling percentage and incubation period observed favorable BPH and MPH, but these crosses mean value showed susceptibility and shorter incubation period. Unexpectedly, in most crosses the susceptible parents crossed with the resistant parents, the resulting MPH had more positive than susceptible parent crossed with susceptible or moderately resistant parents. For instance, higher MPH exploited when parent P1 (susceptible parent) crossed with P2 (resistant parent)than P1 crossed with P3 or P6 (susceptible parents). This result was due to higheraverage mean of the two susceptible parents than the average mean of the susceptible and resistant parents. As well as, whenresistant and susceptible parents used in heterosis calculation,the mid parents mean value became lowered; whileMPH increased in reverse.

In general, heterosis was little(not appreciable) for CWD resistanceimprovement and it was lacking to reduce the number of defoliated leaves,in the present Arabica coffee genotypes. Furthermore, in most crosses CWD resistance was improved by crossing resistant parents with resistant and moderately resistant parents. Therefore, the use of heterosis breeding may berarely essential and if it is necessary both parents should be wilt resistant or moderately resistant; while selection could be an effective method for improvement.For incubation period, some appreciable improvement through hybrid production takes place beyond improvingusing selection. Likewise, Patel and Pathak (2011) were studied on the genetics of resistance to wilt in castor crosses. They stated that heterosis breeding with choice of superior parents would be advantageous for enhancing wilt resistance in Castor along with yield. However, for developing wilt resistant hybrids both the parents should be wilt resistant. The present finding showed some similarity to Mesfin (1982), he studied heterosis over the mid parent and susceptible parent for CBD in Arabica coffee and reported that +2% to +51% and -15.5% to +29.5% percent susceptibilities of the hydrids, respectively. The hybrids resistance crossed susceptible were significantly susceptible to their mid parent values.Bayetta (2001) based on F2 generation in Arabica coffee also conclude that CBD resistance was recessive with susceptibility showing partial to complete dominance. Furthermore, he stated that none of the F2 crosses exhibited better or equal resistance to the resistant or intermediate parents.

Table4. Estimateof heterosis percentage for wilted seedling percentage and incubation period characters

Crosses	WS%				NDL				IP			
	BPH	MPH	SPH	SCH	BPH	MPH	SPH	SCH	BPH	MPH	SPH	SCH
P1 x P2	224.17**	45.55*	-6.15	3.83	142.31	38.12	-3.41	-5.50	-16.34**	-5.56	8.40	16.47*
P1 x P3	3.08	0.33	-2.28	14.05	48.72	25.25	8.18	45.50	7.27	7.47	7.67	15.69*
P1 x P4	3.70	-5.88	-13.85	-4.68	12.27	6.29	0.92	9.83	-10.53	-3.18	5.48	13.33
P1 x P5	173.37**	48.08**	1.54	12.35	138.32	56.52	16.35	14.00	-23.31**	-9.06	11.68	20.00**
P1 x P6	4.62	4.1	3.59	15.75	52.30	7.71	-16.68	49.00	11.31	12.34*	13.38*	19.61**
P1 x P7	77.92*	2.83	-27.69*	-20.00	23.94	-19.95	-41.05	-42.17	-6.36	4.52	18.25**	27.06**
P1 x P8	85.03**	33.66*	4.62	15.75	258.75**	108.22*	46.51	43.50	-21.05**	-8.26	9.49	17.65*
P2 x P3	176.78**	19.21	-24.04	-11.34	153.42	13.93	-26.52	-1.17	-0.84	11.75*	28.00**	38.04**
P2 x P4	126.96**	17.3	-20.91	-27.3	153.42	33.71	-9.19	-1.17	16.34**	21.83**	27.86**	61.96**
P2 x P5	18.94	4.19	-7.3	-61.9**	62.82	46.25	32.75	-36.50	-9.77*	-4.51	1.41	41.18**
P2 x P6	144.20**	8.82	-29.99*	-21.78	236.32**	20.43	-26.66	31.17	-0.28	13.46**	31.60**	38.82**
P2 x P7	4.55	-13.01	-25.52	-66.51**	19.65	8.95	0.00	-53.34	3.95	5.28	6.65	44.71**
P2 x P8	13.47	-23.15	-41.9	-63.65**	13.68	12.24	10.84	-55.67	-2.89	0.41	3.94	44.71**
P3 x P4	12.5	-0.87	-11.4	3.41	11.33	-0.41	-9.91	21.17	-5.57	2.01	10.91	19.61**
P3 x P5	152.66**	31.60	-11.03	3.83	118.46	14.62	-22.30	4.50	-20.55**	-5.93	15.27*	24.31**
P3 x P6	-0.98	-3.13	-5.2	10.64	4.83	-10.00	-21.16	41.00	10.91	12.13*	13.38*	19.61**

Table 4.(Continued)

Crosses	WS%				NDL				IP			
	BPH	MPH	SPH	SCH	BPH	MPH	SPH	SCH	BPH	MPH	SPH	SCH
P3 x P7	142.27**	34.76*	-6.66	8.94	111.80	9.11	-26.52	-1.17	-10.40*	-0.16	12.72	21.57**
P3 x P8	76.64**	23.28	-5.32	10.50	208.34*	41.36	-8.30	23.34	-23.42**	-11.15*	5.81	14.12*
P4 x P5	119.79**	35.81	-1.74	-9.68	71.77	4.89	-24.62	-17.84	-12.78**	-3.60	7.74	36.47**
P4 x P6	3.16	-6.87	-15.13	-5.17	-6.13	-28.97	-42.96*	2.16	-6.19	2.36	12.64	18.82**
P4 x P7	12.36	-26.18	-45.04**	-49.48**	50.01	-9.97	-35.83	-30.00	14.74**	18.68**	22.91**	55.69**
P4 x P8	-42.49	-53.42**	-60.86**	-64.02**	38.75	-25.42	-49.16	-44.50	7.89	16.64**	26.93**	60.78**
P5 x P6	141.26**	29.75	-11.26	-0.85	89.93	-19.71	-49.12**	-9.00	-18.80**	-2.99	20.45**	27.06**
P5 x P7	32.73	26.76	21.31	-45.45**	9.64	8.29	6.94	-48.84	-7.52	-0.94	6.65	44.71**
P5 x P8	14.99	-8.82	-24.46	-52.74**	5.41	-3.99	-11.81	-57.84	-4.76	-2.44	0.00	49.02**
P6 x P7	118.45**	25.37	-12.08	-1.77	157.15*	6.43	-32.96	20.00	-6.36	5.37	20.44**	27.06**
P6 x P8	69.21**	21.46	-5.27	5.85	150.00	-8.61	-44.13*	0.00	-15.26**	-0.77	19.70**	26.27**
P7 x P8	-41.64	-51.19*	-58.05*	-73.76**	11.25	2.69	-4.64	-55.50	12.89**	18.18**	23.99**	68.24**
mean	**66.70**	**7.87**	**-15.79**	**-16.97**	**83.87**	**12.07**	**-13.73**	**-5.54**	**-4.92**	**3.35**	**14.05**	**32.59**
SE(±)	**11.68**	**10.11**	**11.68**	**11.68**	**0.66**	**0.57**	**0.66**	**0.66**	**5.93**	**5.13**	**5.93**	**5.93**

P1 = 75227, P2 = 971, P3 = 74110, P4 =8136, P5 = 79233, P6 = 74144A, P7 = 974 and P8 = 370

Note: Values without asterisk (*) are non-significant; *, ** = significant at 5 % and 1% significant level, SE= standard error,BPH=better parent heterosis, MPH= mid parent heterosis, SCH=susceptible check heterosis, SPH= susceptible parent heterosis

IP = incubation period (days), NDL = number of defoliated leaves, WS%= Wilted coffee seedlings percentage,

4.3.2. Seedling growth characters

The expressions of BPHand MPH for seven growth characters are presented in Table5and 6, respectively. F1 crosses exhibited negative mean BPH for all seedling growth characters (except height and leaf area); ranged from -10.37% for number of nodes to +0.73% for seedling height. The amount of heterosis inindividual characters ranged from -10.09% (P3 x P8) to +13.46% (P1 x P5) for seedling height and -7.42% (P3 x P8) to +1.93% (P4 x P7) for stem diameter.It also ranged from -24.62% (P5 x P7) to +23.27% (P1 x P4) for average inter node length; -24.14% (P6 x P8) to+11.11% (P4 x P7) for number of nodes; -14.77% (P4 x P7) to +10.06% (P2 x P8) for petiole length; -26.46% (P1 x P2)to +37.02% (P4 x P7) for leaf area; and -21.07% (P1 x P3) to +4.35% (P4 x P7) for number of leaves.The top three crosses that showed positive BPH werecrossesP1 x P5,P1 x P4 and P4 x P6for seedling height;crosses P4 x P7, P1 x P4 and P1 x P5for stem diameter; crosses P1 x P4, P5 x P6 and P1 x P3 for average inter node length and crossP4 x P7, P3 x P5 and P2 x P4for number of nodes. Furthermore, crossesP2 x P8, P3 x P5 and P4 x P5 for petiole length; P4 x P7, P1 x P5 and P4 x P8 for leaf area and P4 x P7, P1 x P6 and P2 x P4 for number of leaves exhibited maximum BPH.Three crosses for seedling height and leaf area; two crosses for number of nodes and petiole length; and one cross for average internode length showed positively significant heterosis. Alternatively, 3, 6, 19, 7, 5 and 6 crosses showed negatively significant BPH for height, average inter node length, number of nodes, petiole length, leaf area and number of leaves, respectively. However, no F1 hybrids showed positive and significant BPH for stem diameter and number of leaves.

Estimate of MPH for four stem and three leaf characters are described in Table 6.In reverse to BPH, more than 15 crosses manifested positive MPH for all growth characters, except number of nodes (only seven crosses observed positive value). Its percentage ranged from -6.34% (P2 x P4) to +21.69% (P4 x P6) for seedling height; -5.86% (P3 x P8) to +6.72% ((P4 x P6) for stem diameter; -18.28% (P2 x P4)to +59.39% (P2 x P6) for average inter node length; and -17.55% (P6 x P8) to +20.48% (P4 x P7) for number of nodes. For leaf characters, it also rangedfrom -12.41% (P4 x P7) to +12.92% (P3 x P6) for petiole length; -11.64% (P1 x P2) to +41.85% (P4 x P7) for leaf area; and -17.53% (P1 x P3) to +13.80% (P7 x P8) fornumber of

leaves. Twenty threecrosses displayed positive MPH for seedling height; out of these 13 crosses revealed positively significant.Stem diameterexhibited neither positively nor negatively significant even though 17crosses expressed positive MPH.The top three crosses that showed greater than mid parents were crosses P4 x P6, P1 x P5 and P1 x P6 for seedling height and P4 x P6, P2 x P6 and P4 x P7 for stem diameter.While, crosses P2 x P4, P5 x P7 and P5 x P8 for seedling height and P3 x P8, P1 x P3 and P6 x P8 for stem diameter were the most inferior crosses.For average inter node length 24 crossesobserved positive and 10 crosses showed significantheterosis. Crosses P2 x P4 (-18.28%)and P2 x P6 (+59.39%) expressed theminimumandmaximumvalue, respectively.In case of nodes number, four crosses exhibited positively significant and P4 x P7 (+20.48%), P7 x P8 (+12.22%) and P3 x P5 (+10.16%) werethe three uppermost positive crosses. In addition, crosses P4 x P7, P4 x P5 and P2 x P6 showed higher heterotic effect to leaf area. MPH showed that 11 crosses for leaf area, eightcrosses for petiole length and two crosses for number of leaves showed positively significant heterosis.

More than half of the crosses expressed negative BPH, butless than three crosses showed positively significant for all growth characters. This heterosis expression is not remarkable; possibly had gene interactions that reduce the traits or lacking of dominance genes in desirable direction or present, but interacting in unfavorable condition. The manifestation of positive MPH for mean values and most individual crosses for all growth characters indicates that the possible presence of partialdominance genes or the existence of intra-allelic (multiple alleles effects) that responsible for controlling the inheritance of the characters. But, the mean value showed non-significant for all characters, this result suggests that even though possible presence of dominant genes or allelic interaction for some crosses combination, its effects for overall increment was not significant.Moreover,positiveMPH perhaps the result of inter-allelic interaction (epistasis) and the masking of harmful effects of recessive alleles (reducegrowth) by dominant alleles (responsible for growth improvement) or heterozygosity.Keep an eye on that parentsP4 and P2 had good MPH in their crosses for stem diameter; probablythe presence of either incomplete dominance alleles or heterozygosity effects.While, parents P3 and P1 expressed negative for most of their crosses; it mightbe the existence of few or no dominant alleles or gene interaction effects. Because, according to Falconer and Mackay (1996), heterosis can be expressed when either the presence of some

level of dominance or the relative difference in gene frequency of the two parents to determine the magnitude of heterosis expressed in the cross. If either or both of the conditions do not exist, heterosis will not be manifested. In summary, BPH was inadequate for seedling height, stem diameter, average internode length, number of nodes, petiole length, leaf area and number of leaves characters. However, MPH expressed about one third of the crosses for all growth characters, except stem diameter, number of nodes and number of leaves.

The expression of heterosisin Arabica coffee exploited and studied for their quantitative traits and CBD resistance characters by different scholars and reported the presence of heterosis both at seedling and mature coffee tree. The present growth characters result is in agreement with the work of Wassu (2004) and Ashenafi (2013), they observed heterosis relative to mid parent (MPH %) and better parent (BPH %) for plant height and average inter node length; at least one hybrid exhibited statistically significant relative to MPH and/or BPH either in the positive or negative direction. Different to the current result, they reported the existence of heterosis for stem diameter. Similarly, Bayetta (2001) studied on mature coffee plants and stated thatthe presence of fairly MPH for plant height and main stem nodes.However, hereportedcontradictoryresult for stem diameter (exhibited high BPH) and inter node length and leaf area (lacking heterosis). Generally, he concludes that the amount of heterosis expressed in growth characters was not appreciable.The possible reason for these results variations may be due to the differences and numbers of genotypes involved, growth stage and environmental factors. The current stem diameter result also in contrary to the work of Bayetta (1991) and Mesfin (1982) stated that both BPH and MPH manifested positive and significant heterosis. This controversial result also probably due to the stage of the seedling that the data taken (the former investigators collected data atseven months old seedlings) and the number of parents embraced in the crosses.Moreover, seedling growth media, nursery and greenhouse environmental differences, the presence of coffee wilt disease inside the seedlings and its interaction also the possible reason for result variation.

Table 5.Estimate ofbetter parent heterosis (BPH) for seedling stem and leaf characters

Crosses	SH	SSD	AINL	NN	PL	LA	NL
P1 x P2	0.70	-1.04	6.15	-7.52*	-8.18**	-26.46**	-12.96*
P1 x P3	4.69	-5.45	20.4	-19.51**	-2.70	1.51	-21.07**
P1 x P4	8.79*	1.79	23.27*	1.47	2.70	1.39	0.05
P1 x P5	13.46**	1.69	19.96	-14.50**	0.68	22.52*	-8.1
P1 x P6	3.31	-1.84	-10.53	-7.93**	-6.08	3.46	4.23
P1 x P7	7.07	0.77	19.49	-13.09**	-2.01	9.98	-0.76
P1 x P8	-0.71	-3.69	-8.81	-4.24	-3.85	-13.28	-0.92
P2 x P3	-2.65	0.00	-10.46	-4.88	-4.40	-10.49	1.33
P2 x P4	-6.70	-2.37	-18.99	2.46	-5.66	-1.09	3.29
P2 x P5	-2.78	0.74	-9.28	-2.95	-1.26	-15.29*	-6.2
P2 x P6	0.28	1.04	12.62	-20.69**	-7.55*	-1.29	-0.15
P2 x P7	1.58	-2.37	-5.35	-18.68**	-6.92*	-9.03	-12.96*
P2 x P8	7.43	-0.15	6.54	-13.23**	10.06**	7.51	0.51
P3 x P4	-6.75	-3.73	-22.18*	-19.54**	-1.36	-9.93	-6.61
P3 x P5	7.46	-3.64	-1.01	9.77**	7.48*	15.31	-2.56
P3 x P6	7.13	-0.76	4.06	-17.24**	4.08	11.89	2.61
P3 x P7	5.91	-5.45	2.78	-20.63**	2.01	7.61	-10.51*
P3 x P8	-10.09*	-7.42	-18.85*	-16.36**	-1.28	-18.65**	-7.94
P4 x P5	6.58	1.64	10.63	-14.00**	6.38	14.00	-13.74**
P4 x P6	8.37*	1.64	0.08	-11.49**	2.84	0.55	-2.06
P4 x P7	2.77	1.93	-1.07	11.11**	-14.77**	37.02**	4.35
P4 x P8	-0.33	1.19	1.31	-4.24	0.64	15.93*	2.51
P5 x P6	-0.24	-4.32	22.38	-12.64**	1.42	15.85	-9.36
P5 x P7	-8.11	-1.08	-24.62*	-11.43**	-13.42**	-11.51	-16.85**
P5 x P8	-8.98*	-0.93	-19.11*	-13.02**	-7.05*	-22.55**	-9.36
P6 x P7	-7.55	-3.72	-22.81*	-23.10**	-12.08**	-4.74	-4.23
P6 x P8	-8.23*	-5.33	-19.90*	-24.14**	-0.64	-24.85**	-7.07
P7 x P8	-2.05	0.78	-4.27	-0.14	-0.64	9.53	1.85
Mean	**0.73**	**-1.43**	**-1.70**	**-10.37**	**-2.20**	**0.18**	**-4.74**
SE (±)	**0.54**	**0.09**	**0.33**	**0.08**	**0.02**	**1.10**	**0.35**

P1 = 75227, P2 = 971, P3 = 74110, P4 =8136, P5 = 79233, P6 = 74144A, P7 = 974 and P8 = 370
Note: Values without asterisk (*) are non-significant, SE = standard error
AINL= average inter node length, LA= leaf area, NL= number of leaves, NN= nodes number, PL= petiole length, SH= seedling height, SSD= seedling stem diameter

Table 6. Estimates of mid parent heterosis (MPH) for seedling stem and leaf characters

Crosses	SH	SSD	AINL	NN	PL	LA	NL
P1 x P2	1.65	0.75	7.60	-1.72	-4.89	-11.64	-10.16*
P1 x P3	9.36*	-4.81	36.35**	-12.00**	-2.70	3.14	-17.53**
P1 x P4	9.43*	3.33	23.88*	1.84	5.19	12.17	0.41
P1 x P5	16.59**	2.00	48.63**	-6.83*	3.12	25.47**	-1.35
P1 x P6	15.42**	1.43	25.60	3.35	2.58	14.02	5.34
P1 x P7	7.57*	3.31	21.57*	-5.44	-1.68	17.84*	8.37
P1 x P8	3.30	-2.72	2.05	-0.78	-1.32	4.55	1.63
P2 x P3	2.64	1.12	2.60	-1.95	-0.98	8.94	2.62
P2 x P4	-6.34	-2.08	-18.28	9.27**	0.00	8.56	6.97
P2 x P5	0.86	2.87	13.57	-0.32	4.67	3.75	-2.31
P2 x P6	12.99**	6.23	59.39**	-15.90**	4.26	28.10**	1.99
P2 x P7	2.09	1.85	-5.00	-6.49*	-3.90	2.99	-2.19
P2 x P8	10.75**	2.67	17.80*	-10.92**	11.11**	7.96	1.15
P3 x P4	-2.05	-2.93	-11.49	-11.71**	0.35	1.06	-2.09
P3 x P5	9.29*	-2.68	9.88	10.16**	9.34**	16.24	0.29
P3 x P6	14.95**	3.23	33.89*	-14.79**	12.92**	21.55*	6.12
P3 x P7	11.13**	-2.42	18.16	-6.33*	2.36	17.00*	1.68
P3 x P8	-2.46	-5.86	1.25	-11.54**	1.32	-0.68	-6.18
P4 x P5	10.14**	3.64	37.74**	-5.98*	6.38*	28.82**	-7.05
P4 x P6	21.69**	6.72	51.93**	-0.32	9.85**	21.28*	-0.67
P4 x P7	2.89	6.20	0.16	20.48**	-12.41**	41.85**	13.58**
P4 x P8	3.11	3.90	12.88	-0.43	5.72*	27.69**	5.51
P5 x P6	8.69*	-1.27	44.71*	-9.74**	8.33*	24.92*	-3.61
P5 x P7	-5.14	1.26	-5.38	4.05	-11.03**	-3.07	-3.15
P5 x P8	-2.77	-0.08	9.06	-8.48**	-2.36	-4.86	-4.98
P6 x P7	3.71	-2.93	9.49	-7.08*	-3.68	11.68	5.59
P6 x P8	6.16	-3.13	20.08	-17.55**	11.11**	-2.19	-5.67
P7 x P8	1.47	2.31	5.48	12.22**	1.64	24.44**	13.80**
Mean	**5.97**	**0.78**	**16.91**	**-3.39**	**1.97**	**12.56**	**0.29**
SE (±)	**0.47**	**0.08**	**0.28**	**0.07**	**0.01**	**0.95**	**0.30**

P1 = 75227, P2 = 971, P3 = 74110, P4 =8136, P5 = 79233, P6 = 74144A, P7 = 974 and P8 = 370
Note: Values without asterisk (*) are non-significant, SE = standard error
AINL= average inter node length, LA= leaf area, NL= number of leaves, NN= nodes number, PL= petiole length, SH= seedling height, SSD= seedling stem diameter

4.4. Combining Ability

4.4.1. Analysisof variance for combining ability

Mean squares of general combining ability (GCA) and specific combining ability (SCA) for disease characters (wilted seedling percentage, number of defoliated leaves per seedling and incubation period), seedling stem and leaf characters are given in Table 7. The mean squares of GCA and SCA were significant at $p<0.01$and/or $P<0.05$ for all traits, except number of defoliated leaves per seedling and stem diameter of SCA. The result indicated thatboth GCA and SCA genetic variance were significantly important or the involvement of both additive and non-additive gene actions in the inheritance of wilted seedling percentage, incubation period and six seedlinggrowth characters. But, stem girth and number of defoliated leaves per seedling showed non-significant SCA mean squares; additive gene action was the major genetic component for governing the inheritance of the characters.

The relative predominance of genetic variance was estimated for eight characters (Table 7). GCA to SCA variance ratio for wilted seedling percentage, incubation period and leaf area were greater than one. These indicate that the relative importance of additive over non-additive gene action for controlling the inheritance of traits. Thus, the need to promote pure line selection to improve the genetic potential of Arabica coffee to obtain CWD resistant genotypes or making hybridization, then selection from segregating generation would be the principal approach to improve the characters. However, the calculated GCA to SCA genetic variance ratio being less than one for seedling height, average inter node length, number of nodes, petiole length and number of leaves. The result evidently suggests that the relative predominance of non-additive gene action. Therefore, the importance of choosing appropriate breeding lines that have vigorous and to promote hybrid program beyond selection is essential to improve the genetic potential of these characters.

Musoli *et al*. (2013) studied the inheritance of resistance to *Fussarium xylarioides* in Robusta coffee and reported that GCA variance component for CWD resistance was significant. Contrary to the current result, SCA was non-significant. Generally, he concluded that additive and dominance variances were low compared to the environmental variance.Similarly, Epinat

and Pitrat (1994), Mwanga et al. (2002), Patel and Pathak (2011) and Changaya et al. (2012) on muskmelon downy mildew resistance, sweat potato SPVD reaction, castor and pigeon pea fussarium wilt resistance reported the importance of both additive and non-additive genetic effects, in that order. Disagreeing to the present study, Mert et al. (2004) and Luders et al. (2008) on cotton verticillium wilt, Vander Vosen and Walyaro (2009) on coffee berry disease and Manu *et al*. (2014) on chilli fussarium wilt reported a single dominant gene control the inheritance of resistance.

The present seedling growth characters combining ability analysis confirmed the observation made with other parents and crosses by other investigators (Mesfin, 1982; Walyaro, 1983; Ashenafi, 2013), except seedling diameter character (they reported significant GCA and SCA effects). This is due to the differences in planting materials, coffee growth stage and location of the study. Those above mentioned reports were carried out on mature coffee plants in the field condition. Likewise, those investigators stated the importance of both the additive and non-additive gene actions for the expression of growth traits and non-additive gene action is predominant. The current growth characters results also in agreement with the work of Bayetta (2001), but different and reverse significance results for SCA mean squares of average inter node length, stem diameter and leaf area. He also reported the predominance of GCA over SCA mean squares for most growth characters. Similarly, Bayetta (1991), in his nursery evaluation of indigenous coffee crosses and Wassu (2004), on mature plant at the field, reported the importance of both additive and non-additive gene actions for plant height, number of nodes and inter node length. However, for stem girth they reported differently (both GCA and SCA mean squares significant). The relative contribution of GCA to SCA for inter node and leaf petiole length are in contrary to the report made by Ashenafi (2013), he concluded, higher GCA variance than SCA for the characters. This disagreement could be due to the above mentioned factors.

Therefore, selection and hybridization could be an effective breeding approach to exploit the advantage of both additive and non-additive genetic variances for wilted seedling percentage, incubation period and almost all seedling growth characters improvement.Even though, CWD resistance character is controlled by more of additive gene action, some of non-additive gene action (dominance and epistasis) might be improved the character through hybridization.

Table 7.GCA and SCA analysis of variance for 8x8 parents' half diallel matingdesign using Griffing's(1956) approach

Source	DF	Mean Square and P'									
		WS (%)	IP (days)	NDL (no.)	SH (cm)	SSD (mm)	AINL (cm)	NN (no.)	PL (cm)	LA (cm^2)	LNO (no.)
Block	2	1065.19 (801.08)	397.34	8.22**	9.97**	0.01^{ns}	0.18^{ns}	0.03*	0.0065**	24.11**	2.85**
Genotypes	35	1743.23** (823.29**)	610.80**	1.73**	2.62**	0.022*	1.16**	0.17**	0.0032**	18.02**	0.57**
GCA	7	2259.25** (1106.62**)	718.24**	2.30**	1.77*	0.022**	1.16**	0.13**	0.003**	20.13**	0.41*
SCA	28	161.53** (66.38*)	74.94**	0.14^{ns}	0.65**	0.004^{ns}	0.19**	0.04**	0.0006**	2.48**	0.13**
GCA to SCA variance ratio		2.35	1.22	_	0.32	–	0.81	0.37	0.59	1.04	0.47
Error	70	68.16 (39.34)	17.57	0.22	0.15	0.004	0.053	0.003	0.0001	0.6	0.0595

*=Significant at 5% level of significance, **= significant at 1% level of significance, ns= non-significant, P= probability level
GCA=general combining ability;SCA=specific combining ability,
cm= centimeter, cm^2 = centimeter square, mm=millimeter, no. = number

AINL= average inter node length (cm), IP = incubation period (days), LA= leaf area (cm^2), NDL= number of defoliated leaves per seedling, NL= number of leaves, NN= number of nodes, PL= petiole length (cm), SH= seedling height (cm), SSD= seedling stem diameter (mm),WS%= Wilted coffee seedlings percentage
Data in bracket is arcsine transformed value of wilted seedlings percentage

Mainly,CWD resistance could be incorporated from resistant sources by utilizing methods such as pure line selection, backcross or pedigree method; all of which take advantage of additive gene action (Sleper and Poehlman, 2006). Van der Vossen and Walyaro (1980) and Bayetta (2001) reported similar combining ability estimate for CBD resistance.

4.4.2. General combining ability effects

Estimate of general combining ability (gca) effects for eight parental lines for threeCWDand seven seedling growth characters are given in Table 8. All parental lines, except P4 showed either positive or negative significant ($P<0.01$) gca effects for wilted seedling percentage. The resistant (tolerant) or moderately tolerant parental lines P2, P7, P8 and P5 were exhibited highly significant and negative gca effect, in that order of desirable effects. While, parental lines P3, P1 and P6 were highly significantand positive gca effects for wilted seedling percentage as expected. Similar to mean result, a more negative gca effect showed greater CWD resistant. Therefore, parents P2, P7, P8 and P5 were good general combiners and probably contributed resistance genes towards their progenies; P2 (-17.13) was the best general combiner. Likewise, significant gca effects were found in all parents for incubation period (Table 8). Based on gcaeffects, certainly concluded that parents P8(+7.97), P2(+6.73), P7(+6.40) and P5 (+6.00)were good general combiners and P1(-11.93), P3 (-9.90)and P6 (-8.23) were poor combiners for incubation period, in that order. This result indicates that good general combiner parents (considered as a resistant parents) were desiredand extendedincubation period (days) as compared to the susceptible parents.

For number of defoliated leaves, parents P7, P8, P5 and P2 were also significant negative gcaeffects and good combiners; whileP6, P3 and P1 were positively significant effects. Genotypes that showed low or minimum number of defoliatedleaves andhigh negative gcaeffects wereconsidered as desirable to CWD resistant or tolerant and good combiners; provided an important contribution for CWD resistance. Generally, parent P2 exhibited the largest negative gca effects for wilted seedling percentageand positivegca effects for incubation period with optimum number of leafdefoliation.In conclusion, those parents that had low mean wilted seedling percentage, longer incubation period and minimum number of defoliatedleaves directly related todesirable gca effects.

The gcaeffects ofseedling stem characters (height, stem diameter, numberof nodesand average inter node length) are described in Table 8. There were positive and significant gca effects for parents P8, P1 and P4 for seedling height; P2 and P4 for stem diameter; P5, P6 and P3 for number of nodes; P8 and P1 for average inter node length;in that order of effects and good general combiners. Therefore, these parents could be useful to include in the breedingprograms for improvement. However, different parents responsible for improvement of distinct characters; so either improve each character individually or select the most economical and vital traits. Parental lines P6 and P3 for seedling height; P6 for seedling diameter; P6, P5 and P3 for average inter node length and P7, P1 and P8 for number of nodes showed poor general combiners. Additionally, three parents for seedling heightand average inter node length; five parents for stem diameter; one parent for number of nodes revealed non-significant gca effects.Variation due to gca effects was also significant for three leaf characters. Among the parental lines, P8, P2 and P3 for petiole length; P8, P2 and P4 for leaf area and P5 for leaves number were good general combiners. But, parents P6 and P7 for petiole length; P6, P3and P1 for leaf area and P7 and P1 for leaves number were poor general combiners. As described in Table 8, three parents for petiole length and number of leaves, and oneparent for leaf area showednon-significantgca effects.

On the basis of overall performance, P2 also selectedas desirable combiner for low wilted seedling percentage (CWD resistant),seedling stem diameter,petiole length and leaf area. So, itisimportant to include in resistancebreedingprogram for simultaneous improvement of multiple characters. Likewise, P8 was responsible for seedling height, average inter node length, petiole length and leaf area, and P5 was good combiners for number of nodes and number of leaves.On the other way, P6 was the least desirable parent (poor general combiner) for all growth characters, except nodes and leaves number. A parent exhibiting significantly positive and negative gca effects for a particular character is assumed to have high degree of favorable and unfavorable alleles, respectively (Stangland *et al.*, 1983). So, based on the results of Table 8, presumably favorable and / or unfavorable genes were present in each parent for both disease and growth characters.

Table 8. Estimate of general combining ability effects (gca effects) for CWDparameters and growth characters in eight parents

Parents	WS%	IP	NDL	SH	SSD	AINL	NN	PL	LA	NL
P1	17.56**	-11.93**	0.350*	0.47**	0.003 ns	0.32**	-0.09**	-0.003 ns	-1.13**	-0.18*
P2	-17.13**	6.73**	-0.358*	0.11 ns	0.065**	0.10 ns	0.04**	0.021**	1.56**	0.11 ns
P3	18.75**	-9.90**	0.535**	-0.38**	-0.02 ns	-0.27**	0.08**	0.008*	-1.27**	0.10 ns
P4	-0.56	2.97*	0.045 ns	0.23*	0.070**	0.13 ns	-0.02 ns	-0.007ns	1.27**	-0.02 ns
P5	-8.33**	6.00**	-0.381**	-0.22 ns	0.002 ns	-0.35**	0.14**	-0.006 ns	-1.12**	0.25**
P6	15.04**	-8.23**	0.715**	-0.73**	-0.052*	-0.49**	0.11**	-0.024**	-1.56**	0.03 ns
P7	-15.06**	6.40**	-0.505**	0.04 ns	-0.037 ns	0.06 ns	-0.20**	-0.015**	0.43 ns	-0.39**
P8	-10.27**	7.97**	-0.403**	0.48**	-0.032 ns	0.50**	-0.06**	0.024**	1.82**	0.10 ns
$SE(g_i)$	2.44	1.24	0.14	0.11	0.02	0.07	0.016	0.004	0.23	0.072
$SE(g_i-g_j)$	3.69	1.87	0.21	0.17	0.03	0.10	0.024	0.005	0.35	0.109

P1 = 75227, P2 = 971, P3 = 74110, P4 =8136, P5 = 79233, P6 = 74144A, P7 = 974 and P8 = 370, *=Significant at 5% level of significance, **= significant at 1% level of significance, ns =non-significant, SE= standard error of parents

AINL= average inter node length (cm), IP = incubation period (days), LA= leaf area (cm^2), NL= number of leaves, NDL= number of defoliated leaves per seedling, NN= nodes number, PL= petiole length (cm), SH= seedling height (cm), SSD= seedling stem diameter (mm), WS%= Wilted coffee seedlings percentage

4.4.3. Specific combining ability effects

Fifteen crossesfor wilted seedling percentage andeleven crossesfor incubation periodshowed negative and positive specific combining ability (sca) effect (desirable direction), respectively (Table 9). But, only crosses P4 x P8 (-24.88) and P7 x P8 (-18.01) revealed significant ($P<0.01$) and negative sca effects and good specific combinations for low wilted seedling percentage or CWD resistance. These crosses were the result ofmoderately resistant x moderately resistant and resistant x moderately resistant parents, respectively.The scaeffects showed out of 11 desirable crosses forincubation period, eight crosses showed significant.Crosses P7 x P8 (resistant x moderately resistant), P2 x P4 (resistant x moderately resistant), P4 x P8 (moderately resistant x moderately resistant) were the three topgood specific combinations. While,P1 x P8 (susceptible x moderately resistant), P1 x P2 (susceptible x resistant), P3 x P7 (susceptible x resistant) andP3 x P8 (susceptible x moderately resistant)werethe most undesirable crosses and poor specific combination for both wilted seedling percentage and incubation period.Number of defoliated leaves per seedlingwas not included in the sca effects since SCA mean squareshowednon-significant.Generally, crosses P7 x P8, P4 x P8 and P4 x P7 detected significantfavorable sca effectsfor wilted seedling percentage and incubation period; it associated with lowermean wilted seedling percentage, extended mean incubation period and negative heterosis.

Estimates ofsca effects for seedling height, average inter node lengthand number of nodes is given in Table 9. The positive sca effects manifested on 17 crosses for seedling height; 15 crosses for average inter node length;13 crosses for numberof nodes; and out of these crosses, six, sixand fivecrosses manifested positive and significant ($p<0.01$ and/ or $p<0.05$)sca effects for each character, respectively. Crosses P4 x P6, P2 x P8 and P3 x P7 for seedling height; crosses P2 x P6, P1 x P5 and P4 x P6 for average inter node length and crosses P3 x P5, P4 x P7 and P7 x P8 for number of nodes observed good specific combiners and ranked the top three crosses in their order of specific combination. While, cross P2 x P4 for seedling height; P2 x P4 for average inter node length and crosses P6 x P8 and P2 x P6 for number of nodes showed unfavorable specific combinations.

Table 9. Estimates of specific combining ability effects (sca effects) for CWD and seedling growth characters in artificial inoculation test

Crosses	WS	IP	SH	AINL	NN	PL	LA	NL
P1 x P2	17.01*	-7.73*	-0.43	-0.14	0.02	-0.024*	-2.00**	-0.54*
P1 x P3	-10.87	8.24*	0.32	0.57**	-0.19**	-0.018	-0.16	-0.94**
P1 x P4	-6.22	-6.63	0.42	0.30	0.01	0.024*	-0.11	0.03
P1 x P5	14.88	-3.99	1.31**	0.65**	-0.13*	0.012	1.96**	0.15
P1 x P6	-5.83	9.91*	0.49	-0.14	0.25**	-0.003	0.34	0.38
P1 x P7	-3.73	1.61	0.35	0.35	-0.14**	0.011	0.88	0.36
P1 x P8	19.48*	-7.96*	-0.16	-0.33	0.08	-0.014	0.001	0.18
P2 x P3	3.94	8.57*	-0.03	-0.07	0.08	-0.015	0.74	0.29
P2 x P4	10.75	16.04**	-1.18**	-0.73**	0.21**	-0.006	-0.28	0.36
P2 x P5	-8.59	-4.66	-0.21	0.05	0.05	0.015	-0.20	0.004
P2 x P6	-0.53	7.57	0.71*	0.86**	-0.25**	0.000	2.52**	0.09
P2 x P7	-5.47	-2.06	0.12	-0.22	-0.14**	-0.006	-0.72	-0.33
P2 x P8	-8.02	-3.63	1.36**	0.47*	-0.13*	0.045**	0.72	0.07
P3 x P4	-1.08	-3.33	-0.79*	-0.50*	-0.21**	-0.010	-1.48*	-0.13
P3 x P5	7.03	-2.36	0.61	-0.10	0.38**	0.032**	0.93	0.27
P3 x P6	-11.01	7.87*	0.64	0.15	-0.19**	0.033**	1.02	0.45*
P3 x P7	17.75*	-5.09	1.04**	0.41	-0.11*	0.021*	0.73	-0.01
P3 x P8	14.18	-12.99**	-0.64	-0.13	-0.13*	-0.012	-0.74	-0.33

Table 9.(Continued)

Crosses	WS	IP	SH	AINL	NN	PL	LA	NL
P4 x P5	15.75*	-4.89	0.81*	0.59**	-0.19**	0.020	1.56*	-0.41
P4 x P6	-4.09	-5.66	1.56**	0.64**	0.08	0.022*	0.21	-0.19
P4 x P7	-8.70	11.04**	0.05	-0.11	0.32**	-0.047**	3.09**	0.46*
P4 x P8	-24.88**	13.81**	0.14	0.24	0.01	0.014	2.38**	0.24
P5 x P6	7.07	-1.69	0.01	0.1	-0.12*	0.013	1.08	-0.15
P5 x P7	2.22	-1.33	-0.97**	-0.36	0.06	-0.042**	-1.81*	-0.26
P5 x P8	-8.28	0.77	-0.64	-0.05	-0.13*	-0.028**	-1.53*	-0.21
P6 x P7	13.07	-2.09	-0.38	-0.17	-0.08	-0.018	-0.52	0.06
P6 x P8	14.25	-4.33	-0.03	0.05	-0.25**	0.023*	-1.46*	-0.43
P7 x P8	-18.01*	16.71**	0.09	0.10	0.29**	0.014	2.18**	0.57*
SE ± sij	7.49	3.80	0.35	0.21	0.05	0.01	0.7	0.22
Sii-Sjj	9.04	4.59	0.42	0.25	0.06	0.01	0.85	0.27
Sij-Sik	11.08	5.62	0.52	0.31	0.07	0.02	1.04	0.33
Sij-Skl	10.4	5.30	0.49	0.29	0.07	0.02	0.98	0.31

P1 = 75227, P2 = 971, P3 = 74110, P4 =8136, P5 = 79233, P6 = 74144A, P7 = 974 and P8 = 370

Note: Values without asterisk (*) are non-significant; *=Significant at 5% level of significance, **= significant at 1% level of significance, SE (sij), SE (sii) = standard error of the crosses i and j parents and the same parents, respectively
AINL= average inter node length (cm), IP = incubation period (days), LA= leaf area (cm^2), NL= number of leaves, NDL= number of defoliated leaves per seedling, NN= nodes number, PL= petiole length (cm), SH= seedling height (cm), SSD= seedling stem diameter (mm), WS%= Wilted coffee seedlings percentage

The sca effects for three leaf characters (leaf petiole length, leaf area and number of leaves per seedling) were significant (Table 9). More than half of the crosses; 15, 16 and 16 crosses revealed positive sca effects for petiole length, leaf area and numberof leaves, respectively. Crosses P2 x P8, P3 x P6, P3 x P5, P1 x P4 and P6 x P8 for petiole length; crosses P4 x P7, P2 x P6, P4 x P8, P7 x P8, P1 x P5 and P4 x P5 for leaf area ; crosses P7 x P8, P4 x P7 and P3 x P6 for number of leaves showed positive and significant sca effect at $p<0.01$ and/or $p<0.05$. The result indicated, these crosses were good specific combinations for leaf characters in that order and the two parents probably consists of different genetic composition for the characters. In the other way, crosses P4 x P7, P5 x P7, P5 x P8 and P1 x P2 for petiole length; P1 x P2, P5 x P7, P5 x P8, P3 x P4 and P6 x P8 for leaf area; P1 x P3 and P1 x P2 for number of leaves showed negative and significant sca effects. Hence, these crosses were undesirable specific combination effects for leaf characters and the two parents that build the crosses may involve similar genetic makeup. As well, the positive and negative significant sca effects showed the two lines that produce crosses have different genetic makeup or genetically divergent. According to Stangland *et al*. (1983), who concluded that large negative and positive sca effects of crosses show the two lines that build the crosses have similar and different genetic makeup, respectively.

4.5. Estimation of Genetic Variance Components, Heritabilityand GeneticAdvance

Estimated broad sense heritability for four CWD parameters and seven quantitative characters are presented in Table 10. Low wilted seedling percentage or CWD resistance (88.27%), incubation period (91.37%), few numbers of defoliated leaves (62.06%), seedling height (83.12%), numberof nodes per seedling (94.76%), average inter node length (86.37%),petiole length (86.34%), leaf area (90.01%) and number of leaves (68.47%) showed high heritability values. Medium broad sense heritability was recorded for seedling diameter (39.75%) and number of yellow leaves per seedling (28.29%). CWD resistance heritabilityis disagreement to Musoli, *et al*. (2012),he reported low to medium heritability in Robusta coffee wilt disease resistance.But, in this study CWD resistant showed high broad sense heritability and the transmission of genetic information from parent to offspring possibly high.Furthermore, the current most growth characters heritability results are in line with Mesfin and Bayetta (1983), who reported high broad sense heritability estimates for these quantitative characters in

Arabica coffee. However, a little bit disagreement with WAlyaro and Van der Vossen (1979), they reported medium heritability value for quantitative characters in Arabica coffee.

Estimates of genetic advance as percent of mean (GAM) that could be expected from selecting the top 5% desired treesof the genotype for both CWD parameters and quantitative characters are given in Table 10. Estimates of GAM showed high for low wilted seedling percentage or high survival percentage (68.61%), incubation period (24.00),minimum number of defoliated leaves (52.30%), average inter node length (36.87%) and leaf area (33.64). These high broad sense heritability coupled withhigh GAMindicated, these characters could be improved through simple selection. According to Panwar *et al*. (2015), selection would be much easier for high heritable character; but it will be difficult for a character with low heritability. He determined that heritability estimates along with expected genetic advance are usually more helpful than heritability value alone.

Table 10. Estimates of genetic variance components and heritability of CWD parameters and seedling growth characters

Characters	Estimates of heritability, genetic variance and genetic gain				
	δ^2_p	δ^2_g	h^2_B (%)	GA	GAM (%)
Low wilted coffee seedlings Percentage	581.08	512.92	88.27	43.83	68.61
Incubation period	203.60	186.04	91.37	26.86	24.00
Minimum number of defoliated leaves per seedling	0.58	0.36	62.06	0.97	52.30
Minimum number of yellow leaves per seedling	0.05	0.01	28.29	0.13	13.36
Seedling height (cm)	0.87	0.73	83.12	1.60	12.06
Seedling diameter (mm)	0.01	0.003	39.75	0.07	3.23
Number of nodes	0.06	0.05	94.76	0.46	19.38
Average inter node length (cm)	0.39	0.33	86.37	1.11	36.87
Number of leaves	0.19	0.13	68.47	0.61	9.65
Leaf area (cm2)	6.01	5.41	90.01	4.54	33.64
Petiole length (cm)	0.001	0.001	86.34	0.06	11.81

h^2_B= broad sense heritability, GA = genetic advance, GAM = genetic advance as percent of mean, δ^2_{GCA} = general combining ability variance,δ^2_g = genotypic variance, δ^2_p = phenotypic variance, δ^2_{SCA} = specific combining ability variance

4.6. Phenotypic and Genotypic Correlation among Characters

The estimates of phenotypic and genotypic correlationbetween and among characters are shown inTable 11.There were found highly significant ($p<0.01$) and negative correlation between wilted seedling percentage and incubation period (rp= -0.86, r_g = -0.91) and following components: leaf area (r_p = -0.59, r_g= -0.64) and stem diameter (r_p = -0.34, r_g= -0.41).While, it was positive and highly significant correlation with number of defoliated leaves (rp = +0.86, r_g = +1.00). Therefore, thesecorrelationsindicated that wilted seedling percentage showed strong negative correlation with incubation period;whilestrong positive associations with number of defoliated leaves. Longer incubation period of CWD was positive and highly significance ($p<0.01$) phenotypic and genotypic association with leaf area, but negative and highly significant correlation with number of defoliated leaves. Furthermore, incubation period showed positive and non-significant association with the rest quantitative characters, except seedling diameter and number of leaves for genotypic correlation (significant association). Therefore, low wilted seedling parentage (CWD resistance) could be improved by considering direct selection of longer incubation period, wide stem diameter and leaf area with minimum number of defoliated leaves and number of nodes. Selections for characters based on its positive and significant association are very useful for simultaneous improvement of the associated characters. On the other hand, characters manifesting negative association, simultaneous improvement of characters could be quite difficult and independent selection may have to be carried out to improve the characters (Sylva and Carvalho, 1997).

Theassociations of wilted seedling percentage with seedling height, average inter node length, petiole length,number of leaves and number of yellow leaves per seedling showednegative andnon-significant association. Similar report suggested that morphological traits (height, basal diameter of the axis, and the number of branches) of Japanese pines are associated with resistance to PWN (Toda *et al*., 1986; Toda and Fujimoto, 1987; Kuroda, 2004), a thicker basal diameter predicted to survive better (Yamanobe, 2009).Siddiqui and Singh (2004) at glass-house experiments were conducted to assess the effects of wilt fungus on growth and transpiration of chickpea. Results showed inoculation of *Fussarium oxysporum* reduced plant growth, transpiration and caused severe wilting.

Table 11.Phenotypic (rp) (above diagonal) and genotypic (rg) (below diagonal) correlationamongcharacters

	WS (%)	IP (days)	SH (cm)	SSD (mm)	AINL (cm)	NN (no.)	PL (cm)	LA (cm^2)	NL (no.)	NDL (no.)	NYL (no.)
WS (%)		-0.86**	-0.18	-0.34*	-0.22	0.08	-0.22	-0.59*	-0.10	0.86**	-0.17
IP (days)	-0.91**		0.17	0.29	0.19	0.02	0.16	0.68**	0.28	-0.74**	0.15
SH(cm)	-0.17	0.20		0.44**	0.89**	-0.41*	0.58**	0.60**	-0.08	-0.33*	-0.05
SG(mm)	-0.41**	0.44**	0.52**		0.36*	0.10	0.26	0.51**	0.30	-0.28	0.23
AINL(cm)	-0.25	0.23	0.97**	0.57**		-0.58**	0.55**	0.65**	-0.24	-0.34*	-0.03
NN(no.)	0.08	0.01	-0.44**	0.18	-0.60**		-0.13	-0.18	0.68**	0.23	0.02
PL(cm)	-0.23	0.18	0.61**	0.27	0.64**	-0.15		0.46**	0.25	-0.33*	0.39*
LA (cm^2)	-0.64**	0.75**	0.66**	0.69**	0.72**	-0.17	0.48**		0.20	-0.51**	0.16
NL(no.)	-0.09	0.34*	-0.24	0.25	-0.29	0.83**	0.23	0.19		0.002	0.28
NDL(no.)	1.00**	-0.96**	-0.31	-0.02	-0.43**	0.27	-0.35*	-0.58**	0.13		-0.06
NYL(no.)	-0.37*	0.30	-0.03	0.55**	-0.02	0.004	0.76**	0.27	0.57**	0.01	

Note: Values without asterisk (*) are non-significant; cm= centimeter, cm^2 = centimeter square, mm=millimeter

AINL= average inter node length, IP = incubation period, LA= leaf area, NL= number of leaves, NDL= number of defoliated leaves per seedling, NN= number of nodes, PL= petiole length, SH= seedling height, SSD= seedling stem diameter, WS%= Wilted coffee seedling percentage

5. SUMMARY AND CONCLUSION

Coffee wilt disease is a vascular wilt disease caused by *Gibberella xylarioides* Heim and Saccas. It becomes an increasing importance in Arabica coffee production. Understanding the mode of inheritance of resistance mechanism provide valuable evidence in the development of CWD resistant variety and designing appropriate breeding programs. However, there is lacking of information on combining ability, heterosis, heritability, type of gene action in controlling the inheritance of CWD resistance and correlation among characters. The study was conducted to estimate combining ability, type of gene action, heterosis,heritability and to determine the association of characters. Eightparents crossed in 8 x 8 half diallel mating designand one susceptible check were tested using RCBD with three replications in greenhouse, JARC. Selfing, crossing, seedling raising, pathogen inoculum preparation, inoculation of seedling and management, data collection and analysis weredone properly using suggested methods and procedures.

The result of analysis of varianceshowed thatparents and F1 crosses were highly significant ($p<0.01$) for wilted seedling percentage, incubation period, number of defoliatedleaves and all seedling growth characters. From the result, parents P2 (971) and P5 (79233) exhibited low mean wilted seedling percentage (high survival rate) and small number of defoliated leaves with an extended incubation period. Besides,cross P7 x P8 revealed the lowest mean wilted seedlings percentage along with longest incubation period (143 days).

Percentage of BPH and MPHfor wilted seedling parentage was lacking in the required direction (only crosses P4 x P8 and P7 x P8 showed significant MPH) and number of defoliatedleaves showed non-significant and imperceptible. However, about 89% (50% significant) of the crosses for BPH and 64% for MPH showed undesirable wilted seedling percentage. Moreover, 100% and 68% of the crosses for number of defoliated leaves showed unfavorable heterotic effects over the better parent and mid parent, respectively. Theseheterosis result and mean comparison between parents with F1 crosses suggested that probably partial to complete dominance of susceptible genes over the resistant genes or CWD resistance may be affected by multiple gene effects. Therefore, use of heterosis breeding

maybe rarely essential to improve these characters. For incubation period three and eight crosses exhibited significant positive (desirable direction) BPH and MPH, respectively. However, nearly, in all calculated heterosis cross P7 x P8 expressedin desirable direction and it was supreme cross to gain heterotic effects for CWD characters.All crosses for incubation period and more than half of the crosses for wilted seedling percentage and number of defoliated leaves manifested favorable heterosis effects over susceptible parent and susceptible check.All growth charactersmanifested inadequate BPH; only three crosses for seedling height and leaf area; two crosses for number of nodes and petiole length; and one cross for average internode length showed positively significant.But, MPH expressed about one third of the crosses for seedling height, average internode length, petiole length and leaf area. However, no F1 hybrids showed positive and significant BPH for stem diameter and number of leaves.

The combining ability analysis suggested thatboth additive and non-additive gene actions were involved in controlling the inheritance of all characters, except stem diameter and number of defoliated leaves. The predominance of additive over non additive gene action was observed for wilted seedling percentage, incubation period and leaf area. However, GCA to SCA variance ratio being less than one for all seedling growth characters (except leaf area), evidently indicatedthe relative importance of non-additive gene action. In general, selection and hybridization could be an effective breeding approach to exploit the advantage of both additive and non-additive gene actions in order to improve CWD resistance, incubation period and most growth characters.

Parents P2, P7, P8 and P5 exhibited highly significant negative gca effects and good general combiners for wilted seedling percentage;which are important to enclosure in future resistance breeding program. Whereas, P3, P1 and P6 were exhibited positively significant gca effects and poor general combiners. Moreover, P2 was found desirable combiner for low wilted seedling percentage (CWD resistance), incubation period, seedling stem diameter,petiole length and leaf area.Likewise, P8 for incubation period, seedling height, average inter node length, petiole length and leaf area; P4 for stem diameter; and P5 for number of nodes and number of leaves were good general combiners. Estimation of sca effects showed that crosses P7 x P8 and P4 x P8 were significant and good specific combiners for wilted seedling

percentageand incubation period in the favorable direction. These crosses showed the importance of hybridization to decrease wilted seedling percentage and increase incubation period. Contrary, P1 x P8, P1 x P2, P3 x P7 and P3 x P8 were the most undesirable crosses and poor specific combination for the characters. Parent P4, P7 and P8 was able to combine and contributed desirable scaeffects for CWD resistance.

The estimation of high broad sense heritability coupled with GAM were observed for wilted seedling percentage, incubation period, average inter node length, leaf area and number of defoliated leaves per seedling; it could be improved easily through selection. Moreover, CWD resistant genotypes were significantly associated with extended incubation period, wide stem diameter, extensive leaf area and minimum number of defoliated leaves than the susceptible genotypes. As a result, CWD resistance could be improved by direct selection of these correlated characters.

The study concluded that promising CWD resistant genotypes were identified, the presence of low BPH and MPH for CWD resistance, shows predominance of additive over non additive gene action and estimated high broad sense heritability coupled with high GAM for CWD resistance and incubation period; indicates selection and hybridization are important to improve population and to obtain segregating generation, respectively. Hence, further study on F2, BC1 and BC2 generation both in greenhouse and multi-location (field condition) is needed. Moreover, QTL mapping study should be significant. The resistant genotypes should be also evaluated further for other major diseases, yield and important agronomic traits.

6. REFERENCES

Acharya, S. N. and H.C. Huang, 2003. Breeding alfalfa for resistance to verticillium wilt: A sound strategy. *Advances in Plant Disease Management*, 345-371.

Acquaah,G., 2012. Principles of plant genetics and breeding.John Wiley & Sons. 740p.

Admasu, S. and F. Klause, 2007.National production effect of investment in coffee berry disease resistant selections in Ethiopia.Proceedings of the workshop on four decades of coffee research and development in Ethiopia. Addis Ababa (Ghion Hotel), Ethiopia, 14-17 August 2007.

Agrios, G. N., 2005. Plant pathology.Elsevier Academic Press, USA. 922p.

Allard, R.W., 1999. Principles of plant breeding. John Wiley and Sons, New York. 254p.

Anthony, F., B. Bertrand, O. Quiros, A. Wilches, P. Lashermes, J. Berthaud and A. Charrier, 2001. Genetic diversity of wild coffee (*Coffea arabica* L.) using molecular markers.*Euphytica*, 118(1):53-65.

Anthony, F., M.C. Combes, C. Astorga, B. Bertrand, G. Graziosi and P. Lashermes, 2002.The origin of cultivated *Coffea arabica* L. varieties revealed by AFLP and SSR markers.*Theoretical and Applied Genetics*, **104**(5):894-900.

Arega, Z., 2006. Diversity of Arabica coffee population in afro-mountain rainforests of Ethiopia in relation to coffee berry disease (C*olletotrichum kahawae*) and coffee wilt disease (*Gibberella xylarioids*). An MSC Thesis presented to the school of graduate studies of Addis Ababa University. 81p.

Ashenafi, A., 2013. Heterosis and combining ability for morphological, yield and quality characters in coffee (*Coffea arabica* L.) hybrids. An MSC Thesis submitted to the school of graduate studies of Jimma University. 92p.

Azad, M. A. K., B.K. Biswas, N. Alam, S. S. Alam, 2014. Combining ability and heterosis for some quantitative traits in experimental maize hybrids.*Plant breeding and seed science*, **70**.

Bayetta, B. and A. Mesfin, 1986.Variation among national coffee collections for some agronomic characters.First Ethiopian coffee symposium. Addis Ababa, Ethiopia, August, 1986.

Bayetta B., 1991. Nursery evaluation of heterosis and combining ability in reference to origin and morphology of parents in coffee (*Coffea arabica* L.). An MSC Thesis presented to the school of graduate studies of Alemaya University. 128p.

Bayetta B., 2001. Arabica coffee breeding for yield and resistance to coffee berry disease (*Colleototrichum kahawae sp*. Nov). Doctoral dissertation submitted to the Wye University, London. 272p.

Beckman, C.H., 1987. The nature of wilt diseases of plants.APS Press, USA. 175p.

CABI, 2003. Surveys to assess the extent of coffee wilt disease in East and Central Africa. Final technical report.CABI Regional Center, Nairobi, Kenya. 49p.

Carvalho, A., 1952. *Coffea arabica* L. Taxonomy VI: morphological characters of haploid. *Bragantia*, **12** (4-6): 201-212.

Carvalho, A., F.P. Ferwerda, J.A. Frahm-leliveld, D.M. Medina, A.J.I. Mendes and L.C. Monaco, 1969. Coffee (*C. arabica* L. and *C. canephora* Pierre ex froehner). pp. 186-244. In: F.P. Ferwerda and F. Wilt (eds). Outlines of perennial crop breeding in the tropics. Landbouwhoge school (Agricultural University) Wageningen, Netherland.

Carvalho, A., 1988. Principles and practice of coffee plant breeding for productivity and quality factors: coffea arabia. pp. 129-165. In: R.G. Clarke and R. Macrae (eds). Coffee: Agronomy. Elsevier applied science publishers Ltd, London.

Chala, J., A. Girma, T. Demelash, Z. Arega, B. Sihen, and A. Adem, 2012. Development and release of coffee berry disease resistant varieties to specialty coffee producing regions in Ethiopia. pp.637-644. Proceedings of 24thinternational scientific colloquium on coffee (ASIC). Costa Rica, 12-16 November 2012, Jimma Agricultural Research Centre.

Changaya, A. G., R. Melis, J. Derera, M. Laing, V. W. Saka, 2012. Inheritance of resistance to fusarium wilt and yield traits in pigeon pea. *Euphytica,* **186**:883–896.

Cook R.J., W.L. Bruckart, J.R. Coulson, M.S. Goettel, R.A. Humber, R.D. Lumsden, J.V. Maddox, M.L. McManus, L. Moore, S.F. Meyer, P.C. Quimby, J.P. Stack, and J.L. Vaughn, 1996. Safety of microorganisms intended for pest and plant disease control. A framework for scientific evaluation.*Biol. Control*,**7**: 333-351.

Central Statistical Agency (CSA), 2016.The Federal Democratic Republic of Ethiopia Agricultural sample survey report on area and production of major crops, volume, I. statistical bulletin, 584.

Davenport, C.B., 1908. Degeneration, albinism and inbreeding.*Science*, **28**(718):454-455.

Davis, A.P., F. Rakotonasolo and P. De Block, 2010. *Coffea toshii sp. nov*. (Rubiaceae) from Madagascar.*Nordic Journal of Botany*, **28**(2):134-136.

Demelash, T., 2013.Evaluation of Arabica coffee (*coffea arabica* L.) germplasm for major coffee disease with special emphasis to coffee wilt disease (*Gibberella xylarioids*) at Jimma, Ethiopia. An MSC Thesis presented to the school of graduate studies of Jimma University.

Demelash, T. and B. Kifle, 2015. Evaluation of released Arabica coffee varieties (*Coffea arabica* L.) for major coffee diseases with special emphasis to coffee wilt disease (*Gibberella xylarioides*) at Jimma, Ethiopia. *Journal of Biology, Agriculture and Healthcare*, **5** (15).

Charrier, A. and J. Berthaud, 1985.Botanical classification of coffee. pp. 13-47. In: M. N. Clifford and K. C. Wilson (eds.). Coffee Botany, Biochemistry and Production of Beans and Beverage. Croom Helm, London.

El-Bramawy, M.A.E.H.S., S.E.S. El-Hendawy and W. I. Shaban, 2009.Assessing the suitability of morphological and phenotypical traits to screen sesame accessions for resistance to Fusarium wilt and charcoal rot diseases.*Plant Protect. Sci*. **45**: 49–58.

Epinat, C. M. Pitrat, 1994. Inheritance of resistance to downy mildew (*Pseudoperonospora cubensis*) ´in muskmelon (*Cucumis melo*). I. Analysis of 8 x 8 diallel table. *Agronomie*, *EDP Sciences*, **14** (4):239-248.

Eshetu, D., 1997. Coffee diseases and their significance in Ethiopia.*ASIC*.**17**(I): 723-726.

Eshetu, D., G. Teame and A. Girma, 2000. Significance of minor diseases of *Coffea arabica*L.. In Ethiopia: review. pp. 58–65. Proceedings of the workshop on control of coffee berry disease (CBD) in Ethiopia. Addis Ababa, Ethiopia.

Falconer, D.S. and T.F.C. Mackay, 1996.Introduction to quantitative genetics.Longman and Scientific and technical, London. 438p.

FAO, 2006. Food and Agriculture Organization Corporate Statistical data base (FAOSTAT). Available from: http://faostat.fao.org.

FAO, 2014.Analysis of price incentives for coffee in Ethiopia. Technical notes series, MAFAP, by Kuma Worako, T., MasAparisi, A. and Lanos B., Rome.

Fehr, W.R., 1987. Principles of cultivar development: theory and techniques. Macmillan Publishing Company, New York. 536p.

Fernie, L.M., 1970. The improvement of arabica coffee in East Africa.Crop Improvement in East Africa.*Techn. Comm*, 19:231-249

Flood, J., 2006. Reviews of Fusarium wilt of oil palm caused by *Fusarium oxysporumf. sp. elaeidis*. *Phyto-pathology*.**96**:660–662.

Gardner, C.O. and S.A. Eberhart, 1966. Analysis and interpretation of the variety cross diallel and related populations. *Biometrics*,**22**: 439–452.

Geiser, D.M., M.L.L. Ivey, G. Hakiza, J.H. Juba and S.A. Miller, 2005.*Gibberella xylarioides* (anamorph: *Fusarium xylarioides*), a causative agent of coffee wilt disease in Africa, is a previously unrecognized member of the *G. fujikuroi* species complex. *Mycologia*, **97**: 191-201.

Gharderi, A., M.W. Adams and A.M. Nassib, 1984.Relationship between genetic distance and heterosis for yield and morphological traits in dry edible bean and faba bean.*Crop sci*. **24**:32-34.

International Plant Genetic Resource Institute (IPGRI), 1996.Descriptors for coffee (*coffea spp*. and *psilanthus spp*).International plant Genetic Resources Institute., Rome, Italy. 38p.

Girma A., 1997. Characterization of *Gibberella xylarioides* Heim and Saccas (Fusarium wilt) of coffee (*Coffee arabica* L.). An MSc Thesis presented to the school of graduate studies of Alemaya University. 100p.

Girma, A. and H. Mengistu, 2000. Cultural characteristics and pathogenicity of *Gibberella xylarioides* isolates on coffee. *Pest management Journal of Ethiopia*, **4**:11-18.

Girma A., H. Mengistu and H. Hindorf, 2001.Incidence of tracheomycosis, *Gibberella xylarioides* (*Fusarium xylarioides*), on Arabica coffee in Ethiopia.*J. Plant Dis. and Pro*. **108** (2): 136-142.

Girma A., 2004. Diversity in pathogenicity and genetics of *Gibberella xylarioides* (*Fusarium xylarioides*) population and resistance of coffee spp. in Ethiopia. Doctoral Dissertation presented toUniversity of Bonn, Germany. 81p.

Girma A., H. Hindorf, U. Steiner, H. I. Nirenberg, H. W. Dehne and K. Schellander, 2005. Genetic diversity in the coffee wilt pathogen (*Gibberella Xylarioides*) populations: differentiation by host specialization and RAPD analysis. *J. Plant Dis. and Pro*. **112** (2):134-145.

Girma, A. and J. Chala, 2008. Resistance levels of Arabica coffee cultivars to coffee berry disease, coffee wilt and leaf rust diseases in Ethiopia. pp.92–103. Proceedings of the 12th conference of the crop science society of Ethiopia (CSSE). Addis Ababa, Ethiopia, 22-24 may 2006, Ethiopian Institute of Agricultural Research.

Girma A., A. Million, H. Hindorf, Z. Arega, T. Demelash and J. Chala, 2009a. Coffee wilt disease in Ethiopia. pp. 50-68. In: J. Flood (eds). Coffee wilt disease. Typeset by MTC, Manila and Philippines, UK.

Girma A., D. Biyesse and P.C. Musoli, 2009b. Host - pathogen interaction in Coffea-*Gibberella xylariodes* pathosystem. pp.120-136. In: J. Flood, (eds). Coffee wilt disease. Typeset by MTC, Manila and Philippines, UK.

Griffing, B., 1956. Concept of general and specific combining ability in relation to diallel crossing systems.*Aust. J. Biol. Sci*. **9**: 463–493.

Hedberg, I., S. Edwards and S. Nemomissa, 2003.Flora of Ethiopia and Eritrea.Vol. 4.Part 1, Apiaceae to Dipsacaceae.National Herbarium, Addis Ababa University.

Hallauer, A. R. and J. B. Miranda., 1988.Quantitative genetics in maize breeding.Iowa State University press, Ames.

Heim, R. and A. Saccas, 1950.Tracheomycosis of *Coffea excelsa* and *robusta* in the plantations of Oubangui-Chari. Compte Rendu de l'Academie des Sciences, 231(11):536-538.

Holland, J.B., W.E. Nyquist and C.T. Cervantes-Martinez., 2003. Estimating and interpreting heritability for plant breeding. In: J. Janick, (eds). Plant Breeding Reviews. John Wiley and Sons Inc., New York.

Huber, D.A., 1993. Optimal mating designs and optimal techniques for analysis of quantitative traits in forest genetics. Doctoral Dissertation presented to University of Florida. 151p.

Hurtl, D.L., 1994. Genetics. Jones and Bartlett Publishers, Boston.

Jeger, M. J., and S. L. H. Viljanen-Rollinson, 2001.The use of the area under the disease-progress curve (AUDPC) to assess quantitative disease resistance in crop cultivars.*Theor. Appl. Genet*. **102**:32-40.

Kambal, A.E. and O.S. Webster., 1965.Estimates of general and specific combining ability in grain sorghum.*Crop Sc*. **5**: 521-523.

Kangire, A., 2014. Enhancing resistance to coffee wilt disease in Uganda–the conventional way. Viewpoints: Africa's Future... Can Biosciences Contribute, p.41.

Kassahun, H. and G. Getnet, 2008.Performance, structure and prospects of coffee marketing in Ethiopia. pp. 416–423. In: Girma, A., B. Bayetta, S. Tesfaye, K. Taye and T. Endale (eds). Coffee Diversity and Knowledge: Proceedings of a national workshop four decades of coffee research and development in Ethiopia. Addis Ababa, Ethiopia, 14-17 August 2007.

Kifle, B., T. Demelash and G. Gabisa, 2015.Screening of some coffee Arabica genotypes against coffee wilt diseases (*Gibberella xylarioides* Heim and Saccus).*International Journal of Sustainable Agricultural Research*, **2**(3): 66-76.

Kilambo, D.L., N.M. Ng'homa, D.J. Mtenga, J.M. Teri, and N. Phiri, 2012.Progress in breeding for resistance to coffee wilt disease (tracheomycosis) in Ttanzania. *World research journal of agricultural & bio-systems engineering*, **1**: 12-16.

Kilambo, D.L., N.M. Ng'homa, J. Mtenga, J.M. Teri, T. Nzallawahe, R. Mike, and L. Masumbuko1, 2007. Progress towards searching for durable resistance to Fusarium wilt (*Fusarium xylarioides*) in *Coffea canephora* germplasm in Tanzania. pp. 1386–1389. Proceedings of the 20th International Scientific Conference on Coffee Science (ASIC). Bangalore, India.

Kranz, J. and M. Mogk, 1973.*Gibberella xylarioides* Heim and Saccas on Arabica coffee in Ethiopia.*Phytopath. Z*. **78**: 365-366.

Krug, C.A. and A. Carvalho, 1951.The genetics of Coffea.*Advances in genetics*, **4**:127-158.

Kuroda, K., 1999. Which factor decides dead or alive in tree? pp. 81–82. In: K. Suzuki (eds). Tree health. Asakura Shoten, Tokyo.

Kuroda, K., 2004. Inhibiting factor of symptom development in several Japanese red pine (*Pinus densiflora*) families selected as resistant to pine wilt. *Journal of Forest Research*, **9**: 217–224.

Lamkey, K. R. and J. W. Edwards, 1999.Quantitative genetics in heterosis. pp. 31-48. In: Coors, G. J. and S. Pandey, (eds.). Genetics and exploitation heterosis in crops. Amer. Soci. of Agron. Wisconsin, USA.

Lashermes, P., M. C. Combes, J. Robert, P. Trouslot, A. D'hont, F. Anthony and A. Charrier, 1999.Molecular characterization and origin of the *Coffea arabica* L. genome.*Molecular and General Genetics MGG*, **261**(2):259-266.

Lüders R. R., R. Galbieri, M. G. Fuzatto1, and E. Cia, 2008. Inheritance of resistance to Verticillium wilt in cotton. *Crop Breeding and Applied Biotechnology*,**8**: 265-270.

Manu, D. G., B. V. Tembhurne, B. Kisan, D. S. Aswathnarayana and J. R. Diwan, 2014. Inheritance of fusarium wilt and Qualitative and Quantitative Characters in Chilli (*Capsicum Annuum* L).*Journal of Agriculture and Environmental Sciences*, **3**(2):433-444.

Mathur P.N. and J.R. Mathur, 1983.Combining ability for yield and its components in pearl millet.*Indian J. Genet.Pl. Breeding*.**43**:299-303.

Melaku, W., 1984.Coffee genetic resources in Ethiopia conservation and utilization particular reference to CBD resistance. pp. 203-211. Proceedings of the first regional workshop on coffee berry disease. Addis Ababa, Ethiopia, 19-23 July 1982.

Merdassa, E., 1986. A review of coffee diseases and their control in Ethiopia. pp. 187–195. In: A. Tsedeke, (eds). Proceedings of the first Ethiopian crop protection symposium. Addis Ababa, Ethiopia, 4–7 February 1986, Institute of Agricultural Research.

Mert M., S. Kurt, O. Gencer, Y. Akiscan , K. Boyaci and F. M. Tok, 2005. Inheritance of resistance to Verticillium wilt (*Verticillium dahliae*) in cotton (*Gossypium hirsutum* L.).*Plant Breeding*,**124**:102-104.

Mesfin, A., 1982. Heterosis in crosses of indigenous coffee (*Coffee arabica* L.) selected for yield and resistance to CBD at first bearing stage. *Ethiopian Journal of Agricultural Science*, **4**: 33-43.

Mesfin, A. and B. Bayetta, 1983. Heterosis in crosses of indigenous coffee selected for yield and resistance to coffee berry disease: II-First three years. *Ethiopian Journal of Agricultural Science*, **5**:13-21.

Meyer, F. G., 1965. Notes on wild *Coffea arabica* from Southwestern Ethiopia, with some historical considerations, *Economic Botany*, **19**:136–151.

Ministry of Agriculture (MOA), 2015.Plant Variety Release, Protection and Seed Quality Control Directorate.Crop Variety Register, Issue N. 18, Adiss Ababa, Ethiopia.

Mugiira, R.B., P.F. Arama, J.M. Macharia, and B.M. Gichimu, 2011. Antibacterial activity of foliar fertilizer formulations and their effect on ice nucleation activity of *Pseudomonas syringar pv garcae* Van Hall; the causal agent of Bacterial Blight of Coffee. *Int. J. Agric. Res*. **6**(7): 550 –561.

Musoli, P. C., S. Olal, A. Nabaggala and C. Kabole, 2001.Screening Robusta coffee germplasm for resistance against coffee wilt disease. CORI Progress Report on Coffee Wilt Disease Research and Development, 1997–2000, Uganda.

Musoli, P.C., 2007. Sources of resistance against coffee wilt disease caused by *Fusarium xylarioides* in Uganda. Doctoral Thesis of Montpellier II University, France.

Musoli, P.C., A. Girma, G.J. Hakiza, A. Kangire, F. Pinard, C. Agwanda and D. Bieysse, 2009. Breeding for resistance against coffee wilt disease.pp.180-197. In: J. Flood, (eds). Coffee Wilt Disease. Typeset by MTC, Manila, Philippines, Printed and bound in the UK.

Musoli, P.C., C. Cilas, D. Pot, A. Nabaggala, S. Nakendo, J. Pande, A. Charrier, L. Thierry and D. Bieysse, 2013.Inheritance of resistance to coffee wilt disease (*Fusarium xylarioides* Steyaert) in Robusta coffee (*Coffea canephora* Pierre) and breeding perspectives. *Tree Genetics & Genomes*, **9**:351–360.

Mwanga, R. O. M., G. C. Yencho and J. W. Moyer, 2002. Diallel analysis of sweet potatoes for resistance to sweet potato virus disease. Euphytica, 128: 237–248.

Nelson, P. E., 1981. Life cycle and epidemiology of *Fusarium oxysporium*. pp. 51-80. In: M. E. Mace, A. A. Bell and C. H. Beckman (eds). Academic Press: London.

Olika, K., A. Sentayehu, K. Taye and G. Weyessa, 2011.Variability of quantitative traits in limmu coffee (Coffea arabica L.) in Ethiopia. International Journal of Agricultural Research, **6**(6):482-493.

PBTools, version 1.4., 2014.Biometrics and breeding Informatics, PBGB Division.
International Rice Research Institute, Los Baños, Laguna.

Panwar I. S., R. K. Arya, D. Phougat and S. K. Pahuja, 2015.Use of combining ability, heritability and genetic advance in breeding programmes.*Forage Res*.**41** (3):164-169.

Patel, P.B. and H.C. Pathak, 2011. Genetics of resistance to wilt in castor caused by *Fusarium oxysporum f. sp*. ricini Nanda and Prasad. *Agricultural Science Digest*, **31**(1):30-34.

Pieters, R. and N.A., Van der Graaff, 1980. *Gebberrella xyilarioides* on Arabica coffee: evaluation of testing methods and evidence for the horizontal nature of Resistance. *Neth. J. Pl. Path*. **86**:37-43.

Phiri, N. and P. Baker, 2009.Coffee Wilt disease in Africa.Final Technical Report of the Regional Coffee Wilt Program.CABI, 2009.

Poehlman, J.M., and D.A. Sleper, 1995.Breeding field crops.Iowa state university press, Ames, Iowa50014, India. 494p.

Rutherford, M.A., 2006. Current knowledge of coffee wilt disease, a major constraint to coffee production in Africa. Symposium on Fusarium induced diseases of tropical perennial crops. *Phytopathology*,**96**: 663–666.

Russel, A.J., 1978. Plant breeding for pest and disease resistance. Butterworth Ltd, London. 485p.

Rood, S.B., 1986.Heterosis and the metabolism of [3H] gibberellin A1 in maize. Canadian *journal of botany*, **64**(9):2160-2164.

Statistical analysis system (SAS), 2008.SAS user's guide (version 9.2).SAS Institute, Cary, NC., USA.

Seyoum, S., 2003.Genetic divergence for seedling parameters and associations among agronomic traits in the Ethiopian coffee (*Coffea arabica* L.) germplasm. An MSc thesis submitted to the School of graduate studies of Alemaya University. 91p.

Schlegel, R. H. J., 2003. Encyclopedic dictionary of plant breeding and related subjects.Food Products Press and the Haworth Reference Press, imprints of the Haworth Press, Inc. Alice Street, Binghamton. 563p.

Sharma, P. D., 2004. Plant pathology: a text book for university students. Rackesh Kumar Rastogi publications, New Delhi, India.

Shull, G.H., 1908. The composition of a field of maize.*Journal of Heredity*, **1**:296-301.

Shull, G.H., 1952. Beginning of the heterosis concept. pp. 14-48. In: J.W. Gowen (eds). Heterosis.Hafner publishing, New York.

Siddiqui, Z.A. and L. P. Singh, 2004.Effects of soil inoculants on the growth, transpiration and wilt disease of chickpea.*Journal of Plant Diseases and Protection*, **111**(2): 151-157.

Sihen, B.,A. Girma, L. Fikre and H. Hindorf,2012.Coffee wilt disease (*Gibberella Xylarioides* Heim and Saccas) in forest coffee systems of Southwest and Southeast Ethiopia.*Plant Pathol. J.* **11**(1): 10-17.

Singh, B.D., 1993. Plant breeding. Kalyani Pub. New Delhi, India. 896p.

Singh, R.K. and B. D. Chaudhary, 1985.Biometrical methods in quantitative genetic analysis.Kalyani publishers, New Delhi. 318p.

Sleper, D. A. and J. M. Poehlman, 2006.Breeding field crops. Wiley-Blackwell, India. 424p.

Sprague, G.F. and L.A. Tatum, 1942.General versus specific combining ability in single crosses of corn.*J. Am. Soc. Agron*. **34:** 923-932.

Stangland, G.R., W.A. Russel and O.A. Smith, 1983. Evaluation of the performance and selected lines derived from improved maize population. *Crop sci*.**18**:224-226.

Stewart, R. B., 1957. Some plant diseases occurring in Kaffa province. Imperial Ethiopian College of Agriculture and Mechanical Arts, Alemaya, Ethiopia.

Strange, R.N., 1993.Plant Disease Control.Towards environmentally acceptable methods. Chapman and Hall, New York.

Stuber, C. W., Lincoln, S. E., Wolff, D. W., Helentjaris, T. and E. S. Lander, 1992. Identification of genetic factors contributing to heterosis in a hybrid from two elite maize inbred lines using molecular markers. *Genetics*, **132**(3): 823-839.

Sylvian, P.G.,1955. Some observation on *Coffea arabica* L. in Ethiopian.*Turrialba*,5:37-53.

Toda, T., Y. Fujimoto, K. Nishimura, H. Yamate & T. Maeta, 1986. Resistance to the nematode in hybrid pines (*P. thunbergii* × *P. massoniana*). Proceedings of Kyushu
Branch Congress of Japanese Forest Society, **39**: 67–68.

Toda, T. & Y. Fujimoto, 1987. Resistance to the pine wood nematodes in *Pinus densiflora* (Kirishima-matsu).*The Japanese Forest Society Congress*, **98**:261–262.

Toole, E. R., 1941. Fusarium wilt of mimosa tree (*Albizia julibrisin*). *Phyopath*.**31**: 599-616.

Ulloa, M., R.B. Hutmacher, R.M. Davis, S.D. Wright, R. Percy and B. Marsh, 2006. Breeding for Fusarium wilt race 4 resistance in cotton under field and greenhouse conditions. *J. Cotton Sci*, **10**(11):114–127.

Van der Graaff, N. A. and R. Pieters, 1978. Resistance levels in *Coffea arabica* to *Gibberella xylarioides* and distribution pattern of the disease. *Neth. J.Pl. Pathol*.**84**: 117-120.

Van der Graaff, N.A., 1981. Selection for Arabica coffee types resistant to CBD in Ethiopia. A Doctoral Dissertation presented to Wageningen University, Netherland. 110pp.

Van der Graaff, N. A., 1983. Durable resistance in perennial crops. pp. 263-276. In: L. Lamberti, J. M. Waller and N. A. Van der Graaff (eds). Durable resistance to crops. Plenum Press, New York.

Vanderplank, J. E., 1984.Disease resistance in plants.Academic Press, INC. 194p.

Van der Vossen, H.A.M. and D.J.A. Walyaro, 1980. Breeding for resistance to coffee berry disease in Coffea arabica.II. Inheritance of the resistance. *Euphytica*, **29**:777-791.

Van der Vossen H. A. M. •and D. J. Walyaro, 2009.Additional evidence for oligogenic inheritance of durable host resistance to coffee berry disease (Colletotrichum kahawae) in Arabica coffee (coffea arabica L.).Euphytica. 165(1):105-111.

Van der Vossen, H.A.M., 1985. Coffee selection and breeding. pp. 49-96. In: M.N. Clifford and K.C. Willson (eds). Coffee: Botany, biochemistry and production of beans and beverages. Croom Helm Ltd, London.

Van der Vossen, H.A.M., 2005. A critical analysis of the agronomic and economic sustainability of organic coffee production.*Exper. Agri*. **41**: 449-473.

Verma, P.S .and V.K. Agarwal, 1982.Genetics.Chad,S. and Co. Ltd, Ramnagar, New Delhi. 580p.

Viands, D. R., 1985.Comparison of *Maris Kabul* with *Vertus* alfalfa for resistance to verticillium wilt.*Crop Sci*. **25**:1096-1100.

Walyaro, D.J.A., 1977. Pollen longevity and artificial cross pollination in *coffea arabica*.*Euphytica*, **26**: 25-29.

Walyaro, D.J.A. and H.A.M. Van der Vossen, 1977.Early determination of yield potential in Arabica coffee by applying index selection.*Euphytica*, **28**:465-472.

Walyaro, D.J.A., 1983. Considerations in breeding for improved yield and quality in Arabica coffee (*Coffea arabica* L.). Doctoral Thesis presented to Wageningen University, Netherland. 119pp.

Wassu, M., 2004. Heterosis and combining ability analysis of yield and yield related traits in coffee (*Coffea arabica* L.). An MSC Thesis presented to the school of graduate studies, Alemaya University. 92p.

Wintgens, J.N., 2009. Coffee: growing, processing, sustainable production. A guide book for growers, processors, traders and researchers.Wiley-Vch. 975p.

Workafes, W., and Kassu K., 2000.Coffee production system in Ethiopia. Proceedings of work-shop on control of coffee berry disease in Ethiopia. Addis Ababa, Ethiopia, 13-15 August 1999.

Yamanobe, T., 2009.Relationships between morphological traits and resistance to pine wood nematode in two Japanese pines.*Eur. J. Plant. Pathol*.**124**:543–552.

Yanchuk, A.D. 1996. General and specific combining ability from disconnected partial diallels of coastal Douglas-fir.*Silvae Genet*.**45**: 37–45.

Yonass, B., Bayetta, B. and F. Chemeda, 2014.Performance evaluation of indigenous Arabica coffee genotypes across different environments.*Journal of Plant Breeding and Crop Science*, **6**(11):171-178.

7. APPENDIX

Appendix table 1. Analysis of variance mean squares and probability levels for CWD and growth characters

Characters	Mean squares and p'		
	F1 crosses and parents		
	Block (df=2)	Genotypes (df=35)	Error (df=70)
Disease parameters			
Wilted coffee seedlings Percentage	1065.19** (801.08**)	1743.23** (823.29**)	204.48 (118.02)
Incubation period	397.34**	610.80**	52.69
Number of Defoliated leaves per seedling	8.22**	1.73**	0.66
Number of yellow leaves per seedling	1.58**	0.14^{ns}	0.10
Stem characters			
Seedling height (cm)	9.97**	2.62**	0.44
Seedling stem diameter (mm)	0.01^{ns}	0.022*	0.013
Number of node	0.03*	0.17**	0.009
Average inter node length (cm)	0.18^{ns}	1.16**	0.16
Leaf characters			
Number of leaves	2.85**	0.57**	0.18
Leaf area (cm^2)	24.11**	18.02**	1.80
Petiole length (cm)	0.0065**	0.0032**	0.0004

df= degree of freedom of block, genotypes and error, ** = highly significant at P <0.01, * = significant at p<0.05 and ns =non-significant Data in bracket is arcsine transformed value of wilted seedlings percentage