Ethiop. J. Agric. Sci. 31(4) 19-43 (2021)

Heterosis, Combining Ability and Heritability for Resistance to Coffee Wilt Disease in Arabica Coffee

Admikew Getaneh[*1], Girma Adugna[2], Sentayehu Alamerew[2]
[1]*Department of Coffee Breeding and Genetics at Jimma Agricultural Research Center, EIAR;*
E-mail: adamget21@gmail.com;[2]Department of Horticulture and Plant Science at College of Agriculture and Veterinary Medicine, Jimma University, Ethiopia

አህፅሮት

የቡና ምርትና ምርታማነት ከሚ ቀንሱ የቡና በሽታዎች መካከል የቡናግንድ አድርቅ በሽታ ከፍተኛውንድርሻ ይይዛል፡፡የቡና ግንድ አድርቅ በሽታ በተለያየመጠን የመቋቋም ችሎታ ያላቸው የቡና ጀኖታይፖች በመጠቀም የዘርውርስ ሂደቱን ወይም የመቋቋም ስነባህሪውንበማጥናት በሽታውን ለመከላከል የሚያስችል የመሻሻያ መንገዶችን ለመቀየስ ጥናት ማድረግ አስፈጊ በመሆኑ ይህ የምርምር ስራ ተካሂዷል፡፡ጥናቱ ስምንት የእናት ቡና ጀኖታይፖችንና ከነዚህ የተገኙ 28 የመጀመሪያ ድቃዮችን እንዲሁም አንድ በበታሽው የሚጠቃ ማነፃፀሪያ በመጠቀም ተካሂዷል፡፡ በ2007/8 ዓ.ም በጥናቱ ላይ የተካተቱት የቡና ጀኖታይፖች ለበሽታው ያላቸውን የመቋቋም ችሎታ በጅማ ግብር ምርምር የእፅዋት ጥበቃ ግሪን ሃውስና ላብራቶሪ ውስጥ በመከተብ ተገምግመዋል፡፡ በሽታውን በተከተቡ ችግኞች ላይ የደረቁ የቡና ችግኖች ብዛት በፐርሰንት፤ የበሽታ ምልክት የሚታይበት የጊዜ ርዝመት፤ በበሽታው የረገፉና ወደ ቢጫነት የተለወጡ የቅጠል ብዛት መረጃዎችን በመውሰድ የተለያዩ ስታስቲካዊ ዘዴዎችንና ፓኬጆችን በመጠቀም የመረጃ ትንተና ተደርጓል፡፡ በዚህ ጥናት መሰረት የደረቁ ችግኖች ብዛት በመቀነስ፤ የበሽታ ምልክት የሚታይበት የጊዜ ርዝመት በማሳጠርና በበሽታው የሚረግፉ የቅጠል ብዛት በመቀነስ ረገድ ያላቸው የሃይ-ድቅል መጠን በሽታውን ሊቋቋም በሚየስችል ሁኔታ ወይም በሚፈለገው ደረጃ መሻሻል አላሳየም፡፡እናት ቡናዎች P_2(971)፤ P_7(974)፤ P_8(370 እና P_5(79233) በሽታ የመቋቋም ባህሪያቸውን ለድቃዮቻቸው በማስተላለፍ ከፍተኛ የሆነ ድርሻ እንዳላቸው ታውቋል፡፡ በተመሳሳይ ሁኔታ P_7 x P_8 (974x370) እና P_4xP_8 (8136x370) ድቃዮች በጥሩ ሁኔታ በሽታውን የመቋቋም አቅም አሳይተዋል፡፡ በተጨማሪም ይህን በሽታ የሚቋቋሙ የቡና ጀኖታይፖች የመቋቋም ስነባህሪያቸው እስከ 68.61% የመሻሻልና የመተላለፍ አቅም ከፍተኛ መሆኑ በጥናቱ ተረጋግጧል፡፡ በአጠቃላይ በዚህ ጥናት መሰረት የቡና ግንድ አድርቅ በሽታን የመቋቋም ስነባህሪን በመረጣና ከድቀላ በኋላ የዘር ሀረግን መሰረት አድርጎ መረጣ በማካሄድ ማሻሻል እንደሚቻል ታውቋል፡፡

Abstract

Combining ability, heterosis and heritability studies can provide valuable information for designing appropriate breeding programs for resistance to coffee wilt disease (CWD), which caused by Gibberella xylarioides. The objective of this study was conducted to determine heterosis, combining ability, and heritability for resistance to CWD using an eight-parent half diallel cross (eight parents and 28 F_1 hybrids). A susceptible control was used as a reference. All entities were artificially inoculated by the pathogen, and evaluated for CWD in the greenhouse at Jimma Agricultural Research Center (JARC), Ethiopia in 2015/16. The reactions of inoculated genotypes were measured as a percent of wilted seedling, incubation period, and number of yellow and defoliated leaves. Combined analysis of variance showed significant difference among genotypes for the characters measured. Better-parent heterosis (BPH) and mid-parent heterosis (MPH) for percent of wilted seedlings and the number of defoliated leaves showed negligible heterosis in desirable direction. However, considerable MPH was noticed for longer incubation period. Both additive and non-additive gene actions were involved in controlling the inheritance of CWD resistance and incubation period; the additive gene effects being predominant. Parents P_2 (971), P_7 (974), P_8 (370), and P_5 (79233) showed highly significant negative general combining ability (gca) *effects and found to be good general combiners for resistance to CWD. Moreover, specific combining ability (sca) effects of hybrids P_7 x P_8 (974 x 370) and P_4 x P_8 (8136 x 370) revealed that they are good combinations for resistance (low mean wilted seedlings percentage) and incubation period. Percent wilted seedlings showed high broad (88.27%) and narrow (75.41%) sense heritability coupled with 68.61% genetic advance. Generally, both pure line selection and pedigree selection after hybridization could be an effective resistance breeding approach for CWD management in Arabica coffee.*

Keywords: Arabica coffee, *Coffea arabica*, coffee wilt disease, combining ability, gene effects, *Giberella xylarioides*, heritability, heterosis

Introduction

Coffee is a stimulant, woody perennial evergreen dicotyledonous plant. A mature coffee tree consists of a shoot and root systems; flowers are white and fragrant (Hadberg *et al.*, 2003; Wintgens, 2009).Arabica coffee (*Coffea Arabica* L.)is the only known tetraploid (2n=4x=44 chromosome number) and auto-gamous species in the genus Coffea. While, all other coffee species are diploid (2n=22) and self-incompatible (Charrier and Berthaud, 1985; Lashermes *et al*., 1999). Southwestern Ethiopia is the primary center of origin and genetic diversity of Arabica coffee (Anthony *et al*., 2001 and 2002). However, productivity of the crop is low due to traditional production systems, use of local genotypes, presence of abioticstresses, poor agronomic practices and widespread of coffee diseases such as, coffee berry disease (CBD), coffee leaf rust (CLR) and coffee wilt disease (Melaku, 1984; Eshetu, 1997; Eshetu *et al.,* 2000; Girma *et al.,* 2009a).

Coffee wilt disease (CWD) is a fungal vascular disease caused by *Gibberella xylarioides* (*Fusarium xylarioides*) (Heim and Saccas, 1950; Geiser *et. al*., 2005). The fungus invades coffee treesand colonizes the xylem system. Successive survey on the occurrence and prevalence of *Gibberella xylarioides* in major coffee growing regions ascertained the existence of the disease with varying intensities (Merdassa, 1986; Girma, 1997; Girma *et al.*, 2001; Sihen *et al.*, 2012). Reports showed that there were variations in the incidence of CWD between coffee genotypes at fields that attributed to differences in their genetic background, age of coffee trees, cultural practices and environmental condition at a specific location. Generally, the prevalence and importance of the disease has been markedly increasing throughout coffee producing areas of the country (Girma *et al.*, 2001; Girma, 2004). In Ethiopia, the national incidence and severity of CWD is about 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 practices are uprooting and burning of infected coffee trees, prevention of tree wounding, use of protective fungicides, use of disease free planting materials, disinfecting farm implements and use of biological control. However, these methods are difficult to implement; and use of resistant varieties is the most cost-effective and eco-friendly method for controlling the disease (Rutherford, 2006; Phiri and Baker, 2009; Girma *et al.*, 2009a). According to Girma *et al.* (2005), there were highly significant differences between genotypes, *Gibberella xylarioides* isolates and genotype-isolate interactions in seedling test; suggesting the presence of qualitative (vertical) with predominance of quantitative (horizontal) resistance.

Knowledge about the genetic control of CWD resistance and related traits in Arabica coffee is useful in planning breeding programsfor this economically important crop. Estimationsof combining ability and heterosis are important parts of crop breeding to understand the inheritance controlling mechanism of different traits, and improve disease resistance. It also 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). Estimate of heritability along with genetic advance and the association between the traits are also important selection parameters to select the required traits (Panwar *et al.*, 2015). In line with this, Musoli *et al.* (2013) have investigated the inheritance of resistance to CWD in Robusta coffee using partial diallel crossing and they reported that the gene controlling resistance is polygenic; and itsheritability is low to moderate. They have concluded that it is difficult to derive hybrid populations with such parental lines and breeding for CWD resistant is possibly through selecting tolerant clones.

Despite extensive work had done to manage CWD; the inheritance of resistance controlling mechanism in Arabica coffeeis unknown. Therefore, the present study was conducted to estimate combining ability, heterosis, heritability and the type of gene effects controlling the inheritance of resistance to CWD, which is useful in designing appropriate breeding program.

Materials and Methods

Coffee genotypes and experimental design

The study was conducted in a greenhouse at Jimma Agricultural Research Center (JARC) in Southwest Ethiopia. Eight Arabica coffee parents, namely 75227 (P_1), 971 (P_2), 74110 (P_3), 8136 (P_4), 79233 (P_5), Arbagugu (P_6), 974 (P_7), 370 (P_8) and one susceptible control (Geisha) were selected based on their CWD resistance level under greenhouse and field conditions.The parental lines were selected from three CWD reaction groups that were identified as resistant (P_2, P_5 and P_7), moderately resistant (P_8, P_4 and P_6) and susceptible (P_1 and P_3) (Table 1). The eight-parents were crossed in an 8 x 8 half diallel mating design using Griffing (1956) method 2 and model I in the breeding blocks at Gera Agricultural Research Sub Center, Ethiopia.

Two to three uniformly grown coffee trees were identified from each genotype before flowering (blooming stage). Then, healthy branches with sufficient flower buds were selected, selfed, crossed and labeled in February 2014.After harvesting the seeds and raising seedlings, 28 F_1hybrids along with eight parents and one susceptible control were inoculated and evaluated for disease reaction in a greenhouse in 2015/16. The experiment was laid out in randomized complete block design (RCBD) with three replications.

Table1. Description of Arabica coffee genotypes selected for studying inheritance of resistance to coffee wilt disease

Parental lines	Coffee genotypes	Origin	Reaction to coffee wilt disease (CWD) and other desirable traits
P_1	75227	Gera, Jimma	Susceptible to CWD, CBD resistant and good yielder (Girma and Chala, 2008; Demelash and Kifle, 2015)
P_2	971	Gelana Abaya, Borena	Resistant to CWD (Jefuka et al., 2012)
P_3	74110	Metu, Illubabor	Susceptible to CWD, resistant to CBD and good yielder (Demelash and Kifle, 2015)
P_4	8136	Gera, Jimma	Moderately resistant to CWD, resistant to CBD and CLR (Girma and Chala, 2008)
P_5	79233	International collection	CWD resistant in naturally infested soil (personal observation)
P_6	Arbagugu	Metu, Illubabor	Moderately resistant to CWD in naturally infested soil, susceptible to CBD (personal observation)
P_7	974	Gelana Abaya,Borena	Resistant to CWD (Jefuka et al., 2012)
P_8	370	Seka-Chekorsa, Jimma	Resistant to CWD, susceptible to CBD (Girma and Chala 2008; Demelash, 2013)
Susceptible control	Geisha	International collection	Highly susceptible to CWD (Girma and Chala, 2008; Demelash, 2013)

Source: JARC / Coffee Breeding and Genetics division database

Seedling raising and inoculums preparation

After removing the parchment, fresh seeds of each Arabica coffee genotype were soaked in distilled sterile water for about 48 hours. Then, forty seeds of each genotype were sown indisinfected plastic pots (each has 5652 cm^3 capacity), which consists ofheat sterilized and moistened sandy soil (Girma and Mengistu, 2000). Sterile water was applied at a day interval to maintain adequate moisture for seed germination and seedling growth. After germination, the seedlings were thinned to twenty-five seedlings per pot (20 seedlings were used for artificial inoculation test and the remaining five seedlings used as a control in each pot).

The five non-inoculated seedlings in each pot were not infected by the pathogen until the end of the experiment.

A representative and aggressive Gera isolate of *Gibberella xylarioides* was taken and multiplied for inoculation using the method of Pieters and Van der Graaff (1980) with some amendments (Girma and Mengistu, 2000). The spore concentration was counted with haemo-cytometer, and adjusted to 2 x 10^6 conidia per ml (Girma *et al.*, 2009b).

Seedlings inoculation, management and disease assessment

Twenty coffee seedlings per pot for each genotype were inoculated at fully opened cotyledon stage (10 weeks old) with viable conidial suspension of *Gibberella xylarioid*es by stem nicking technique (Pieters and Van der Graaff, 1980; Girma and Mengistu, 2000). The treated plants were immediately kept in an air-conditioned growth room with high relative humidity (>95%) and optimum temperature (23±2^{o}C) for infection. After 10 days, the inoculated seedlings were transferred to greenhouse with a temperature of 25±4^{o}C and 60-80% relative humidity (Girma *et al.*, 2009b).

Data collection

An effective and reliable method of quantifying resistance was applied for comparison of results and selection of resistant genotypes. Percentage of dead (wilted) seedlings was computed as the number of infected (wilted) plants that recorded based on external symptoms over the total number of inoculated plants per pot multiplied by 100 to determine the relative resistance among genotypes (Girma and Mengistu, 2000; Girma *et al.*, 2009b; Musoli *et al.*, 2009). Incubation period in number of days, and the amount of defoliated and yellow leaves per seedling were also recorded. Re-isolation of the fungus was carried out that confirm seedlings death was caused by the inoculated isolate.

Statistical analysis

Mean values of data collected from five randomly taken seedlings from each pot were subjected to analyses of variance (ANOVA) using SAS program version 9.2 (SAS,2008). Least significant difference (LSD) test was used to compare treatment means. The analysis was carried out according to the following model.

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

Where, Y is the response variable corresponding to treatment i^{th} measure on block j^{th}, b_i is the effect of i^{th} replication, g_j is the effect of j^{th} genotype and e_{ijk} is the residual term.

Estimate of variance components

Percent wilted or dead seedlings were calculated from cumulative number of wilted over total number of seedlings (wilted plus healthy) for a total recording during six month.

Wilted seedling Percentage (%) = $\frac{Cumulative \quad number \quad of \quad wilted \quad seedlings}{Total \quad number \quad of \quad seedlings \quad (wilted \quad plus \quad healthy)} * 100$

The phenotypic, genotypic and environmental variances were estimated based on the method suggested by Singh and Chaudhury (1985). Heritability and geneticadvance were also estimated according to Allard (1999) method.

Heterosis

Heterosis of CWD traits were estimated following the formulae suggested by Falconer and Mackay (1996);

Mid parent heterosis = $\left[\frac{F_1 - MP}{MP}\right] * 100$

Heterobeltisois (Better parent heterosis) = $\left[\frac{F_1 - BP}{BP}\right] * 100$

Susceptible control (SC)heterosis = $\left[\frac{F_1 - SC}{SC}\right] * 100$

Susceptible parent (SP) heterosis = $\left[\frac{F_1 - SP}{SP}\right] * 100$

The standard error of the difference for heterosis was calculated as follows: SE (d) for MP = $\pm \sqrt{\frac{3Me}{2r}} * t$

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

Where, F_1 is the mean value of the hybrid, MP denotes the mean of the two parents producing the F_1, BP denotes the better parent mean value, SE (d) is standard error of the difference, Me is error mean square, r is number of replicationsand t is the value at error degree of freedom.

Test of significance for heterosis was done by comparing (F_1-MP) with SE (d) for mid parent, (F_1 -BP) with SE (d) for better parent, (F_1 -SP) with SE (d) for susceptible parent and (F_1 -SC) with SE (d) for susceptible control heterosis. The minimum values were considered as better parent in the case of wilted seedling percentage and number of defoliated leaves.

Combining ability analysis

Disease data collected from F_1 generations and selfed parental lines 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 hybrid control the results. Combining ability was computed using the following mathematical model;

$$Y_{ij} = \mu + g_i + g_j + s_{ij} + \frac{1}{bc}\sum_k \sum_l e_{ijkl}$$

Where, $Y_{ij}$is the value of a trait measured on hybrid of i^{th}andj^{th} parents, μ = overall mean, g_i, g_j are the general combing ability effect of the i^{th} and j^{th} parents, respectively, S_{ij} = the specific combing ability effect of the hybrid i x j, $\frac{1}{bc}\sum_k \sum_l e_{ijkl}$ = the mean error effect of the $ijkl^{th}$ observation and n, b and c are number of parents, blocks and sampled plants, respectively.

GCA and SCA sum squares, mean squares, general combining ability effect (g_i) and specific combining ability effect (s_{ij}) were estimated using the equation developed by Griffing (1956):

$$g_i = \frac{1}{n+2}\left(Y_{i.} + Y_{ii} - \frac{2}{n} Y_{..}\right)$$

$$s_{ij} = Y_{ij} - \frac{1}{n+2}\left[Y_{i.} + Y_{ii} + Y_{.j} + Y_{jj}\right] + \frac{2}{(n+1)(n+2)} Y_{..}$$

Where, $Y_{i.}$and$Y_{.j}$ are mean of the i^{th} and j^{th} parents, respectively, Y.. is grand mean, n is number of parent lines

The relative size of variances due to GCA and SCA for model I was computed using the formula developed by Singh and Chaudhury (1985);

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

Results and Discussion

Analysis of variance

Results of the analysis of variance showed that the difference among genotypes was highly significant (p<0.01) for wilted seedling percentage, incubation period and number of defoliated leaves (Table 4). However, number of yellow leaves exhibited non-significant differences. On the other hand, F_1 hybrids showed significant differences (p<0.05) for number of defoliated and yellow leaves per seedling. All disease parameters (except number of yellow leaves) showed significant differences among parental lines. This result confirmed the existence of genetic diversity between the parental lines and F_1 hybrids for CWD traits (Figure 2); meeting the prerequisites for detail genetic analysis as suggested by Griffing (1956).

Mean performance of parents and F_1 hybrids

The mean performance of F_1 hybrids, parental lines and susceptible control for CWD traits are summarized in Table 2. Percentage of wilted seedlings ranged from 25.1% for tolerant (resistant) parent P_2 to 91.4% for the susceptible parent P_3; and from 20.6% for tolerant (resistant) hybrid P_7x P_8 to 90.7% for susceptible hybrids P_1 x P_6 and P_1 x P_8.Thehybrids showed relatively wider range of percentage death compared to the parents, butonly one hybrid (P_7 x P_8) exhibited lower proportion of wilted seedlings than did the resistant parent (P_2). Parental line P_2 followed by P_5, P_7, P_8, and hybrids P_7 x P_8, P_2 x P_7, P_4 x P_8, P_2 x P_8, P_2 x P_5, P_5 x P_8, P_4 x P_7 and P_5 x P_7 exhibited relatively higher survival rate or lowermean wilted seedling percentage (more CWD resistance). In contrast, parental lines P_3, P_6, P_1, and hybrids P_1 x P_6, P_1 x P_8, P_1 x P_3, P_1 x P_5 and P_3 x P_6 showed the highest wilting percentage (highly susceptible).

In general, the mean performance of coffee genotypes showed that parents P_2, P_5, P_7 and P_8 had relatively lower proportion of wilted seedlings (CWD resistant), longer incubation period and minimum number of defoliated leaves; indicating the potential to transfer their genetic constitutions to the resulting hybrids. In agreement with this, various investigators reported for CWD tolerance both at seedling stage and in mature plants (Girma, 2004; Girma *et al.*,2005; Arega, 2006; Sihen *et al*., 2012; Demelash and Kifle, 2015). Similarly, Jefuka *et al.* (2012) have reported that release coffee varieties Feyate (971) and Odicha (974) were considered as CWD resistant. Demelash (2013) has also reported that 370-genotype showed resistant reaction to CWD although this finding contradicts with the current results. In this study, genotypes with resistant reaction had longer incubation period; while susceptible reaction was expressed by early development of wilting symptom and death. Girma and Chala (2008) and Kifle *et al.* (2015) have also reported the positive relationship of CWD resistance with extended incubation period. Among the hybrids, P_7 x P_8 showed the lowest mean percentage of wilted seedlings with the longest incubation period (143 days). The result of the present study indicated that when resistant parents hybridized with each other or with moderately resistant ones, it is most likely to get resistant and moderately resistant progenies; while susceptible parents hybridized with any CWD reaction groups (resistant, moderately resistant or susceptible parents) would give susceptible progenies. Therefore, this implies that genes governing susceptibility might be partially or completely dominant over the resistant genes in the inheritance of CWD resistance. The mean percentage of wilted seedlings ranged from 20.6% to 91.4%; suggesting that the traits showed continuous variation or is quantitative in nature.

Mean incubation period ranged from 89.7 to 133.0 days for parents and 96.3 to 143.0 days for F_1 hybrids. Accordingly, the incubation period in P_5 was the longest (133 days) compared to other parents, and stood fourth among all genotypes. The top three hybrids that showed prolonged incubation period were P_7 x P_8 (143.0 days), P_2 x P_4 (137.7 days), and P_4 x P_8 (136.7days). Conversely, hybrids P_1 x P_3 (96.3 days), P_3 x P_8 (97.0 days), P_1 x P_3 (98.3 days) and P_1 x P_2 (99.0 days) showed early disease symptoms. Therefore, significant differences for incubation period also indicate the existence of variability among Arabica coffee genotypes for *Gibberella xylarioides* reaction. This might be due to differences in host (coffee genotypes) defensive ability against the disease.

Number of defoliated leaves also showed significant differences among the genotypes; but the difference due to number of yellow leaves was statistically non-significant. Parental lines P_2, P_8, P_7 and P_5 had few, while P_6, P_3 and P_4 showed large numbers of defoliated leaves; the overall mean value of which was also higher for the F_1 hybrids than for the parents.

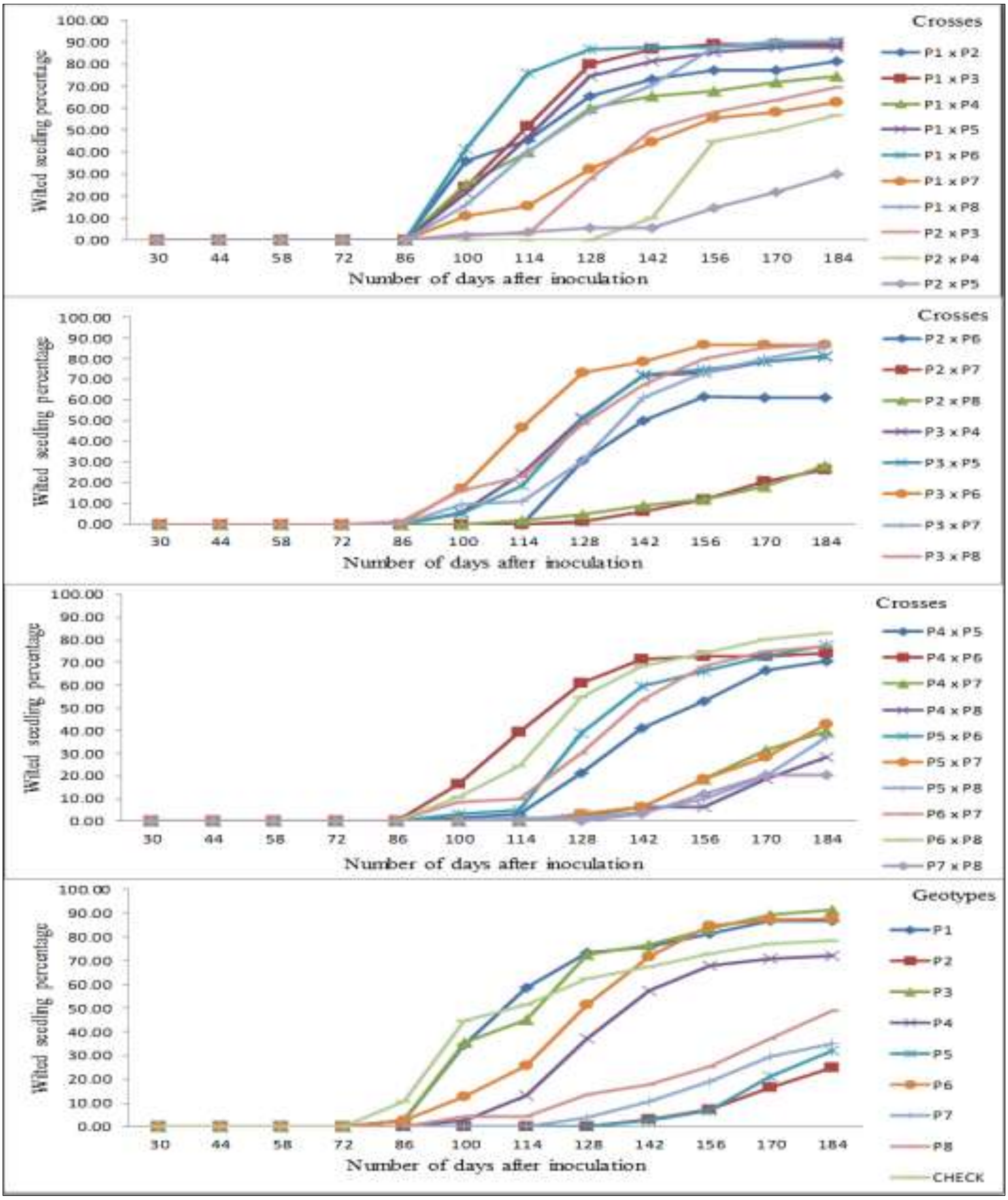
Consecutive measurements for mean proportion of wilted seedlings showed variable responses to *Gibberella xylarioides* (Figure 1 and 2). It was observed that the genotypes had variable levels of resistance and progressed at varying rate after infection. Parental lines P_2, P_5 and P_7, and F_1 hybrids P_7x P_8, P_2x P_7, P_4 x P_8, P_2x P_5, P_2 xP_8, P_4 x P_7, P_5 x P_8 and P_5 x P_7 showed late disease infection and low percent of disease progress in six month of assessment (12 times recorded at 14 days' interval). The rate of development of the disease appeared to be lower in these genotypes until four months after inoculation when high number of seedlings started wilting with increasing disease severity for most genotypes. Increased severity of the disease with time may be due to well establishment of the pathogen, production of micro and macro conidia, and mycelium and spores to colonize the host tissue and hinder the normal physiological processes. Therefore, genotypes that show late symptoms expression and low proportion of wilted seedlings are important for further hybridization or breeding program in order to manage CWD through resistance variety development.

Table2. Mean performance of Arabica coffee parents, F_1 hybrids and susceptible control for CWD traits.

Genotypes	WS (%)	IP(Days)	NDL	Genotypes	WS (%)	IP(Days)	NDL
Parents				Hybrids			
P_1	86.7a	91.3ij	1.96b-i	P_2 x P_7	26.2hi	123.0cd	0.93hi
P_2	25.1i	118.3de	0.78i	P_2 x P_8	28.5hi	123.0cd	0.89hi
P_3	91.4a	91.7hij	2.69a-d	P_3 x P_4	81.0abc	101.7ghi	2.42a-f
P_4	72.0a-e	107.7efg	2.18b-h	P_3 x P_5	81.3abc	105.7fg	2.09b-i
P_5	32.2hi	133.0abc	0.96hi	P_3 x P_6	86.7a	101.7ghi	2.82abc
P_6	87.5a	89.7j	3.58a	P_3 x P_7	85.3ab	103.3gh	1.98b-i
P_7	35.2ghi	115.3def	0.93hi	P_3 x P_8	86.6a	97.0g-j	2.47a-e
P_8	49.0e-h	126.7bcd	0.80i	P_4 x P_5	70.8a-e	116.0def0	1.64c-i
Mean	**59.9**	**109.2**	**1.73**	P_4 x P_6	74.3a-d	101.0g-j	2.04b-i
Hybrids				P_4 x P_7	39.6f-i	132.3abc	1.40d-i
P_1 x P_2	81.3abc	99.0g-j	1.89b-i	P_4 x P_8	28.2hi	136.7ab	1.11f-i
P_1 x P_3	89.3a	98.3g-j	2.91abc	P_5 x P_6	77.7a-d	108.0efg	1.82b-i
P_1 x P_4	74.7a-d	96.3g-j	2.20b-h	P_5 x P_7	42.7f-i	123.0cd	1.02ghi
P_1 x P_5	88.0a	102.0ghi	2.28a-g	P_5 x P_8	37.0ghi	126.7bcd	0.84i
P_1 x P_6	90.7a	101.7ghi	2.98ab	P_6 x P_7	76.9a-d	108.0efg	2.40a-f
P_1 x P_7	62.7b-f	108.0efg	1.16e-i	P_6 x P_8	82.9abc	107.3efg	2.00b-i
P_1 x P_8	90.7a	100.0g-j	2.87abc	P_7 x P_8	20.6i	143.0a	0.89hi
P_2 x P_3	69.5a-e	117.3def	1.98b-i	Mean	**65.0**	**112.7**	**1.89**
P_2 x P_4	56.9d-g	137.7ab	1.98b-i	Control	78.33	85.00	2.00
P_2 x P_5	29.8hi	120.0d	1.27e-i	LSD (0.05)	**23.29 (17.69)**	**11.82**	**1.32**
P_2 x P_6	61.3c-f	118.0de	2.62a-d	CV (%)	**22.38 (19.84)**	**6.49**	**43.64**

P_1=75227, P_2=971, P_3=74110, P_4=8136, P_5=79233, P_6=Arbagugu, P_7= 974 and P_8=370

IP = incubation period; NDL= number of defoliated leaves per seedling; WS%= Wilted coffee seedling percentage; Number of defoliated leaves and yellow leaves were measured at 4 months and wilted seedling percentage was recorded at 6 months after artificial inoculation. CV and LSD value in bracket is arcsine-transformed value of wilted seedling percentage.Figures followed by same letter(s) are not significantly different and P = 0.05.



P_1=75227, P_2=971, P_3=74110, P_4=8136, P_5=79233, P_6=Arbagugu, P_7= 974 and P_8=370

Figure 1. Percentage wilted seedlings of Arabica coffee genotypes at different times after inoculation.

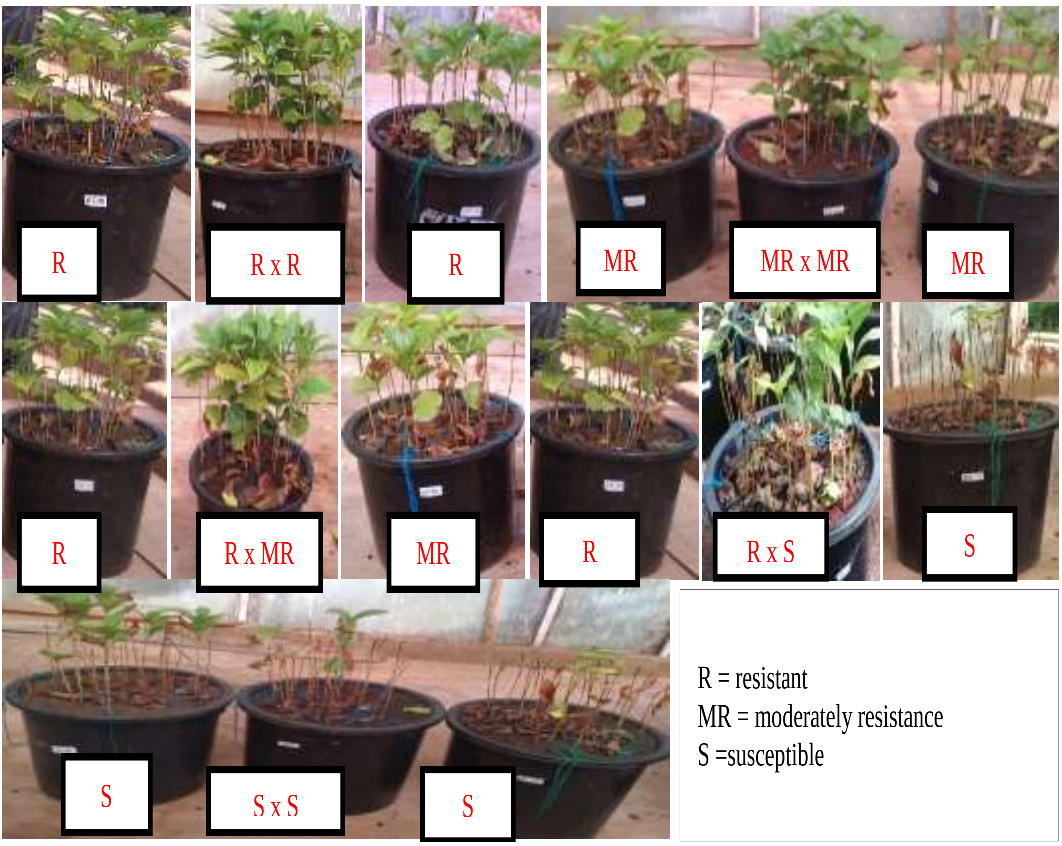


Figure 2. Comparison of Arabica coffee genotypes (parents and hybrids)reaction to CWD under greenhouse condition

Heterosis

Percentage of better-parent heterosis (BPH), mid-parent heterosis (MPH), susceptible-parent heterosis (SPH) and susceptible-check heterosis (SCH) for percentage of wilted seedlings, number of defoliated leaves and incubation period are presented in Table 3, 4 and 5. BPH ranged from -42.49% to 224.17% with +66.70% overall mean value for wilted seedlings percentage. It was observed that 14 hybridsexpressed positive and significant undesirable heterosis. Although, no hybrid showed significantly negative BPH, hybrids P_4 x P_8 and P_7 x P_8 manifested desirable effects. Heterosis for negative traits like disease, smaller values (negative values) are desirable for resistance. However, in this study, about 50% of the F_1 hybrids exhibited positive and significant BPH; probably due to lack of dominance of resistance, which could also be masked by the harmful effect of susceptible genes in controlling the inheritance of resistance.

MPH for percent wilted seedlings ranged from -53.42% (P_4 x P_8) to + 48.08% (P_1 x P_5). Out of 10 negative heterosis, only two hybrids (P_4 x P_8 and P_7 x P_8) showed significant ($p<0.01$and/or $p<0.05$); while four hybrids (P_1 x P_5, P_1 x P_2, P_3 x P_7 and

P_1 x P_8) exhibited significantly positive MPH. The values of SPH and SCH ranged from -60.86% (P_4 x P_8) to +21.31% (P_5 x P_7) and from -73.52% (P_1 x P_6 and P_1x P_8) to +15.75% (P_7 x P_8), with five and eight hybrids depicting significantly negative heterosis (favorable effect), respectively. This result suggests, hybrids that showed negative mid parent, susceptible parent and susceptible check (control) heterosis were desirable for resistance.

The value of BPH and MPH for number of defoliated leaves ranged from -6.13 % to +258.75%, and from -28.97% to +108.22%, respectively. All hybrids (except hybrid P_4 x P_6) showed positive BPH (unfavorable effects). Additionally, all hybrids (except for hybrid P_1 x P_8 that showed significantly positive response) had non-significant MPH; with nine hybrids manifesting negative, but 19 hybrids positive values. On the other hand, 20 hybrids expressed negative SPH, and all hybridsshowed non-significant SCH. Moreover, P_4 x P_6, and P_4 x P_6, P_4 x P_8 and P_5 x P_6 were found to be the most favorable hybrids with desirable effects for BPH and MPH, respectively. Generally, about 96% and one third of the hybrids of BPH and MPH, respectively, expressed with undesirable effects for number of defoliated leaves.This could be related to effectiveness of some genes responsible for the production of hormones, such as abscisic acid (ABA), that favor abscission of leaves during host pathogen interaction.

For incubation period, BPH ranged from -23.42 % (P_3 x P_8) to +16.34 % (P_2 x P_4) with -4.92% overall mean. Positive and significant MPH was observed in eight hybrids (desirable direction), although only hybrid P_3 x P_8 showed significantly negative heterosis. Both BPH and MPH results exhibited that P_2 x P_4, P_4 x P_7, and P_7 x P_8 were superior with significantly positive values in the order of desirable magnitude for incubation period. Moreover, all F_1 hybrids displayed positive SPH and SCH; with 13 and 27 hybrids showed significant heterosis, respectively.

Most hybrids revealed undesirable and insignificant BPH and MPH for percent wilted seedlings and number of defoliated leaves (no hybrid exhibited significant desirable heterosis). However, for incubation period three and eight hybrids (about 29% of the hybrids) manifested significantly positive BPH and MPH, respectively. Some hybrids also showed longer incubation period of incubation than did any one of the parents. Therefore, this result indicates that the existence of probably partial to complete dominance of genes for incubation period in favorable direction. Relatively smaller or negative MPH (favorable effect) was detected for hybrids that had less mean percentage of wilted seedlings. Conversely, hybrids that expressed heterosis in favorable direction are not always advantageous. Because, some hybrids, such as P_3 x P_6 showed favorable BPH and MPH for percent wilted seedlings and incubation period, but their mean values showed susceptibility and shorter incubation period. Unexpectedly, in most cases MPH resulting from hybrids of susceptible parents with resistant parents had more positive response than did susceptible parents hybridized with susceptible or moderately resistant parents. For instance, more positive MPH was manifested and obtained parent P_1 (susceptible parent) hybridized with P_2 (resistant parent) than P_1 hybridized with P_3 or P_6 (susceptible parents). This result is due to that the differences between F_1 hybrids mean of the two susceptible parents and their parental average mean was lower than the difference between F_1 hybrid mean of the susceptible and resistant parents and their parental average mean based on Falconer and Mackay (1996) formal. In addition, when resistant and susceptible parents used in heterosis estimation, the mid parents mean value became lowered; while MPH increased in reverse.

Generally, heterosis was small (not appreciable) for CWD resistance improvement in genotypes considered in the present study. Consequently, the use of heterosis breeding may be rarely essential and, if it is necessary, both parents should be wilt resistant or moderately resistant. Hence, selection of parents could be an effective method for improvement. In line with the present finding, Patel and Pathak (2011) studied the genetics of resistance to wilt in castor bean hybrids and reported that heterosis breeding with a choice of superior parents would be advantageous for enhancing wilt resistance along withyield. The present finding also showed similarity to the results of Mesfin (1982) and Bayetta (2001) on CBD resistance.

Table 3. Estimate of heterosis percentage for percent wilted seedlings (%), incubation period and number of defoliated leaves

Hybrids	Wilted seedlings (%) percentage			
	BPH	MPH	SPH	SCH
P_1 x P_2	224.17**	45.55*	-6.15	3.83
P_1 x P_3	3.08	0.33	-2.28	14.05
P_1 x P_4	3.70	-5.88	-13.85	-4.68
P_1 x P_5	173.37**	48.08**	1.54	12.35
P_1 x P_6	4.62	4.10	3.59	15.75
P1 x P_7	77.92*	2.83	-27.69*	-20.00
P_1 x P_8	85.03**	33.66*	4.62	15.75
P_2 x P_3	176.78**	19.21	-24.04	-11.34
P_2 x P_4	126.96**	17.30	-20.91	-27.30
P_2 x P_5	18.94	4.19	-7.30	-61.9**
P_2 x P_6	144.20**	8.82	-29.99*	-21.78
P_2 x P_7	4.55	-13.01	-25.52	-66.51**
P_2 x P_8	13.47	-23.15	-41.90	-63.65**
P_3 x P_4	12.50	-0.87	-11.40	3.41
P_3 x P_5	152.66**	31.60	-11.03	3.83
P_3 x P_6	-0.98	-3.13	-5.20	10.64
P_3 x P_7	142.27**	34.76*	-6.66	8.94
P_3 x P_8	76.64**	23.28	-5.32	10.50
P_4 x P_5	119.79**	35.81	-1.74	-9.68
P_4 x P_6	3.16	-6.87	-15.13	-5.17
P_4 x P_7	12.36	-26.18	-45.04**	-49.48**
P_4 x P_8	-42.49	-53.42**	-60.86**	-64.02**
P_5 x P_6	141.26**	29.75	-11.26	-0.85
P_5 x P_7	32.73	26.76	21.31	-45.45**
P_5 x P_8	14.99	-8.82	-24.46	-52.74**
P_6 x P_7	118.45**	25.37	-12.08	-1.77
P_6 x P_8	69.21**	21.46	-5.27	5.85
P_7 x P_8	-41.64	-51.19*	-58.05*	-73.76**
mean	**66.7**	**7.87**	**-15.79**	**-16.97**
SE(±)	**11.68**	**10.11**	**11.68**	**11.68**

P_1=75227, P_2=971, P_3=74110, P_4=8136, P_5=79233, P_6=Arbagugu, P_7= 974 and P_8=370

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

Table4. Estimate of heterosis percentage for number of defoliated leaves.

Hybrids	Number of defoliated leaves			
	BPH	MPH	SPH	SCH
P_1 x P_2	142.31	38.12	-3.41	-5.50
P_1 x P_3	48.72	25.25	8.18	45.50
P_1 x P_4	12.27	6.29	0.92	9.83
P_1 x P_5	138.32	56.52	16.35	14.00
P_1 x P_6	52.30	7.71	-16.68	49.00
P1 x P_7	23.94	-19.95	-41.05	-42.17
P_1 x P_8	258.75**	108.22*	46.51	43.50
P_2 x P_3	153.42	13.93	-26.52	-1.17
P_2 x P_4	153.42	33.71	-9.19	-1.17
P_2 x P_5	62.82	46.25	32.75	-36.50
P_2 x P_6	236.32**	20.43	-26.66	31.17
P_2 x P_7	19.65	8.95	0.00	-53.34
P_2 x P_8	13.68	12.24	10.84	-55.67
P_3 x P_4	11.33	-0.41	-9.91	21.17
P_3 x P_5	118.46	14.62	-22.30	4.50
P_3 x P_6	4.83	-10.00	-21.16	41.00
P_3 x P_7	111.80	9.11	-26.52	-1.17
P_3 x P_8	208.34*	41.36	-8.30	23.34
P_4 x P_5	71.77	4.89	-24.62	-17.84
P_4 x P_6	-6.13	-28.97	-42.96*	2.16
P_4 x P_7	50.01	-9.97	-35.83	-30.00
P_4 x P_8	38.75	-25.42	-49.16	-44.50
P_5 x P_6	89.93	-19.71	-49.12**	-9.00
P_5 x P_7	9.64	8.29	6.94	-48.84
P_5 x P_8	5.41	-3.99	-11.81	-57.84
P_6 x P_7	157.15*	6.43	-32.96	20.00
P_6 x P_8	150.00	-8.61	-44.13*	0.00
P_7 x P_8	11.25	2.69	-4.64	-55.50
mean	**83.87**	**12.07**	**-13.73**	**-5.54**
SE(±)	**0.66**	**0.57**	**0.66**	**0.66**

P_1=75227, P_2=971, P_3=74110, P_4=8136, P_5=79233, P_6=Arbagugu, P_7= 974 and P_8=370

Table5. Estimate of heterosis percentage for incubation period.

Hybrids	Incubation period			
	BPH	MPH	SPH	SCH
P_1 x P_2	-16.34**	-5.56	8.40	16.47*
P_1 x P_3	7.27	7.47	7.67	15.69*
P_1 x P_4	-10.53	-3.18	5.48	13.33
P_1 x P_5	-23.31**	-9.06	11.68	20.00**
P_1 x P_6	11.31	12.34*	13.38*	19.61**
P1 x P_7	-6.36	4.52	18.25**	27.06**
P_1 x P_8	-21.05**	-8.26	9.49	17.65*
P_2 x P_3	-0.84	11.75*	28.00**	38.04**
P_2 x P_4	16.34**	21.83**	27.86**	61.96**
P_2 x P_5	-9.77*	-4.51	1.41	41.18**
P_2 x P_6	-0.28	13.46**	31.60**	38.82**
P_2 x P_7	3.95	5.28	6.65	44.71**
P_2 x P_8	-2.89	0.41	3.94	44.71**
P_3 x P_4	-5.57	2.01	10.91	19.61**
P_3 x P_5	-20.55**	-5.93	15.27*	24.31**
P_3 x P_6	10.91	12.13*	13.38*	19.61**
P_3 x P_7	-10.40*	-0.16	12.72	21.57**
P_3 x P_8	-23.42**	-11.15*	5.81	14.12*
P_4 x P_5	-12.78**	-3.60	7.74	36.47**
P_4 x P_6	-6.19	2.36	12.64	18.82**
P_4 x P_7	14.74**	18.68**	22.91**	55.69**
P_4 x P_8	7.89	16.64**	26.93**	60.78**
P_5 x P_6	-18.80**	-2.99	20.45**	27.06**
P_5 x P_7	-7.52	-0.94	6.65	44.71**
P_5 x P_8	-4.76	-2.44	0.00	49.02**
P_6 x P_7	-6.36	5.37	20.44**	27.06**
P_6 x P_8	-15.26**	-0.77	19.70**	26.27**
P_7 x P_8	12.89**	18.18**	23.99**	68.24**
mean	**-4.92**	**3.35**	**14.05**	**32.59**
SE(±)	**5.93**	**5.13**	**5.93**	**5.93**

P_1=75227, P_2=971, P_3=74110, P_4=8136, P_5=79233, P_6=Arbagugu, P_7= 974 and P_8=370

Combining ability analysis

Mean squares of general combining ability (GCA) and specific combining ability (SCA) for all traits are presented in Table 6. The mean squares of GCA and SCA were significant at p<0.01and/or P<0.05 for percentage of wilted seedlings and incubation period. The result indicated that both GCA and SCA variance were significantly important or the involvement of both additive and non-additive gene effects has paramount importance in the inheritance of both traits.The GCA to SCA variance ratio of percent wilted seedlings and incubation period was greater than one, indicating that the higher contribution of additive over non-additive gene effects for the traits. As a result, both selection and hybridization could be effective breeding methods to improve resistance. Mainly, CWD resistance could be incorporated from resistant sources by utilizing pure line selection, or pedigree selection (to obtain resistant segregate generation); both of which take advantage of additive gene actions (Poehlman and Sleper, 2006).

Table 6. Analysis of variance for 8 x 8 parents' half diallel mating design using Griffing's (1956) approach

Traits	Mean Squares, P' value and GCA to SCA variance component ratio						
	Block (df=2)	Genotypes (df=35)	Error (df=70)	GCA (df = 7)	SCA (df =28)	Error (df= 70)	δ^2_{GCA}/ δ^2_{SCA} ratio
Wilted coffee seedlings (%)	1065.2** (801.1)	1743.2** (823.3**)	204.5 (118.0)	2259.3** (1106.6**)	161.5** (66.4*)	68.2 (39.3)	2.4
Percentage Incubation period	397.3**	610.8**	52.7	718.2**	74.9**	17.57	1.2
Number of defoliated leaves	8.22	1.7**	0.66	2.3**	0.14^{ns}	0.22	_
Number of yellow leaves	1.58	0.14^{ns}	0.10	_	_	_	_

*=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; Data in brackets is arcsine transformed value of wilted seedlings percentage

General combining ability effects

Estimate of general combining ability (gca) effects for eight parental lines for percent wilted seedlings, incubation period and number of defoliated leaves are given in Table 7. All parents, except P_4, showed either significantly positive or negative (P<0.01) effects for percentage of wilted seedlings. Parent P_2 had the highest negative and significant gca effect followed by P_7 and P_8. Therefore, parents P_2, P_7 and P_8 were found to be good combiners for developing resistant single hybrids. Moreover, low mean percent wilted seedlings with more negative gca effect indicates greater CWD resistance, whereas susceptible lines P_3, P_1 and P_6 had significantly positive effects. All parents exhibited significant gca effects for incubation period. Based on the result, it could be concluded that P_8 had the highest positive effect followed by P_2. This indicated that good general combiner resistant parents had extended incubation period as compared to the susceptible parents.

For number of defoliated leaves, estimation of gca effects showed significant differences between parents; where P_7, followed by P_8, showed the highest negative value. Genotypes that showed low or minimum mean number of defoliated leaves are considered as desirable for resistance. Similarly, parents with high negative gca effects were good combiners and had important contribution to CWD resistance. On the other hand, parents P_1, P_3 and P_6 were poor general combiners for all traits.

Specific combining ability effects

Estimates of specific combining ability (sca) effects for 28 F_1 hybrids for percent wilted seedlings and incubation period are shown in Table 7. Fifteen hybrids showed negative sca effects for percent wilted seedlings and, thus, were in desirable direction. The single hybrids P_4 x P_8 (-24.88) and P_7 x P_8 (-18.01) showed significant (P<0.01) and negative sca effects, and good specific combinations with low percentage of wilted seedlings or CWD resistance.The result indicated that the resistant P_7 and moderately resistant P_4 could produce better resistant single hybrids in combination with moderately resistant lines. Eight hybrids, out of 11 hybrids that showed desirable incubation periods showed significant sca effects. Furthermore, most hybrids resulting from resistant lines P_7 and P_2, and moderately resistant lines P_8 and P_4 were produced better resistant hybrids. While, P_1 x P_8 (susceptible x moderately resistant), P_1 x P_2 (susceptible x resistant), P_3 x P_7 (susceptible x resistant) and P_3 x P_8 (susceptible x moderately resistant) were the most undesirable hybrids with poor specific combination for both percent wilted seedlings and incubation period. Generally, hybrids P_7 x P_8 and P_4 x P_8 exhibited significantly favorable sca effects for both traits; associated with low mean percentage of wilted seedling, extended mean incubation period and negative heterosis.

In Ethiopia, this is the first study that estimates combining ability, heterosis, heritability and genetic gain for wilt disease resistance in Arabica coffee. However, Musoli *et al.* (2013) have studied the inheritance of CWD resistance in Robusta coffee and found that estimates of GCA variance component for resistance were significant. Contrary to the current findings, their result was non-significant for sca effects; which may be due to differences in host species, pathogenic population and inoculation methods. Moreover, they reported that additive and dominance variances were low compared to the environmental variance. Similar to the present result, Epinat and Pitrat (1994) on muskmelon downy mildew resistance, Patel and Pathak (2011) on castor fussarium wilt resistance, and Changaya *et al.* (2012) on pigeon pea fussarium wilt resistance have reported the importance of both additive and non-additive genetic effects. Van der Vossen and Walyaro (1980) and Bayetta (2001) have also reported similar estimates of combining ability to CBD resistance. Contrary to this, the findings of Mert *et al.* (2005) and Lüders *et al.* (2008) on cotton verticillium wilt, Vander Vossen and Walyaro (2009) on coffee berry disease and Manu *et al.* (2014) on chilli fussarium wilt have indicated that a single dominant gene controls the inheritance of resistance.

Estimates of gca and sca effects showed significant differences between parents and hybrids, respectively. In general, low mean percentage of wilted seedlings, longer incubation period and minimum number of defoliated leaves parents were directly related to desirable gca effects. Hence, it is important to include those desirable parents and hybrids in hybridization or resistance breeding program for simultaneous improvement of CWD traits. A parent exhibiting significantly positive and negative gca effects for a particular trait is assumed to have high degree of favorable and unfavorable alleles, respectively. Furthermore, significantly positive or negative sca effects show that the two lines that produce hybrids have divergent or similar genetic background, respectively (Stangland *et al*., 1983).

Table 7. Estimates of gca and sca effects for CWD traits in artificial inoculation test.

gca effects				sca effects					
Parents	WS	IP	NDL	Hybrids	WS (%)	IP	Hybrids	WS (%)	IP
P_1	17.6**	-11.9**	0.35*	P_1 x P_2	17.0*	-7.7*	P_3 x P_7	17.8*	-5.1
P_2	-17.1**	6.7**	-0.36*	P_1 x P_3	-10.9	8.2*	P_3 x P_8	14.2	-12.99**
				P_1 x P_4	-6.22	-6.6	P_4 x P_5	15.8*	-4.9
P_3	18.8**	-9.9**	0.54**	P_1 x P_5	14.9	-3.99	P_4 x P_6	-4.1	-5.7
P_4	-0.56 ns	2.97*	0.045ns	P_1 x P_6	-5.8	9.9*	P_4 x P_7	-8.7	11.4**
				P_1 x P_7	-3.7	1.6	P_4 x P_8	-24.9**	13.8**
P_5	-8.3**	6.0**	-0.38**	P_1 x P_8	19.5*	-7.96*	P_5 x P_6	7.1	-1.7
				P_2 x P_3	3.9	8.6*	P_5 x P7	2.2	-1.33
P_6	15.0**	-8.2**	0.72**	P_2 x P_4	10.8	16.0**	P_5 x P_8	-8.3	0.7
				P_2 x P_5	-8.6	-4.7	P_6 x P_7	13.1	-2.1
P_7	-15.1**	6.40**	-0.51**	P_2 x P_6	-0.5	7.6	P_6 x P_8	14.3	-4.3
				P_2 x P_7	-5.5	-2.1	P_7 x P_8	-18.0*	16.7**
P_8	-10.3**	7.97**	-0.40**	P_2 x P_8	-8.0	-3.6	SE ± s_{ij}	7.5	3.8
				P_3 x P_4	-1.1	-3.3	S_{ii}- S_{jj}	9.0	4.6
SE(g_i)	2.4	1.2	0.14	P_3 x P_5	7.0	-2.4	S_{ij}-S_{ik}	11.1	5.6
SE(g_i-g_j)	3.7	1.9	0.21	P_3 x P_6	-11.0	7.9*	S_{ij}-S_{kl}	10.4	5.3

P_1=75227, P_2=971, P_3=74110, P_4=8136, P_5=79233, P_6=Arbagugu, P_7= 974 and P_8=370
Note: Values without asterisk (*) are non-significant; *=Significant at 5% level of significance, **= significant at 1% level of significance, ns =non-significant, SE= standard error of parents, SE (sij), SE (sii) = standard error of the hybrid i and j parents and the same parents, respectively. IP = incubation period, WS % = percent of wilted seedling, NDL = number of defoliated leaves

Estimation of variance components, heritability and genetic advance

Estimated broad and narrow sense heritability for four CWD traits are presented in Table 8. Low percent wilted seedlings or CWD resistance ($h^2{}_b$=88.27%, $h^2{}_n$=75.41%), prolonged incubation period ($h^2{}_b$= 91.37%, $h^2{}_n$= 68.83%) and few number of defoliated leaves ($h^2{}_b$= 62.06%, $h^2{}_n$= 72.39%) showed high heritability and transmission of genetic information from parents to offspring's. Results of the present study on heritability of CWD resistance in Arabica coffee contradict with the findings of Musoli *et al.* (2013) who reported low to medium heritability on Robusta coffee.

Estimates of genetic advance as percent of mean (GAM) that could be expected from the top 5% desired trees of the genotype for all CWD traits are given in Table 8. There was high GAM for percent wilted seedlings or seedling survival rate (68.61%), and incubation period (24.00) and minimum value for number of defoliated leaves (52.30%). Such a high GAM coupled with high heritability indicates that the traits could be improved through simple selection. According to Panwar *et al.* (2015), selection could be much easier for high heritable trait; but it will be difficult for a trait with low heritability. They have further indicated that heritability estimates along with expected genetic advance are usually more helpful than heritability value alone.

Table 8. Estimation of variance components, heritability and genetic gain of CWD traits

Traits	δ^2_p	δ^2_g	δ^2_{GCA}	δ^2_{SCA}	h^2_B (%)	h^2_n (%)	GA	GAM (%)
Low wilted coffee seedlings Percentage	581.1	512.9	219.1	93.370	88.3	75.4	43.8	68.61
Incubation period	203.6	186.0	70.1	57.375	91.4	68.8	26.9	24.0
Minimum number of defoliated leaves per seedling	0.58	0.4	0.21	_	62.1	72.4	0.97	52.3
Minimum number of yellow leaves per seedling	0.05	0.01	_	_	28.3		0.13	13.4

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

Conclusion

In this study, CWD resistance was examined in terms of percent wilted seedlings, incubation period, and number of defoliated and yellow leaves per seedling using eight parents and their 8 x 8 half diallel crossesin artificial seedling inoculation test. It was observed that heterosis was lacking for CWD resistance. Moreover, results showed the predominance of additive over non-additive gene effects, and high heritability estimates coupled with GAM for resistance and incubation period, which could be easily improved through selection. Due to their respective gca and sca effects, parents P_2, P_7, P_8 and P_5, and hybrids P_7x P_8 and P_4 x P_8 were found to be the best combiners and combinations for CWD resistance.

Acknowledgments

The Ethiopian Institute of Agricultural Research (EIAR), Jimma Agricultural Research Center and Jimma University (College of Agriculture and Veterinary Medicine) supported the necessary facilities and has financed the study.

References

Allard RW. 1999. Principles of plant breeding. John Wiley and Sons, New York

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

Anthony F, Combes MC, Astorga C, Bertrand B, Graziosi G and P Lashermes.2002. The origin of cultivated *Coffeaarabica* 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*olletotrichumkahawae*) and coffee wilt disease (*Gibberellaxylarioids*). MSC thesis presented to the school of graduate studies of Addis Ababa University, Ethiopia

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

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

Carvalho A. 1952.*Coffeaarabica* taxonomy VI: morphological traits of haploid. *Bragantia*, 12 (4-6): 201-212

Changaya AG, Melis R, Derera J, Laing M, VW Saka. 2012. Inheritance of resistance to Fusariumwilt and yield characters in pigeon pea. *Euphytica*, 186:883–896

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

Demelash T. 2013.Evaluation of Arabica coffee (*coffeaarabica* L.) germplasm for major coffee disease with special emphasis to coffee wilt disease (*Gibberellaxylarioids*) at Jimma, Ethiopia. MSC thesis presented to the school of graduate studies of Jimma University, Ethiopia

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

Epinat C and MPitrat. 1994. Inheritance of resistance to downy mildew (*Pseudoperonosporacubensis*) ´in muskmelon (*Cucumismelo*). 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, Teame G and A Girma2000. Significance of minor diseases of *Coffeaarabica* L. in Ethiopia: review. Proceedings of the work shop on control of coffee berry disease (CBD) in Ethiopia. Addis Ababa, Ethiopia, pp58–65

Falconer DS and T FC Mackay.1996. Introduction to quantitative genetics. Longman and Scientific and technical, London

Geiser DM, Ivey MLL, Hakiza G, Juba JH and SA Miller. 2005.*Gibberellaxylarioides* (anamorph: *Fusariumxylarioides*), a causative agent of coffee wilt disease in Africa, a previously unrecognized member of the *G. fujikuroi* species complex. *Mycologia*, 97: 191-201

Girma A. 1997.Characterization of *Gibberellaxylarioides* Heim and Saccas (Fusarium wilt) of coffee (*Coffee arabica* L.). MSc thesis presented to the school of graduate studies of Alemaya University, Ethiopia

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

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

Girma A. 2004.Diversity in pathogenicity and genetics of *Gibberellaxyilarioides*(*Fusariumxylarioides*) population and resistance of coffee spp. in Ethiopia. Doctoral Dissertation presented to University of Bonn, Germany

Girma A, Hindorf H, Steiner U, Nirenberg HI, Dehne HW and KSchellander. 2005. Genetic diversity in the coffee wilt pathogen (*Gibberellaxyilarioides*) 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 in Ethiopia. InSebil vol. 12 Proceedings of the 12th conference of the crop science society of Ethiopia (CSSE). Addis Ababa, 22-24 may 2006, Addis Ababa, Ethiopia, pp92–103

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

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

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

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

Heim R and ASaccas. 1950.Tracheomycosis of *Coffeaexcelsa* and *robusta* in the plantations of Oubangui-Chari. *CompteRendu de l'Academie des Sciences*, 231(11):536-538

Jefuka C., Adugna G., Teferi D., Zeru A., Bogale S. and AAdem. 2012. Development and release of coffee berry disease resistant varieties to specialty coffee producing regions in Ethiopia. Proceedings of 24th international scientific colloquium on coffee (ASIC). Costa Rica, 12-16 November 2012, pp636-644

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

Kranz J and M Mogk1973.*Gibberellaxylarioides* Heim and Saccas on Arabica coffee in Ethiopia. *Phytopath*,78: 365-366.

Krug CA and A Carvalho1951.The genetics of Coffea. *Advances in genetics*, 4:127-158

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

Lüders RR, GalbieriR, Fuzatto1 MG, and E Cia. 2008.Inheritance of resistance to Verticilliumwilt in cotton. *Crop Breeding and Applied Biotechnology*, 8: 265-270

Manu DG, Tembhurne BV, Kisan B, Aswathnarayana DS and JR Diwan. 2014. Inheritance of fusarium wilt and Qualitative and Quantitative traits in Chilli (*Capsicum Annuum* L). Journal *of Agriculture and Environmental Sciences*, 3(2):433-444

Mathur PN and JR 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. Proceedings of the first regional workshop on coffee berry disease. Addis Ababa, 19-23 July 1982, Ethiopia, pp203-211

Merdassa E. 1986.A review of coffee diseases and their control in Ethiopia. In: Tsedeke A (eds) Proceedings of the first Ethiopian crop protection symposium. Addis Ababa, 4–7 February 1986, Institute of Agricultural Research, Ethiopia, pp187–195

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

Mesfin A. 1982.Heterosis in hybrids 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

Meyer FG. 1965. Notes on wild *Coffeaarabica* from Southwestern Ethiopia, with some historical considerations, *Economic Botany*, 19:136–151

Musoli PC, Girma A, Hakiza GJ, Kangire A, Pinard F, Agwanda C and D Bieysse. 2009. Breeding for resistance against coffee wilt disease. In: Flood J, (eds) Coffee Wilt Disease. Typeset by MTC, Manila, Philippines, Printed and bound in the UK, pp180-197

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

Panwar IS, Arya RK, Phougat D and SK Pahuja. 2015. Use of combining ability, heritability and genetic advance in breeding programmes. Forage Res. 41 (3):164-169

Patel PB and HC Pathak. 2011. Genetics of resistance to wilt in castor caused by *Fusariumoxysporum f. sp*. ricini Nanda and Prasad. *Agricultural Science Digest*, 31(1):30-34

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

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

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

Poehlman, JM and DA Sleper. 2006. Breeding field crops. Iowa state university press, Ames, Iowa50014, India

Rutherford MA. 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

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

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

Singh RK and BD Chaudhary. 1985. Biometrical methods in quantitative genetic analysis. Kalyani publishers, New Delhi

Sprague GF and LA Tatum. 1942. General versus specific combining ability in single hybrids of corn. *J. Am. Soc. Agron.*, 34: 923-932

Stangland GR, Russel WA and OA Smith. 1983. Evaluation of the performance and selected lines derived from improved maize population. *Crop sci.*, 18:224-226

Sylvian PG. 1955.Some observation on *Coffeaarabica* L. in Ethiopian.*Turrialba*, 5: 37-53

Van der GraaffNA and R Pieters. 1978. Resistance levels in *Coffeaarabica* to *Gibberellaxylarioides*and distribution pattern of the disease. *Neth. J.Pl. Pathol.*, 84: 117-120

Van der Vossen HAM and DJ Walyaro. 2009. Additional evidence for oligogenic inheritance of durable host resistance to coffee berry disease (*Colletotrichumkahawae*) in Arabica coffee (*Coffeaarabica* L.). *Euphytica*, 165(1):105-111

Van der Vossen HAM and DJA Walyaro. 1980. Breeding for resistance to coffee berry disease in *Coffeaarabica*. II. Inheritance of the resistance. *Euphytica*, 29:777-791

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