Eur J Plant Pathol (2008) 122:451–460
DOI 10.1007/s10658-008-9310-5

Spatial and temporal analysis of coffee wilt disease caused by *Fusarium xylarioides* in *Coffea canephora*

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Received: 8 January 2007 / Accepted: 20 March 2008 / Published online: 13 May 2008

Abstract Coffee wilt disease (CWD) caused by *Fusarium xylarioides*, considered to be a soil-inhabiting fungus, is endemic in several African countries, affecting commercially important coffee species and causing serious economic losses. Coffee wilt disease development in naturally infected *Coffea canephora* fields at the Coffee Research Institute in Uganda was assessed from April 2001 to March 2006 to generate information about temporal and spatial spread of the disease. Maps of diseased trees were also generated from the data. Semi-variance analysis was performed on the data to show the spatio-temporal structure of disease. Host influence on the spatio-temporal structure was deduced from the distribution pattern of diseased and healthy trees and analysis of variance. Results show that the temporal disease epidemic progress was slow. The disease was found to spread from initial infections to healthy neighbouring trees, resulting in an aggregated pattern. An infected tree could infect up to three healthy trees away, in any direction. Disease foci formed and expanded with time, coalescing but punctuated in spots planted with resistant hosts. There were varying levels of susceptibility among host genotypes, affecting the rates and levels of epidemic development. The implications of the findings to the control of CWD are discussed.

Keywords *Robusta coffee* · Epidemiology · Geostatistical analysis · *Gibberella xylarioides* · Variograms

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Introduction

Annual green-bean coffee production worldwide is about 7 million tons (FAO 2006). After oil it is the second biggest commodity on the international markets, earning more than 9,000 million dollars per year. More than 60 million people in 51 countries depend on production and export of coffee for their income (Rutherford 2006). Arabica coffee (*Coffea arabica*) and Robusta coffee (*Coffea canephora*), are the main cultivated species. Robusta coffee constitutes 35% of global coffee production (ICO 2006).

Coffee wilt disease (CWD), also called tracheomycosis or carbuncullariosis, is a vascular disease caused by *Fusarium xylarioides*, the conidial stage of *Gibberella xylarioides*. Fraselle (1950) and Saccas (1951) described the disease symptoms on coffee trees in the field. Although Saccas (1956) reported that *F. xylarioides* attacked coffee bushes through wounds, direct infection through the root system without wounding is also possible (G Hakiza, CORI, Uganda, personal communication). Infection is possible from the cotyledon stage to mature plants. The length of the incubation period in the host is not known but it is suspected to range from days in young plants to about 6 months in established plants, before invasion of vascular tissues occurs and causes wilting (Saccas 1956; Muller 1997). Diseased trees die within one to 15 months but often within as short as six months (P. Musoli, personal observations).

Coffee wilt disease was first reported in 1927 on *C. liberica* var. *excelsa* in the Central African Republic (CAR; Figueres 1940). The disease subsequently destroyed the same crop during the 1930s–1950s in the Cameroon (Guillemat 1946; Fraselle 1950; Saccas 1951; Muller 1997). During the same period, it destroyed *C. canephora* in the Ivory Coast (Delassus 1954). Fraselle (1950) reported it on *C. canephora* at Yangambi in the Democratic Republic of Congo (DRC, formerly Zaire) in 1948 and subsequently the disease became a serious problem in many parts of the country. Lejeune (1958) reported similar disease symptoms on *C. arabica* in Ethiopia and later Kranz and Mogk (1973) confirmed that the disease on Arabica coffee was also caused by *F. xylarioides.* Pieters and Van der Graaff (1980) reported that the disease was endemic in all coffee growing parts of Ethiopia.

This first CWD epidemic in central and west African countries was controlled by combined use of resistant varieties and cultural practices (Muller 1997). Although this resulted in CWD being considered economically as a minor disease on *C. canephora*, *C. excelsa* never recovered as a commercial crop (Rutherford 2006). However, in the 1970s, new outbreaks were reported in the DRC (Flood and Brayford 1997; Rutherford 2006) on *C. canephora,*. By 1992, the disease was causing widespread destruction of *C. canephora* in DRC and in 1993 was first reported in neighbouring Uganda. Simultaneously, CWD in Ethiopia became endemic in all *C. arabica* producing areas (Rutherford 2006). Recent surveys have now confirmed the presence of CWD on *C. canephora* in DRC, Uganda and Tanzania; currently, CWD provides a major threat to the coffee industry in Africa and these countries in particular.

In Uganda, coffee is the most important cash crop, both in terms of employment and value of production, and is a major source of foreign currency. Uganda is the second African Robusta producer after the Ivory Coast (ICO 2006). CWD has rapidly devastated the Robusta crop, disrupting the national economy and decreasing the incomes of coffee producers (Lukwago and Birikunzira 1997). Surveys carried out in 2002 found the disease in all *C. canephora* growing areas and on over 90% of the farms in Uganda. The disease has already destroyed over 44% of the crop nationwide (Oduor et al. 2005).

Information on the epidemiology of CWD is scanty, yet essential for the design and effective implementation of control strategies. The scarcity of epidemiological information may partly account for the failure to control CWD in Uganda using phytosanitory measures (Wetala et al. 2000). Roguing and burning of affected trees is of some benefit but their adoption is constrained by on-farm resources. Future prospects for the effective management of CWD are thought to depend largely on host resistance and most research and development efforts are focused in this direction (Rutherford 2006).

Spatial and temporal analyses have been applied in the field of plant pathology at a variety of scales, from single plots to agricultural regions, to analyze the interactions between pathogens, hosts and the environment in relation to plant disease epidemics (Chellemi et al. 1988). The study of spatial and temporal patterns can provide quantitative information on population dynamics, aid in the design of epidemiological studies, sampling programmes for disease or pathogen monitoring, and be used to generate hypotheses about underlying ecological processes (Ristaino and Gumpertz 2000). Among others, semi-variance (variogram) analyses are used in the study of spatial–temporal dynamics of different plant diseases (Rekah et al. 1999; Van de Lande and Zadoks 1999; Jaime-Garcia et al. 2001).

In this study, the spatial–temporal pattern of CWD in a naturally infected field of mixed Robusta coffee genotypes was analyzed using a combination of classical and geo-statistical analyses. The study used

semi-variance (variograms) analysis to relate the spatial and temporal spread of the CWD among the coffee trees. The disease probability maps derived by kriging were related to the spatial and temporal pattern of diseased trees in the field. The study addressed three specific questions: (1) how does the CWD epidemic develop in time? (2) How does the CWD pattern change in space and time?, and (3) how does host heterogeneity affect the disease epidemic and the spatial pattern?

Materials and methods

Studies were carried out in Uganda on a Robusta coffee field experiment at the Coffee Research Institute (CORI) in Kituza. The experimental field was planted in October 1997. The trial was initially designed to evaluate varieties for yield, growth habit, quality and resistance to coffee leaf rust (*Hemileia vastatrix*) and red blister disease (*Cercospora coffeicola*), since at the time of planting CWD was not a serious problem in the country and had not yet reached Kituza.

The experimental field occupied 0.50 ha planted with twenty *C. canephora* clones. A total of 16 out of 20 clones were single-tree selections among progenies of specific crosses and the remaining four were commercial clones; 1s/2, 1s/3, 223/32 and 257/53. All the clones were selected for agronomic traits (yield and cup quality) other than resistance to CWD. The experimental plot was weeded, trained, pruned and supplied with organic and inorganic fertilizers following routine procedures for maintaining *C. canephora* gardens (MAAIF 1995).

The experiment was laid out in a randomised complete block design with four replicates running along contours. Each replicate was sub-divided into 20 plots of six trees arranged in straight line patterns of three rows×two columns at 3×3 m spacing. Each of the clones was randomly allocated and planted in a plot in each of the replicates. Clones Q/6/1 and Q/1/1 were only planted in replicates 1 and 2 due to insufficient planting materials. Clones H/4/1 and R/14 were not planted in replicate 4 for the same reason. The experimental plot was surrounded by two guard lines of non-experimental coffee materials and a guard line in between replicates.

Data collection and analysis

CWD was first observed in the experimental field in 1999 but systematic assessment did not start until April 2001 and lasted until March 2006. From March 1999 to March 2001, all affected trees were uprooted to minimise and, if possible, eradicate the disease. Eventually assessment of CWD took precedent to other traits, because of its importance. Non-experimental coffee trees in rows and columns surrounding experimental gardens and separating replicates were not assessed.

All trees in experimental plots were assessed every 2 weeks on a disease severity scale of 1 to 5 where 1 = no disease, 2 = 1–25% defoliation, 3 = 26–50% defoliation, 4 = 51–75% defoliation, 5=76–100% defoliation. All plants in level 5 were considered dead. Percentage tree mortality was also estimated from these observations and plotted over time to give the disease progress curve (Fig. 2a). The percentage new tree mortality per 6-month time period was plotted to give the disease epidemic curves (Fig. 2b). This measure is more accurate than the percentage of first symptoms, which is often difficult to detect.

Maps for spatial distribution of diseased (sick and/or dead) coffee trees at annual intervals were generated from the disease severity data using SAS (SAS Institute Inc., Cary, NC 1989). The maps were used to visualize the spread of the disease over time.

Isotopic semi-variance analysis was performed on disease severity marks to determine the spread of CWD over time measured with and without host effects. An ANOVA with clones as a studied factor is used to remove host effects: semi-variance analysis was performed on residuals from ANOVA. For the analysis without host effects, residuals were calculated for all individual tree scores by subtracting corresponding clone means. The residual data were then subjected to semi-variance auto-correlation statistical analysis, and semi-variograms were derived to show the spatial spread of the disease measured without host effects. Disease severity data sets from the start of the experiment until the end were analyzed using semi-variance analysis performed with GS^+ (Gamma Design Software 2004). In this analysis, semi-variances on the data for all possible pairs of trees were determined as:

$$Y(h) = \frac{1}{2N(h)} \sum_{N(h)} [z(s_i) - z(s_i + h)]^2 \quad (1)$$

where $Y(h)$ is the semi-variance for interval distance class h, $N(h)$ is the total number of pairs of trees for the lag interval h (lag distance), $z(s_i)$ is the disease severity value of a coffee tree located at point s_i, and $z(s_i+h)$ is the disease severity value of a coffee tree at distance h from s_i. Semi-variances for each interval class were plotted against corresponding lag distances to constitute a semi-variogram. The GS$^+$ programme provides five types of isotropic models, each of which can be described by three parameters (Fig. 1) that describe spatial structure of the disease.

The three parameters are: (1) nugget variance or C_0: this is the y-intercept of the model. It represents variance due to error, (2) sill or C_0+C: the model asymptote which provides an estimate of total sample (population) variance where C is the variance due to spatial structure and C_0 the nugget variance, and (3) range or A: the separation distance over which spatial dependence is apparent, sometimes called the effective range in order to distinguish range (A) from the model's range parameter (A_0). The software also provides three statistics to aid the interpretation of the model output. Firstly, the residual sum of squares (rss), which provides an exact measure of how well the model fits the variogram data; the lower the residual sums of squares, the better the model fits. When GS$_+$ autofits the model, it uses rss to choose parameters for each of the variogram models by determining the combination of parameter values that minimizes rss for any given model. Secondly r^2, which provides an indication of how well the model fits the variogram data; this value is not as sensitive or robust as the rss value for best-fit calculations. Thirdly, the proportion $C/(C_0+C)$ provides a measure of the proportion of sample variance (C_0+C) explained by spatially structured variance C. This value will be 1.0 for a variogram with no nugget variance (where the curve passes through the origin); conversely, it will be 0 where there is no spatially dependent variation at the range specified. Thus, a high ratio of $C/(C_0+C)$ implies there is a large spatial dependence. Since the analysis is performed on repeated measurements, it allows analysis of changes in the spatial dependence from the variogram parameters for the different data sets.

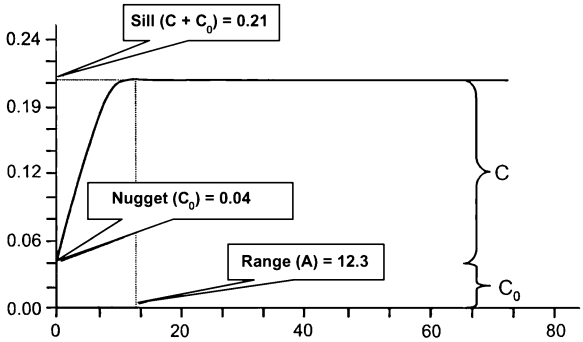

Fig. 1 Generalized variogram model. (Adopted from GS$^+$ User's Guide Version 7, Gamma Design Software 2004)

Results

Temporal pattern

The level of CWD, as indicated by percentage mortality, was relatively high at the beginning of the assessment in April 2001 (25.2%, Fig. 2a) and increased from 25.2% to 64.5% in March 2006. The disease epidemic, as indicated by new mortalities (Fig. 2b), was highest between April 2001 and June 2002 but subsequently decreased and levelled off over time before finally reaching 0.4%.

Analysis of host influence

Percentages of dead trees indicated that most hosts (clones) were affected to some degree by CWD in April 2001. The hosts showed tree mortalities ranged from 0% for J/1/1 and Q/3/4, to 54% for P/3/6 (Table 1). By March 2006, the percentage of tree mortality varied from 0% for J/1/1 to 96% for clone C/1/7 (Table 1). The comparisons of clones on disease severity score gave approximately the same rankings as the comparisons on the percentage of tree mortality (Table 1).

Spatial pattern

In Fig. 3 maps with individual trees classified as either healthy, diseased or dead for the years 2001 to 2006 are shown. Trees affected by CWD aggregated into clusters of varying sizes. The number of affected trees among the aggregates increased gradually over time. Areas of the experimental field planted with clones 1s/3, Q/3/4 and R/1/4, had very few affected trees. Parts of the experiment planted with clone J/1/1 did not have any trees with wilt symptoms. Analysis

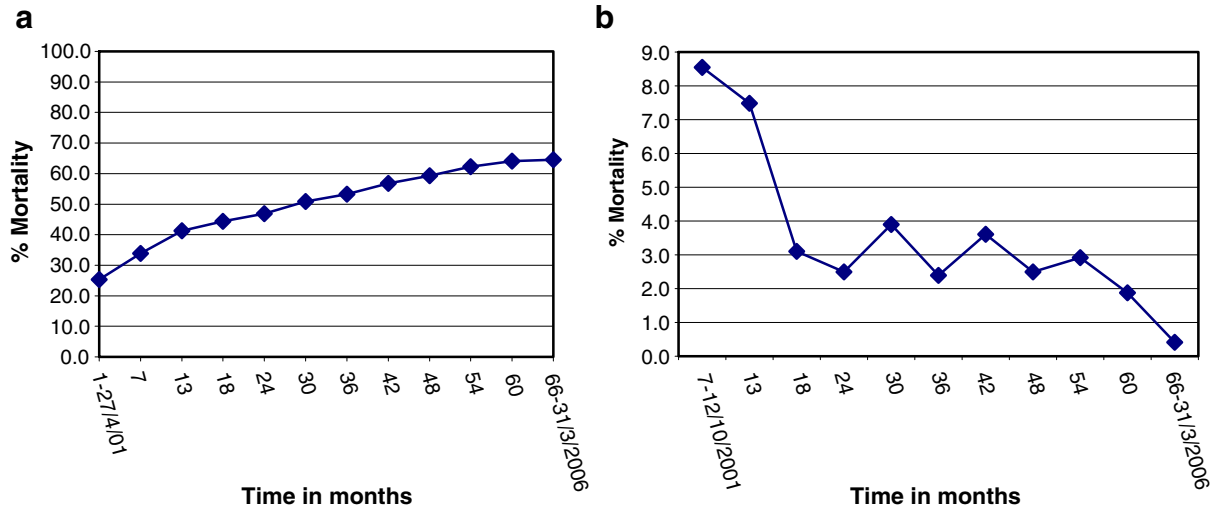

Fig. 2 Coffee wilt disease progress curves and epidemic rates. (**a**) Increase of percent mortality, (**b**) percent new mortality (per 6-month period)

of variance performed on the disease severity data from the different assessment dates shows that variation due to blocks (replicate) was not significant ($P<0.05$)

Semi-variance analysis performed on the disease severity data showed that the exponential model fitted best the data with host effects. The rss ranged from 0.058 to 0.510 for the 11 semi-variograms used to illustrate the spatial dependence of CWD (Table 2). The same model also provided the best fit, with the rss ranging from 0.032 to 0.136 for the 11 semi-variograms used to illustrate spatial dependence of

Table 1 Variogram characteristics and model parameters describing the spread of coffee wilt disease

Clone	No. of plants	First assessment: April 2001		Last assessment: March 2006	
		% Mortality	Disease severity mark	% Mortality	Disease severity mark
J/1/1	24	0a	1.00a	0.0a	1.00a
Q/3/4	24	0a	1.00a	4.2b	1.25a
R/1/4	24	11.1b	1.44abc	33.3c	2.67bc
1S/3	18	12.5b	1.50abc	33.3c	2.33b
B/2/1	24	29.2bc	2.25abcd	50.0cd	3.25bcdef
Q/6/1	12	50d	3.00cd	50.0cd	3.00bcd
C/6/1	24	12.5b	1.50abc	54.2cd	3.13bcde
223/32	24	12.5b	1.63abc	58.3cde	3.37bcdefg
L/2/7	24	12.5b	1.71abc	62.5def	3.50bcdefg
Q/1/1	12	41.7cd	2.67abcd	66.7defg	3.67bcdefg
B/1/1	24	29.2bc	2.17abcd	75.0defgh	4.00cdefg
257/53	24	29.2bc	2.42abcd	83.3efgh	4.33defg
G/3/7	24	25bc	2.13abcd	83.3efgh	4.42defg
1S/2	24	4.2ab	1.17ab	87.5fgh	4.54efg
E/3/2	24	20.8bc	2.08abcd	87.5fgh	4.50defg
P/5/1	24	54.2d	3.33d	87.5fgh	4.50defg
B/6/2	24	37.5cd	2.75bcd	91.7gh	4.79fg
P/3/6	24	54.2d	3.46d	91.7gh	4.67fg
H/4/1	18	27.8bc	2.61abcd	94.4gh	4.78g
C/1/7	24	41.7cd	2.92cd	95.8h	4.83g

Values with the same letter in a column are not significantly different according to Newman Keuls multiple range test ($P=0.05$)

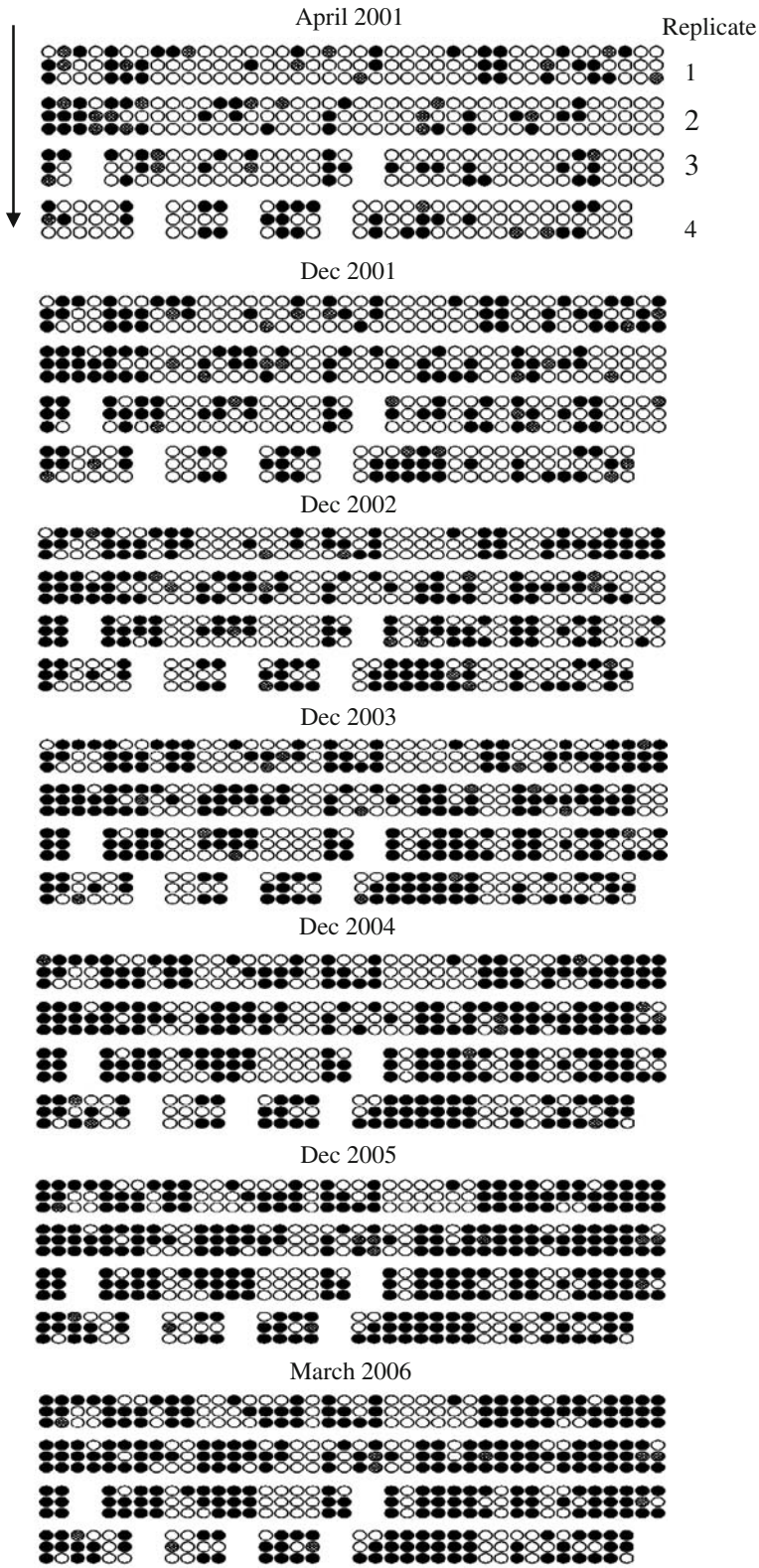

Fig. 3 Pattern of healthy, diseased and killed trees at plot level. Arrow is pointing down the slope; each tree is represented with a *circle*; *black circles* are trees killed by CWD; *circles with cross squares* show sick tree and *white circles* show uninfected trees; four replicates in the field separated by *larger clear lines; two rows × three columns of circles* in each replicate represent a clone; *white gaps* within replicates represent missing data; trees uprooted prior to April 2001 are included among dead trees

CWD without host effects. Small nugget (error) effects were observed for all semi-variance analyses of disease patterns measured with and without host effects (Table 2). The proportion $C/(C_0+C)$ of structural variance (C) to total variance (C_0+C) for the disease measured with host effects ranged from 0.85, observed in June and November 2005 and March 2006, to 0.90 in April and October 2001 data delete. The proportion of spatial structural variance $C/(C_0+C)$ for disease measured without host effects ranged from 0.82 in June and December 2004 to 0.94 in December 2003, November 2005 and March 2006 (Table 2). These relatively high proportions of spatial structural variance $C/(C_0+C)$ indicate that there is a strong spatial correlation between trees with CWD.

The spatial dependence distance (the effective range A), increased from 1.9 trees in April 2001 to 3.3 trees in March 2006 (Table 2), showing that the disease spread from diseased trees to infect up to two neighbouring coffee trees away in April 2001 and up to at least three trees away by March 2006, in all directions. Spatial dependence was also observed in the analysis without host effects, *i.e.* on the residuals of ANOVA with clones as the factor (Table 2). Changes in spatial dependence distance over time for disease measured without host effects were smaller than those observed for disease measured with host effects. The effective range (A) for disease measured without host effects varied slightly between years, from 0.96 in March 2002 to 1.29 in April 2001, which indicates that in all cases, the disease spread to infect up to one coffee tree away.

Maps revealed numerous disease foci of varying sizes scattered in all parts of the experimental plot at the beginning of the assessment (Fig. 3). The disease foci expanded in all parts of the experimental plot and by March 2006, they had coalesced to form large continuous zones of high disease severity levels surrounding a few patches with lower disease severity levels or no disease. The patches of low disease severity levels or no disease corresponded to

Table 2 Comparison of clones for percentages of mortality at the first and last assessments

Date	Rss	$C/(C_o+C)$	Nugget (C_o)	Sample variance (C)	Sill (C_o+C)	Range (A_o)	Effective range (A)
Measured with host effects							
April 2001	0.10	0.90	0.29	2.61	2.90	0.62	1.86
October 2001	0.17	0.90	0.48	4.13	4.61	0.69	2.07
March 2002	0.12	0.89	0.54	4.41	4.95	0.68	2.04
November 2002	0.17	0.88	0.63	4.76	5.39	0.76	2.28
May 2003	0.14	0.88	0.73	5.41	6.136	0.83	2.49
January 2004	0.24	0.87	0.75	5.19	5.94	0.88	2.64
July 2004	0.33	0.87	0.77	5.14	5.91	0.88	2.64
December 2004	0.43	0.86	0.83	4.90	5.73	0.99	2.97
June 2005	0.49	0.85	0.83	4.83	5.66	1.02	3.06
November 2005	0.51	0.85	0.81	4.74	5.55	1.07	3.21
March 2006	0.06	0.85	0.86	4.88	5.74	1.10	3.30
Measured without host effects (on residuals of ANOVA with Clone effects)							
April 2001	0.04	0.92	0.20	2.18	2.38	0.34	1.02
October 2001	0.14	0.90	0.35	3.17	3.52	0.37	1.11
March 2002	0.08	0.89	0.39	3.17	3.56	0.26	0.78
November 2002	0.06	0.88	0.43	3.15	3.58	0.32	0.96
May 2003	0.04	0.87	0.46	3.04	3.50	0.39	1.19
January 2004	0.03	0.89	0.42	3.32	3.74	0.39	1.19
July 2004	0.04	0.89	0.42	3.30	3.72	0.34	1.02
December 2004	0.04	0.89	0.39	3.10	3.49	0.33	0.99
June 2005	0.04	0.88	0.40	3.07	3.47	0.32	0.96
November 2005	0.06	0.89	0.38	2.99	3.37	0.40	1.20
March 2006	0.06	0.88	0.41	3.10	3.51	0.43	1.29

Rss Residual sum of squares, C_0 nugget variance, C variance due to spatial structure

areas planted with clones J/1/1, Q/3/4, 1s/3 and R/14 as well as some areas with missing data.

Discussion

Geo-statistics can be used to quantify the degree and range of the spatial dependence of variables and has been used in plant pathology to quantitatively characterize changes in the spatial patterns of disease over time (Rekah et al. 1999). Several studies have used geo-statistics to study the spatial distribution and temporal development for annual disease cycles (Rekah et al. 1999; Van de Lande and Zadoks 1999; Jaime-Garcia et al. 2001). This study on CWD showed that at the early stages of the epidemic, a more or less random distribution pattern was observed. In time, clusters of diseased and dead plants were formed, expanding in all directions (Fig. 3). The high structural or sample variance revealed a high degree of spatial dependence for the disease, which implies that plant to plant infection plays a key role in CWD spread. However, the source for initial infections, which initiate an epidemic, remains a key question to be answered by more in-depth studies of the epidemiology of CWD. In the case of wilt on perennial crops due to *Fusarium oxysporum* f. sp. *elaïdis* on *Elais guineensis* and *F. oxysporum* f. sp. *albedinis* on *Phoenix dactylifera*, the disease was shown to spread by roots (de Franqueville 1995; Malençon 1949). For CWD, the transmission by roots is suspected but not demonstrated. Wounding transmission, notably by pruning, is also strongly suspected. The transmission of the disease may be mainly due to human activities, thus the management of plantations has important implications in controlling the disease.

The effective range, derived from semi-variance analysis of assessments with host effects, indicated that diseased coffee trees can infect coffee trees up to approximately 10 m away (about three contiguous trees). This distance was initially shorter, between one and two coffee trees, but increased with increasing disease incidence. Rekah et al. (1999) found that the infection range of *F. oxysporum* f. sp. *radicis-lycopersici*, the causal agent of crown and root rot of tomato, ranged between 1.1 and 4.4 m in the exponential phase of the disease. Spatial dependence of *Phytophthora capsici* reached 15 m (Larkin et al. 1995).

Information on the role of diseased trees in disease spread as well as knowledge on the effective range, is important for designing effective coffee management strategies. Roguing of infected trees at the earliest opportunity will minimize disease spread and inoculum potential, thus slowing the CWD epidemic. Present results also suggest that for roguing to be effective, coffee trees up to 10 m away from the diseased tree have to be rogued. As indicated in historical papers, rigorous systematic roguing was used to eliminate the previous CWD problem on *C. liberica* in Cameroon (Muller 1997). Roguing can be applied in those regions where the disease is appearing for the first time and when disease levels are still low, most likely <10%, or it is still localized. From a certain disease level upwards, roguing may be uneconomical since there will be many foci scattered throughout the fields, which would result in a very substantial, if not total, loss of the coffee plantation. However, at certain infection levels, current control measures will not suffice or will be too expensive to implement and hence total loss of the coffee plantation will be unavoidable. Future studies should include economic analyses to clarify these questions.

The disease progress (Fig. 2a) illustrates that once CWD has invaded a Robusta coffee field, it continues to infect susceptible coffee trees, leading to tree mortality. The epidemic rate was highest, approximately 10% per annum, when mortality was between 25–45%. The epidemic rate was reduced drastically as tree mortality exceeded 50%, because the number of susceptible trees was greatly reduced. This is typical of wilt diseases of many perennial tropical plants (Ploetz 1991, 2003, 2006). In addition, contamination of the soil with a soil-borne pathogen can preclude the planting of susceptible crop genotypes for decades. This observation stresses the need for resistant varieties for re-planting since most of the Robusta coffee growing areas in Uganda and the DRC are infected with CWD. The near 65% mortality observed in our study field, within a period of seven years, illustrates the devastating effects of the disease. This reiterates the urgent need to develop effective control strategies and confirms earlier reports that CWD is a very serious impediment to investment in coffee farming (Lukwago and Birikunzira 1997; Rutherford 2006).

The variability observed in disease progression and final disease levels on the *C. canephora* clones in this study illustrated that host genotypes influence spatial and temporal CWD development. These data show the presence of resistance among *C. canephora* genotypes within Uganda. This should be explored further to identify more resistant clones needed for re-planting country-wide. As reported by Ploetz (2006), *Fusarium* wilt diseases are managed most effectively by using host resistance. Saccas (1956) and Meiffren (1957) mentioned that resistant varieties were replanted to control the previous CWD epidemic in the DRC and Ivory Coast. This observation also indicates that the phenomenon of discrete patches of particular genotypes withstanding coffee wilt in a highly infected field can be explored for identifying resistant varieties in a farmer's fields, where the wilt has devastated most of the coffee trees. Interestingly, in Uganda, a number of susceptible but carefully selected indigenous clonal lines of *C. canephora* have been multiplied and been provided to farmers. When these are interplanted in rows, each row comprising a different clone, overall crop losses due to CWD are reduced and yields increased, possibly because the spread of the pathogen across rows is reduced (Rutherford 2006).

It should be noted here that although these studies generated valuable information for disease control, future studies must address other aspects of the disease epidemiology including pathogen transmission. This information is necessary for timing phytosanitory interventions. It is also imperative that the resistant clones identified in this study are evaluated in different agro-ecological areas to verify their resistance and determine their performance for other agronomic traits.

Acknowledgements We thank Dr. Denis Kyetere for assistance in several aspects of this work and Ms. Agnes Nabaggala for technical support with the field work. This work was supported by grants from the European Union within the framework of the EU INCO-COWIDI project 'Development of long-term strategy based on genetic resistance and agro-ecological approaches against CWD in Africa', 'EU-NARO support to coffee wilt research' projects and CIRAD.

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