

# Potential Effects, Biology and Management Options of Coffee Leaf Rust (*Hemileia Vastatrix*): A Review

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**Abstract:** Despite coffee plays significant role in social, cultural and national economy of the globe, the coffee industry is potentially at risk due to biotic and biotic stress. Among biotic stress; coffee leaf rust (Hemileia vastatrix) is one of the major disease which has been causing high crop lose world-wide. The breeding programme of coffe leaf rust research center (CIFC) was essentially based on the utilization of HDT as a resistant parent. The main hybrids produced at CIFC with HDT were: HW26 = Caturra Vermelho x HDT 832/2; H361 = Villa Sarchi x HDT 832/2; H528 = Catuaí Amarelo x HW26/13; H529 = Caturra Amarelo x H361/3. Ethiopian commercial varieties: Catimor J-19. Catimor J-21 and Gesha show partial resistance to the rust. However, in the last few years, some improved commercial varieties are gradually losing their resistance to rust due to the new increase of H. vastatrix virulence races. This shows the importance of re-evaluating existing varieties with the new races and the care needed before the release of new ones. Priority must also be given to the identification of sources of more durable resistance. Ethiopia as the source of origin perhaps for both the host and pathogen plays an important role in science either for breeders or pathologists. Hence, for further advanced investigations on the host and pathogen to improve the durability of coffee resistance to the disease the last ecosystems of rainforest/wild coffee in Ethiopia urgently need to be protected.

Keywords: Coffee, Hemileia vastatrix, biology, Dynamics

## **1. INTRODUCTION**

Coffee Arabica originated in Ethiopia and its wild variety currently only grows in Ethiopia, Uganda, and Kenya (Koebler, 2013). Its cultivated form is grown throughout Africa and South America (Koebler, 2013). The cultivated, commercial form of Arabica coffee lacks genetic diversity, which makes it especially prone to diseases (Paramaguru, 2012). The largest and most diverse populations of indigenous (wild) Arabica occur in the highlands of south-western Ethiopia (TeketayD. 1 999, Gole TW., 2003, Labouisse *et al.*,2008).

The genetic diversity of wild Arabica populations far exceeds that of cultivated varieties used in crop production and accessions held in germplasm collections (Labouisse *et al.*, 2008). The wild populations also have high functional diversity in terms of disease (Adugna G, 2005), and pest and drought tolerance (Taye K, 2006). Meseret (1996) evaluated large number of coffee accessions including coffee berry disease (CBD) resistant selections and reported existence of partial resistance to CLR. Similarly, Girma and Chala (2009) tested large number of Arabica coffee collections for resistance to CLR and reported existence of significant differences among the collections. Despite of the existences of substantial gene pool of *C. Arabica*, the country is not yet fully utilizing its coffee genetic resources as expected in terms of improving coffee productivity and the livelihood of the rural community due to biotic and abiotic constraints (Paulos and Teketay, 2000; Gole *et al.*, 2000).

Consequently the production and productivity of coffee per unit area remains very low as compared to global average. Among biotic constraints like diseases, Coffee Leaf Rust (CLR) (*Hemileia vastatrix*) is economically important one in the world (Mugiira *et al.*, 2011). Therefore, the objective of this paper is to review and to generate information from the potential effect, the biology, and dynamics, resistant mechanisms and disease management options.

#### 2. COFFEE LEAF RUST (CLR) (HEMILEIA VASTATRIX)

Coffee leaf rust (CLR) is one of the most important diseases of *C. arabica* in the world (Kushalappa and Eskes, 1989). Coffee rust has likely been around since Arabica coffee was only growing wild in Africa, but was not 'officially' detected there until the 1870's. Its first recorded impact began in the end of the 19th when a large outbreak in Ceylon (now Sri Lanka) devastated the coffee industry on that small island, ending in the crop being replaced with tea (Abbay, 1876).

By the 1960s it had spread throughout Indonesia, and in the 1970s it finally made its way to the Americas, to the Bahia State in Brazil (Correspondent B., 1970; Monaco, 1977). From Brazil, despite the best efforts to quarantine or eradicate the pathogen, it was spread around central and South America over a 15 year period. From what is now known about the viability of spores, it is thought that intercontinental dispersal was possible through wind. Now, it is always around, just in varying degrees. Despite the availability of effective fungicides and resistant varieties for the control coffee leaf rust, it may still cause losses varying between 10 and 40% in different countries (Silva *et al.*, 2006). In Brazil, losses have been estimated to be about 30% and an annual loss of about 4500 tons of coffee was estimated in Kenya in the 1960s.

The pathogen prefers a temperature range of 20–28 °C needs a leaf wetness period only during spore germination and penetrates with the germination hyphae into the stomata of the host. The fungus tolerates longer seasons without rainfall and spores are wind-borne, only attacking leaves and needs no other host for completing the life cycle. Due to the fact that coffee is a perennial host with green leaves all through the year, the pathogen produces the uredinal, telial, and basidial stages, but only the dycariotic urediospores are responsible for the disease. Coffee grown in lower altitudes is more predisposed to the disease and suffers more attacks. A heavy infestation of leaves not only reduces the assimilation area but also results in a complete defoliation diminishing the next year's crop tremendously.

CLR was first reported in Ethiopia in 1934 (Sylvain PG., 1958), but the disease had existed for a long time in other countries without causing epidemics or eradications of certain varieties of *C. arabica*. The long-term coexistence of coffee and rust coupled with the high genetic diversity of coffee populations and a high level of horizontal resistance might have kept the rust at low levels (Van der Graaff NA., 1981). Other factors such as the low average productivity associated with shade and the existence of biological agents such as the hyperparasite *Verticillium lecanii*, were also believed to play an important role in maintaining CLR at low levels.

## 2.1. Pathogen Biology

The coffee leaf Rust was first reported by an English explorer on wild *Coffea* species in the Lake Victoria region of East Africa in 1861. In 1869, the Reverend H. J. Berkeley and his assistant, Mr. Broome, reporting in the *Gardeners' Chronicle*, described the fungus they found associated with the disease on some dried coffee leaves sent from Ceylon (now Sri Lanka). They gave the name *Hemileia vastatrix* to the devastating fungus with half-smooth spores (Figure 1).

The Coffee Rust is an obligate parasitic fungus, which means it is a microorganism that must take energy and nutrients from a specific live host (coffee) and reproduces differently than either plants or animals. It belongs to the family of Pucciniaceae in the order of Uredinales of the class Basidiomycetes (Mayne WW., 1932). The genus has unknown pycnidial and aecidial stages and only the dikaryotic uredospores are responsible for the disease development (Kushalappa and Eskes, 1989). The pathogen exists in different physiologic groups.



Figure1.

*Hemileia vastatrix* exists primarily as dikaryotic (having pairs of haploid nuclei that divide in tandem), nutrient-absorbing mycelium ramifying intercellular within the leaves of its coffee host. Clusters of short pedicels bearing dikaryotic urediniospores stick out through the stomata on the undersides of the leaves (Figure 2). Occasionally under cool, dry conditions toward the end of the season, teliospores are produced among the urediniospores on older, attached leaves. Following karyogamy and meiosis, the teliospores germinate to produce basidia, each of which forms four haploid basidiospores (Figure 3).





#### Figure3.

The basidiospores will germinate in vitro, but it is not known what plant, if any, they can infect. It is clear that they do not infect coffee. It is not known whether the basidospores are functional or are simply remnants of an ancestral, long-cycled (up to five different spore stages) rust fungus. No alternate host is necessary; *H. vastatrix* can survive and reproduce quite nicely by urediniospores alone. Often a hyperparasitic fungus, *Verticillium hemileiae*, will colonize the coffee rust lesions. Hyperparasites are parasites that parasitize other parasites and are sometimes used as biological control agents. With coffee rust, this hyperparasitism reduces the viability of the urediniospores, but it has very little impact on overall rust development.

#### 2.1.1. Symptoms and Signs

Infections occur on the coffee leaves. The first observable symptoms are small, pale yellow spots on the upper surfaces of the leaves (Figure 4). As these spots gradually increase in diameter, masses of orange urediniospores (= uredospores) appear on the undersurfaces. The fungus sporulates through the stomata rather than breaking through the epidermis as most rusts do, so it does not form the pustules typical of many rusts. The powdery lesions on the undersides of the leaves can be orange-yellow to red-orange in color, and there is considerable variation from one region to another.



Figure4.

Figure5.

Figure6.

While the lesions can develop anywhere on the leaf, they tend to be concentrated around the margins, where dew and rain droplets collect (Figure 7). The centers of the spots eventually dry and turn brown, while the margins of the lesions continue to expand and produce urediniospores. Early in the season, the first lesions usually appear on the lowermost leaves, and the infection slowly progresses upward in the tree. The infected leaves drop prematurely, leaving long expanses of twigs devoid of leaves (Figure 8). When a coffee plant does not have the optimal amount of leaf area, it does not have the ability to accumulate adequate energy via photosynthesis and store up the appropriate resources for fruit

production. This is why there is generally a loss of yield even the year after rust outbreaks (Avelino and others 2004).



Figure7.



Figure8.

## 2.1.2. Disease Cycle and Epidemiology

The fungal life cycle is a complex and ingenious one, where organisms asexually produce thousands of tiny spores (reproductive bodies) that can travel in water, rain, or air and remain viable for long distances (Kushalappa and Eskes, 1989).



Figure9.

The disease cycle is a simple one. Urediniospores initiate infections that develop into lesions that produce more urediniospores.

## 2.1.3. Survival

*Hemileia vastatrix* survives primarily as mycelium in the living tissues of the host, and since infected leaves drop prematurely (figures 7 and 13); this effectively removes a huge amount of potential inoculum from the epidemic. But a few green leaves always persist through the dry season, and dry

urediniospores can survive about 6 weeks, so there is always some viable inoculum to infect the newly formed leaves at the start of the next rainy season.



Figure7.

# Figure10.

Figure5.

## 2.1.4. Spore Dispersal

The urediniospores can be dispersed by both wind and rain. By observing patterns of infection on individual leaves and among leaves within the canopy, it is clear that splashing rain is an important means of local dispersal. The patterns of infection on a regional scale, particularly in those areas where the fungus was newly introduced, have shown that the long-range dispersal is primarily by wind (King'ori and Masaba, 1994). A small, perhaps epidemiologically insignificant amount of urediniospore dispersal is by thrips, flies, wasps, and other insects. Movement across oceans, deserts, and mountain ranges has very likely been caused by human intervention.

## 2.1.5. Infection

Urediniospores germinate only in the presence of free water (rain or heavy dew) (Nutman and others 1963); high humidity alone is not enough. The whole process of infection requires about 24 to 48 hours of continuous free moisture, so while heavy dew is enough to stimulate urediniospore germination, infection usually occurs only during the rainy season. The seasonal variation in disease incidence is primarily due to variation in rainfall. Where there are two rainy seasons per year, there are two peaks in severity of coffee rust. Temperature also effects rust development. If it is too cold (below 15°C) spores will not germinate and the growth of the fungus will be slowed. Likewise, if it is too warm (more than 35°C) the fungal growth with slow. The most optimal temperature for the growth and proliferation of the rust is between 21-25°C with100percentRH (Nutman et al. 1963).

Light can also change how the fungus affects coffee plants, although there is some mixed evidence around this. Leaves under full sun (high intensity light) have been shown to be more susceptible to rust, and once infected light can alter the growth rate of the fungus. However, very high intensity light has also been shown to slow the fungus growth (Muller et al. 2009). Infection only occurs through stomata on the underside of the leaf.

## **Fungal infection process**

The following properties appear to be characteristics of these fungi:

(i) highly developed infection structures; (ii) limited secretory activity, especially of lytic enzymes; (iii) carbohydrate-rich and protein-containing interfacial layers that separate fungal and plant plasma membranes; (iv) haustoria, which are specialized hyphae for nutrient absorption and metabolism; (v) both long-term suppression of host defenses and induction of specific host genes for the establishment of biotrophy (Voegele and Mendgen, 2003). The initiation of the dycariotic phase of *H. vastatrix* on coffee leaves, as with other rust fungi (Mendgen and Voegele, 2005) involves specific events including appressorium formation over stomata and penetration by inter- and intracellular colonization (Silva et al., 1999a, 2002).

Thus, in susceptible coffee leaves, after urediospore germination and appressorium differentiation over stomata (figure 1C), the fungus penetrates (from 12h after inoculation) (figure1D) forming a penetration hypha (figures 1E) that grows into the sub-stomatal chamber. This hypha produces at the advancing tip two thick lateral branches; each hypha and its branches resemble an anchor (figure 1F). Each lateral branch of the anchor bears a hypha (haustorial mother cell, HMC), the subsidiary cells being the first invaded by haustoria (figure 1G), whose formation starts around 36h after inoculation. The fungus pursues its growth with formation of more intercellular hyphae, including HMCs, and a large number of haustoria in the cells of the spongy and the palisade parenchyma and even of the upper epidermis.



**Figures1A-G.** Coffee – H. vastatrix interaction. Figure 1A - Pustules of uredosporic sori on the lower side of the leaf, 21 days after inoculation. Figure 1B. Scanning electron micrograph. Uredosporic sori (x700). Figure 1C - Germinated urediospores (U) with germ tubes (T) and appressorium (A) on the lower surface of the leaf, 24h after inoculation (x1,000). Figure 1D - An empty appressorium (A) over leaf stomata, indicating that the fungus already penetrated (x3,300). Figure 1E - Urediospore (U), appressorium (A) over stomata and penetration hypha (arrow), 24 h after inoculation (x800). Figure 1F - Appressorium (A) over stomata and an anchor (arrow), 48 h after inoculation (x650). Figure 1G - Appressorium (A) over stomata and intercellular hypha with haustoria (arrows) in the subsidiary cells, 72h after inoculation (x650).

### 2.1.6. Sporulation

It takes 10-14 days from infection for new uredinia to develop and urediniospores to be formed. The rust lesions continue to enlarge over a period of 2 to 3 weeks. A single lesion will produce four to six crops of spores, releasing about 300,000 urediniospores over a period of 3 to 5 months. Secondary cycles of infection occur continuously during favorable weather, and the potential for explosive epidemics is enormous.

#### 2.1.7. Fungal Variability

The first race differentiation of *H. vastatrix* was carried out by Mayne (1932) in India, who differentiated the local rust samples into four physiologic races. No other studies were made on the physiological specialization of *H. vastatrix* until D'Oliveira initiated a world survey of coffee rust races in 1952 in Portugal (D'Oliveira, 1965). The work carried out at the Coffee Rusts Research Center (CIFC) enabled the characterization of about 45 rust races (Rodrigues *et al.*, 1993; Várzea et al., 2002a). Among these, five physiologic races were reported to exist in Ethiopia (Meseret *et al.*, 1987). Molecular studies to detect genetic diversity in *H. vastatrix* were carried out by Nandris *et al.* (1998). The RAPD (Random Amplified Polymorphic DNA) method revealed polymorphism between individuals. However, a linkage between the molecular markers obtained and the pathotypes used was not established. In recent studies at CIFC, using RAPD and MSP-PCR (Microsatellite-Primed Polymerase Chain Reaction), a considerable degree of variability among the populations studied was observed, although no clear relationship was obtained between host, geographical origin and physiologic race (Gouveia et al., 2005).

## 3. OCCURRENCE OF COFFEE LEAF RUST IN ETHIOPIA

Investigations of the occurrence of major diseases were carried out in four different rainforest regions of the southeast (Harenna in the Bale Mountains) and south west (Bonga, Berhane-Kontir and Yayu) of Ethiopia. disease frequency of indigenous coffee in the four major rainforest areas in 2005 is taken to represent the situation in general during the investigation period of 2003–2008.



Fig11. Disease incidence in indigenous coffee 2005 (Zeru A. et al, 2009)

A large number of urediniospore samples were collected in the Ethiopian rainforests and identification was carried out during 2003/04 in the Institute of Botany,Tübingen University (Ritschel A.,2005). Measurements of urediniospores of CLR from the indigenous coffee population revealed detailed data with typical sizes for the species of *H. vastatrix* and had spore dimensions between  $29.7\mu \times 18.9$  (minimum) and $34.5\mu \times 23.7$  (maximum). These spore sizes were compared with those identified in highly susceptible Ethiopian selections such as Arba, Guga and Harrar and others from Indonesia and Colombia. The results showed that measurements were to a large extent identical and confirmed the presence of the species *H. vastatrix* (Table1).

Location	Coll. date	Length	Width	Variations
I. Harenna 1	8.2004	33.7	22.1	$31 - 36 \times 21 - 23$
II. Bonga 1	5.2004	32.7	23.7	$29 - 36 \times 21 - 26$
II. Bonga 2	11.2003	30.9	20.4	$30 - 33 \times 20 - 22$
II. Bonga 2	11.2003	30.5	21.2	$29 - 32 \times 20 - 23$
II. Bonga 2	11.2003	30.1	19.8	$28-31 \times 18-21$
II. Bonga 2	11.2003	30.0	20.5	$28 - 32 \times 20 - 22$
II. Bonga 2	11.2003	30.9	20.4	$30 - 33 \times 20 - 22$
II. Bonga 3	1.2004	30.3	18.9	$29 - 33 \times 18 - 20$
II. Bonga 3	5.2004	31.8	23.3	$27 - 37 \times 20 - 26$
III. Berhane-Kontir 2	1.2004	33.2	19.5	$30 - 36 \times 18 - 21$
III. Berhane-Kontir 2	5.2004	29.7	21.9	$26 - 33 \times 17 - 25$
III. Berhane-Kontir 3	1.2004	32.1	19.9	$30 - 34 \times 19 - 21$
IV. Yayu 1	11.2003	30.4	20.4	$29 - 32 \times 20 - 22$
IV. Yayu 1	5.2004	30.3	22.7	$27 - 34 \times 20 - 25$
IV. Yayu 2	11.2003	31.2	19.8	$30 - 33 \times 19 - 20$
IV. Yayu 2	5.2004	34.4	21.3	$31 - 38 \times 19 - 23$
Mean	2003/04	31.61	21.19	$26-40 \times 17-26$

Table1. Sizes of urediniospores of Hemileia spp.

The identification proof of the species *H. vastatrix* by morphological characteristics was assisted by scanning electron microscopic photos of rust sori and urediniospores(Ritschel A.,2005). A typical sorus extruding from a stoma on the lower side of the leave shad15–25 lemon-shapedone-celled urediniospores



Fig12. Urediniosorus and urediniospores of Hemileia vastatrix.

Chala *et al.*(2010) suggested that the CLR assessments in the rainforests of Ethiopia revealed its presence in all fields differing in incidence with time (season) and location. A significantly high rust incidence of 31.1% was recorded, for instance, in 2008 at Yayu, followed by Berhane-Kontir (21.4%) and Bonga (7.9%) in forest coffee populations. Rust incidences were consistently highest in Yayu, lower in Berhane-Kontir and lowest in Bonga forests during all seasons. The occurrence of rust in the forest coffee populations varied significantly from season to season. Higher rust incidences were found in

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January (29.6%) and April (22.7%), while lower incidences were observed in July (13.9%) and October (14.3%). Comparing rust occurrence during the complete period of the surveys from 2003 to 2007 a slight increase of the disease could be observed in the wild coffee population (Fig. 13).



**Figure13.** Coffee leaf rust incidence (%), severity (%) and sporulation lesion density (SLD) in three montane forest coffee populations of southwestern Ethiopia (July 2007-April 2008). Values were mean of four assessments (July 2007, October 2007, January 2008 and April 2008) for each disease parameter at each location. Bars with the same letters within each disease parameter are not significantly different according to LSD at P = 0.05.



Fig14. CLR severity during 2003 and 2007 in indigenous coffee populations of Ethiopia (Zeru A. et al , 2009).

## 3.1. Race types in Ethiopia

There was little emphasis on race-typing of Ethiopian rust samples until the beginning of the 1980s and the 1990s, when the Institute of Biodiversity Conservation (IBC, formerly gene-bank) included coffee in their conservation system. Wondimu *et al.*, (1987) observed that race III was the most frequent in forest coffee and race II in other areas. Other races were I, X and XV. In 2005 the first race-typing of CLR collections of indigenous coffee was carried out at the Centre of Coffee Leaf Rust Research (CIFC) in Oeiras, Portugal using their differentials (Varzea, personal communication). In this study the race specification identified race II at Berhane-Kontir and race III and X in Bonga with corresponding virulence genes v 1, 4 and 5 (Adugna G. *et al*, 2008).

## 4. INHERITANCE OF RESISTANCE

Based on quantitative and Mendelian genetic studies (Bettencourt AJ, 1988), the occurrence of at least nine dominant resistance (*R*) genes (SH1-SH9), singly or associated in *Coffea* spp., and a similar number of fungal virulence genes virulence (v1-v9), has been inferred. Major genes SH1, SH2, SH4 and SH5 were found in pure Arabicas from Ethiopia origin, the gene SH3 is considered to derive from *C. liberica*, and SH6, SH7, SH8 and SH9 were only found in Hibrido de Timor (HDT) (*C. arabica* x *C. canephora*) derivatives, therefore supposedly coming from the Robusta side of the hybrid (Bettencourt and Rodrigues Jr., 1988). Besides these SH genes it is likely that other major and minor genes might condition the coffee-rust interactions (Bettencourt and Rodrigues Jr., 1988).

It is thus commonly accepted that the outcome of coffee/rust interactions, whether the plant resists pathogen attack (incompatibility) or develops disease (compatibility), relies on the gene-for-gene model

(Flor, 1942) which has been recently amended (Jones JD and Dangl JL., 2006). Once delivered into coffee cells, *H. vastatrix* effector proteins, and the intracellular perturbations that they trigger, are supposed to be perceived by specific R-proteins. The recognition step promotes the launching of signaling defense pathway(s) and subsequent resistance. Alternatively, virulent rust races are believed to secrete effectors that escape or even counteract the host surveillance system, which allow for the high jacking of coffee cell metabolism and tissue colonization (Fernandez D, 2011).

The coffee genotypes are classified based on complete resistance or susceptibility to several rust races. Group A, characterized by resistance to all the known rust races, such as hybrids between *C. Arabica* x *C. canephora*, either spontaneously as in the HDT or man-made as in Icatú (D'Oliveira *et al.* 1979). Plants of group A have also been found in *C. liberica*, *C. dewevrei*, *C. eugenioides*, *C. congensis*, etc. (D'Oliveira and Rodrigues Jr. 1961) while the E-group, characterized by susceptibility to almost all known races, includes the traditional Typica and Bourbon cultivars (Bettencourt and Rodrigues Jr., 1988).

#### 5. DURABILITY OF RESISTANCE

In the last few years, some improved commercial varieties from HDT and other interspecific tetraploid hybrids gradually losing their resistance to leaf rust in some countries, due to the appearance of new virulent races (Rodrigues *et al.*, 2000). Some genotypes of coffee varieties, however, maintain their resistance and others, although infected in the field, present an incomplete type of resistance with others heavily infected, suggesting that they probably possess a polygenic type of resistance, like the variety Colombia (Alvarado, 2005). On the other hand, some Arabica varieties like Rume Sudan and Tafarikella with low yields and classified at CIFC as belonging to the susceptible group E, showed a very high partial resistance in the field for many years (Várzea et al., 2000).

At CIFC the level of resistance of HDT derivatives and some lines of Rume Sudan are now being reevaluated. Even though several varieties of coffee that are resistant to *H. vastatrix* have been used for introgression purposes (Bettencourt AJ *et al.*,1988) such alternatives are time-consuming and do not provide durable resistance due to the rapid co-evolution of races of the fungus that harbor new virulence genes (Várzea VMPand Marques DV, 2005). Therefore, additional methods to control leaf rust in the fields are required.

#### 6. CYTOLOGICAL AND BIOCHEMICAL RESISTANCE MECHANISMS

During incompatible interactions with biotrophic pathogens, the plant resistance phenotype results from the onset of a complex and multilayered-defense response, which is the so-called hypersensitive response or HR (Cacas JL., 2010), Although little is still known about the molecular mechanisms that govern resistance to *H. vastatrix*, several studies have advanced the case for the existence of a HR-like phenomenon in coffee plants. Resistant varieties that were inoculated with avirulent fungal strains displayed a morphotype that exhibits many HR characteristics. These include rapid host cell death, which is localized at the infection site and that is associated with fungal hyphae collapse (Silva MC *et al.*, 2002), callose encasement of haustoria and subsequent cell wall lignification (Silva MC *et al.*, 2002), early oxidative burst (Ganesh D *et al.*, 2006), and the activation of typical defense-related genes (Ramiro D *et al.*, 2010). For a number of coffee (*Coffea* spp.) genotypes, the resistance is post-haustorial and is expressed by the rapid hypersensitive cell death (HR).

Cell death began to be observed around 2 days post-inoculation, in the guard cells only, or in both the guard and subsidiary cells at the infection sites in which the fungus reached the stage of appressorium or penetration hypha (Silva 1996; Silva *et al.*, 2000, 2002). Death of subsidiary and mesophyll cells invaded by a haustorium was observed from the 3<sup>rd</sup> day after inoculation. During the time course of infection, cell death spread to adjacent epidermal and mesophyll noninvaded cells, as has been generally described for other coffee resistant genotypes (Martins *et al.*, 1985; Rijo *et al.*, 1991).

## 7. DISEASE MANAGEMENT

## 7.1. Fungicides

There is no quick solution to this problem. The outbreaks have already occurred, and are known to proliferate into multi-year events. The usual means of keeping the fungus at lower level is no longer solutions to this problem. Since coffee rust spread over the producing regions of the world, fungicides have been used to lessen the outbreaks. Copper based fungicides are most common, but have short

periods of effectiveness and applications must be timed carefully. In the end, these metallic fungicides can be detrimental to the environment and are limited in capacity to combat coffee rust.

There has been some research into a more biological or organic approach to combat the rust. Certain 'hyperparasitic' fungi (that are parasitic to parasites) have been identified in nature that preys on the coffee rust fungus (Muller et al. 2009). One such fungus, known as the 'white halo' fungus, has been getting lots of press lately. Scientifically, there is some evidence that this and possibly other microorganisms can reduce the viability of the rust (Jackson *et al.*, 2012). However, there has never been any practical application suggested or implemented for this biological control in coffee.

Unfortunately, at this juncture there is no real way to implement these additional microorganisms on a large agricultural scale. Nor has their ability been proven to eradicate large rust outbreaks, at the size and severity that the industry is currently faced with.

## 7.2. Coffee Breeding for Resistance

Since 1927 the Central Coffee Research Institute (CCRI) in Balehonnur, in India, has carried out an important national breeding programme. In the longer term, it will become critical to develop high quality resistant varieties. Kent was perhaps the first variety to show good resistance to coffee rust in India. However, this resistance was lost after about 10 years of exposure (Rodrigues and Eskes 2009). This phenomenon of gradually losing resistance has also been noticed in some *C.liberica* and *C. canephora* varieties. This led to the discovery that there were many 'races' of coffee rust and that new races could develop (Muller et al. 2009). Today, there are over 45 races identified, although a select few are likely responsible for most outbreaks (Fernandez *et al.*, 2012; Rodrigues and Eskes 2009). In Ethiopia, Catimor J-19. Catimor J-21, and Gesha show partial resistance to the rust, lowland areas with an altitude ranging 1000–1550 m.a.sl, but will break with new race of the pathogen.

In the coffee-growing countries, among progenitors for disease resistance were Hibrido de Timor, Rume Sudan, Kaffa and Geisha which were crossed to varieties SL 28, SL 34, N 39, KP 423 and H 66 (van der Vossenand Walyaro, 1980, 1981). The breeding programme of CIFC was essentially based on the utilization of HDT as a resistant parent. The main hybrids produced at CIFC with HDT were: HW26 = Caturra Vermelho x HDT 832/1; H 46 = Caturra Vermelho x HDT 832/2; H361 = Villa Sarchi x HDT 832/2; H528 = Catuaí Amarelo x HW26/13; H529 = Caturra Amarelo x H361/3.

#### 8. SUMMARY AND CONCLUSION

Coffee is a vital crop in social, cultural and national economy of Ethiopia. Despite coffee plays dominant role in social, cultural and national economy, the country's coffee industry is potentially at risk due to biotic and abiotic stress related with climate change. Among biotic stress like disease; coffee leaf rust (*Hemileia vastatrix*) is one of the major disease which has been causing high crop lose worldwide including Ethiopia. Ethiopia as the source of origin for perhaps both, the host and pathogen, plays an important role in science either for breeders or pathologists. Knowing the biology of the pathogen and epidemiology of the disease is vital for designing mitigation from its hazardous effect. The assessment study revealed that Coffee leaf rust occurs in Ethiopia in nearly all areas and under all growing systems like wild, forest, and garden and plantation coffee. For further genetically, morphological and phytopathological investigations on the host and pathogen the last ecosystems of rainforest/wild coffee in Ethiopia urgently need to be protected.

In the last decades, considerable success has been attained in the use of classical breeding for resistance to control economically important plant diseases, such as coffee leaf rust. However, in the last few years, some improved commercial varieties are gradually losing their resistance to rust due to the new increase of H. vastatrix virulence races. This shows the importance of re-evaluating existing varieties with the new rust races and the care needed before the release of new ones. Priority must also be given to the identification of sources of more durable resistance. It has long been recognized that more effective durable forms of disease control might be devised if we had a better acknowledge of both the dynamics of the pathogen populations and the factors that determine host resistance or susceptibility. In bringing different research approaches together (genetics, molecular biology, cytology and biochemistry), important advances have been made recently in coffee resistance, particularly against leaf rust. In the future, significant contributions can be expected to improve the durability of coffee resistance to both diseases that take into account the environmental needs and the development of a sustainable coffee economy.

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