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Relationships between host growth dynamics in maize rust pathosystem and spatio-temporal epidemics of *Puccinia sorghi* Schw. in Hararghe Highlands, Ethiopia

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Abstract

An epidemic is the progress of disease in time and space. Each epidemic has a structure whose temporal dynamics and spatial patterns are jointly determined by the pathosystem characteristics and environmental conditions. One of the important objectives in epidemiology is to understand such spatio-temporal dynamics via mathematical and statistical modeling. Maize growth occurs during the course of an epidemic and these changes in maize leaf area may influence the rate at which the disease increases and the shape of the epidemic curve. Thus, erroneous conclusions about the nature of the observed disease progress may be drawn if host growth is disregarded in the analysis of epidemics. This limitation can be overcome by adopting methods of correcting for host growth when calculating the apparent infection rate. Host plant resistance is also an important factor that influences the rate of epidemic development. There are various types and levels of host plant resistance that may vary from small, where the rate of disease increase is slowed but only slightly, to large where incomplete pathogenesis occurs and pathogen reproduction is slowed to a greater extent. The dynamic interaction of the components of an epidemic and their changes over time due to the external variables influencing them can be quantitatively analyzed through modeling. Comparative epidemiology is regarded as an important research tool in which studies across plant disease epidemics are conducted. The aim is to evaluate the differences and similarities between diseases and their hosts, or their relevant attributes and parameters. For example, the influence of environmental factors and human interferences on epidemics may be compared across different climatic conditions or agricultural practices either within the same disease host combination or for multiple disease epidemics. Ultimately, comparative epidemiology is of great importance in developing integrated and sustainable crop protection strategies. In this paper, we outline common methodologies that are used to quantify and model spatio-temporal epidemic dynamics of plant diseases, with emphasis on developing temporal forecast models and on quantifying spatial patterns of common rust of maize. Therefore, the objective of this review article is to elucidate the role of host growth modeling and spatio-temporal epidemics of *Puccinia sorghi* under Ethiopian condition. This information can be used by plant pathologists and physiologists in designing and modeling the relationship between the effects of the pathogen on maize growth dynamics and the spatio-temporal epidemic progression of the disease before undertaking an ecologically based management strategies against the disease. Hence, it is recommended that modeling maize growth dynamics and the biology of the pathogen helps in easily understanding the nature of epidemic development of the disease on spatial and temporal scales.

Keywords: Host growth, Pathosystem, Comparative Epidemiology, Epidemics, Models; Logistic regression, Logistic, Gompertz

Introduction

Maize growth occurs during the course of an epidemic and these changes in maize leaf area may influence the rate at which the disease increases and the shape of the epidemic curve. Thus, erroneous conclusions about the nature of the observed disease progress may be drawn if host growth is disregarded in the analysis of epidemics. The dynamic interaction and relationships of the components of an epidemic and their changes over time due to the external variables influencing them can be quantitatively analyzed through modeling. Such dynamics can be evaluated through comparative epidemiology which is regarded as an important research tool in which studies across plant disease epidemics are conducted. The aim is to evaluate the differences and similarities between diseases and their hosts, or their relevant

attributes and parameters. For example, the influence of environmental factors and human interferences on epidemics may be compared across different climatic conditions or agricultural practices either within the same disease host combination or for multiple disease epidemics. Mathematical modeling of crop disease is a rapidly expanding discipline within plant pathology. The first models of the temporal development of epidemics were developed by Van der Plank (1960; 1963), and have since formed the basis for disease modeling (Campbell and Madden, 1990; Madden *et al.*, 2007).

The dynamic interaction of the components of an epidemic and their changes over time due to the external variables influencing them can be quantitatively analyzed through modeling. In epidemiology, modeling aims to understand the main determinants of epidemic development in order to develop sustainable strategies for strategic and tactical management of diseases (Fininsa, 2001). According to their objectives epidemiological models can be classified in several ways. Kranz and Royle (1978) classified them into three types-descriptive, predictive and conceptual.

Plant diseases cause serious losses in yield of many food crops globally. It is estimated that at least 10-16% of the global food production is lost due to plant diseases annually. Consequently, more than 11% of the global population faced a serious food shortage while about 19% live on less than $1 a day. These facts draw attention to the necessity of developing and implementing adequate, economically feasible and environmentally acceptable control strategies to suppress plant disease epidemics and thus avert potential crop losses (Campbell and Madden, 1990).

A significant progress has been made over the last century in the management of plant diseases. For instance, the development of crop cultivars that are resistant to disease and the integrated use of chemical, biological as well as cultural control methods have had a major impact on agricultural productivity (Madden et al., 2007). This success is in part attributed to an increased understanding of how diseases develop in host plant populations and how various biotic and abiotic factors influence their epidemic development (Xu, 2006). It follows that as more information about each of the major and sub-components of an epidemic are known, the better it is to describe the epidemic and predict its direction and severity at a given time or space (Agrios, 2005).

The interactions of the components of a plant disease epidemic, i.e. host plant, pathogen, and environment, have been often viewed as a disease triangle (Campbell and Madden, 1990; Agrios, 2005; Pangga et al., 2011). Thus, a disease is capable of developing and progressing only if a virulent pathogen and a susceptible host plant are present under favorable environmental conditions. Since the activities of humans may also have considerable influence on disease epidemics, this review has included human interferences on plants, pathogens and the environment as a component of a plant disease epidemic and growth modeling.

It is widely acknowledged that diseases interfere with the different physiological functions of the host plant and their growth but they do so in processes that are often dynamic and complex (Agrios, 2005).Based on the type of damage they cause on crop growth and yield, pests in general and plant diseases in specific can be classified into seven categories: tissue consumers, leaf senescence accelerators, stand reducers, light stealers, photosynthetic rate reducers, assimilate sappers, and turgor reducers (Booteet al., 1983).

Two general categories can be drawn from this classification: either effect on radiation interception or on radiation-use efficiency. In this connection, comprehensive study on the relationship between host growth dynamics and pathogen modeling is not so far studied in Ethiopia for maize rust pathosystem and other diseases. Therefore, the objective of this review article is to elucidate the role of host growth modeling and spatio-temporal epidemics of *Pucciniasorghi* under Ethiopian condition. This information can be used by plant pathologists and physiologists in designing and modeling the relationship between the effects of the pathogen on maize growth dynamics and the spatio-temporal epidemic progression of the disease before undertaking an ecologically based management strategies against the disease.

Bio-trophy and Host Growth Dynamics: Plant pathogenic fungi are also often broadly divided into bio-trophic and necrotrophic fungi based on their modes of nutrition (Lopes, 1999). Bio-trophic pathogens, such as the rust and powdery mildew fungi, are parasites that feed on living host tissue, and therefore do not kill their host plants immediately. Thus, a highly specialized as well as structurally and biochemically complex relationship exists between bio-trophic pathogens and their host.

Bio-trophic pathogens such as the rusts are able to penetrate the host, evade detection or suppress immune responses while, simultaneously, diverting the plants' nutrients using specialized feeding structures such as haustoria to further their own growth at the expense of plant growth. Generally, bio-trophic pathogens do not produce toxins and only secrete limited amounts of lytic enzymes in exceptional cases.

In contrast, necrotrophic pathogens, such as the blight and rotting fungi, are facultative saprophytes that actively destroy host tissue using various phytotoxins, cell wall degrading enzymes and other depolymerising enzymes that are secreted both prior to and during colonization (Lopes, 1999; Stone, 2001; Agrios, 2005). These destructive pathogenesis mechanisms often result in extensive necrotic lesions which are photosynthetically unuseful and culminate in plant death and decay. Necrotic lesions induced by some necrotrophic fungi pathogens not only hinder photosynthesis in the necrotic spots, but also interfere with photosynthesis of those leaves lower in the canopy by intercepting light before it reaches them. For instance, Booteet al. (1983) demonstrated for *Cercospora* spp. on peanut (*Arachishypogae*L.) that photosynthesis of diseased plant canopies was reduced not only by loss of leaves which abscised as a result of infection, but also because diseased leaves that remained on the plants were less efficient in fixing CO_2 .

Although extensive studies have been dedicated towards understanding pathogen dynamics and the effects which diseases have on their host, there has also been a renewed and greater interest than just a few decades ago to integrate host dynamics in the analyses and description of the dynamics of disease epidemics (Campbell and Madden, 1990; Madden et al., 2007; Pangga et al., 2011). This arises from the knowledge that changes in the size and characteristics of a host that occur during their growth and development influence epidemic progression by either increasing the amount of susceptible leaf tissue (Ferrandino, 2008) or decreasing the disease severity through growth flushes of the host and defoliation of already diseased area (Hau, 1990).

Moreover, changes in the size, density and architecture of the canopy modify the prevailing canopy microclimate which in turn influences disease progression. Leaf wetness, temperature, wind, and radiation are important microclimatic components that influence plant disease epidemics (Pangga et al., 2011).

Host plant resistance is also an important factor that influences the rate of epidemic development. There are various types and levels of host plant resistance that may vary from small, where the rate of disease increase is slowed but only slightly, to large where incomplete pathogenesis occurs and pathogen reproduction is slowed to a greater extent. The dynamic interaction of the components of an epidemic and their changes over time due to the external variables influencing them can be quantitatively analyzed through modeling (Campbell and Madden, 1990).

By definition, a plant disease model is a simplified conceptual representation of the interactions between a pathogen/disease, a host plant, and the environment that determine whether and how an epidemic develops over time and space. In epidemiology, models are an essential tool in understanding, describing, predicting, and comparing epidemics or their components. For example, disease forecasts provide information that enable growers to make timely and tactical disease management decisions (Madden et al., 2007).

Some of the earliest works in modeling epidemics of plant diseases were done by van der Plank (1963). Subsequently, growth curve analysis, empirical models, analytical, and simulation models, among others, have been employed in modeling disease development of many pathosystems (Campbell and Madden, 1990; Hau, 1990; Xu, 2006; Madden et al., 2007; Pangga et al., 2011).

However, based on the aforementioned reasons, it is apparent that modeling studies that account for the synchronous interaction of the host and disease under given conditions of the environment would offer a better description of the variability in epidemic behavior and the capacity of host dynamics to modify epidemic progress (Calonnec et al., 2008) than studies that only focus on pathogen development. Specific research topics are the epidemiological consequences of a changing host area either through growth flushes of the host or loss of the diseased area through leaf abscission (Hau, 1990).

Certainly, there may be instances when it is not critical to correct for host growth in epidemic models, for instance in the case of systemic diseases where host growth does not essentially influence disease progress (van der Plank, 1963; Madden et al., 2007) or in epidemics such potato late blight (*Phytophthora infestans*) that increase rapidly over a short period of time thus there are only slight changes in the host area. In these instances, the basic assumption of the availability of a constant host area that can be diseased would be sufficient. However, for many pathosystems such as coffee rust, barley powdery mildew (Hau, 1990), and Alternaria blight of pigeon pea.

Modeling Host Growth Dynamics: Host growth occurs during the course of an epidemic and these changes in host area may influence the rate at which the disease increases and the shape of the epidemic curve (Campbell and Madden, 1990). Thus, erroneous conclusions about the nature of the observed disease progress may be drawn if host growth is disregarded in the analysis of epidemics. This limitation can be overcome by adopting methods of correcting for host growth

when calculating the apparent infection rate, such as proposed by van der Plank (1963).

The other important feature in modeling host-disease interactions is the loss of the diseased area through leaf defoliation. However, this aspect is often left unquantified or ignored in many modeling studies. To our knowledge, Waggoner (1986), Jeger (1986) and Madden et al. (2007) have made significant contributions towards incorporating defoliation in plant disease models. However, a key limitation of their models is that the total amount of leaf area formed as well as the total defoliated leaf area increase over time without bound. One of the primary reasons for modeling disease epidemics and their interaction with host dynamics arises from our desire to compare epidemics.

Comparative Epidemiology: Comparative epidemiology is regarded as an important research tool in which studies across plant disease epidemics are conducted. The dynamic interaction of the components of an epidemic and their changes over time due to the external variables influencing them can be quantitatively analyzed through modeling. Comparative epidemiology is regarded as an important research tool in which studies across plant disease epidemics are conducted. The aim is to evaluate the differences and similarities between diseases and their hosts, or their relevant attributes and parameters.

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Fundamentally, the development of successful disease control strategies as well as accurate estimation of future crop losses depends on the understanding of the epidemiological variables that govern the dynamic interactions between the host and the pathogen systems (Newton et al., 1995; Bergamin Filho et al., 1997; Xu, 2006; Pangga et al., 2011). Since the pioneering work of Van der Plank (1963), there has been significant interest in developing models which incorporate the dynamics of host growth and epidemics of a disease (Boote et al., 1983; Jeger, 1986; Waggoner, 1986; Hau, 1990; Madden et al., 2007; Ferrandino, 2008; Calonnec et al., 2008).

These modeling approaches led not only to a better understanding of how diseases affect their hosts but also gave insight on the reciprocal effects of host factors on epidemic development (Boote et al., 1983; Pangga et al., 2011). For the bean rust pathosystem and particularly with regard to empirical quantification of host disease interactions, the contributions of Mersha and Hau (2008; 2011) are to our knowledge some of the most notable. Moreover, Berger et al. (1995) developed and validated (Amorim et al., 1995) FERRUGEM, a simulation model of bean rust epidemics based on infections of *U. appendiculatus* that occurred on daily cohorts of bean leaves. However, what was not included in their model was the acceleration of defoliation due to disease and the contribution of total lesion (the entire chlorotic area including the pustule and surrounding halo) to disease progress.

Similarly, a study conducted by Hau (1990) drew attention to the epidemiological consequences of a changing host either by an increase of susceptible tissue through growth flushes of the host or by the loss of the diseased tissue through defoliation. Moreover, disease-induced defoliation, besides being an important injury component leading to yield loss, may also strongly influence the course of an epidemic by reducing the amount of inoculum present within the canopy. Amongst others, Waggoner (1986), Jeger (1986) and Madden et al. (2007) have incorporated the negative effect of defoliation and disease on host growth in their models. However, a major drawback to these models is the inherent characteristic that total leaf area formed as well as total leaf area defoliated in the disease situation will increase over time without any limitation. This is in agreement with the present observation made in the maize-common rust pathosystem at Hararghe areas eastern Ethiopia in which maize leaves started defoliating after 35 days of common rust development on maize leaves leading to reduced photosynthetic area and carbohydrate production.But so far quantification of common maize rust pathogen in Ethiopia has not been conducted and thus, this information can be used to include maize rust pathogen monitoring system in maize pathology research for sustainable production of the crop in common rust prone areas such as the Hararghe highlands.

Modeling Common Maize Rust Disease Progress: In a model analogous to that of polycyclic inoculum production, the rate of change in disease is proportional to amount of disease at any point in time. Therefore, in differential form, the equation to describe polycyclic epidemics is:

$dx/dt = xr$

As with the monocyclic model, x is a dimensionless proportion between zero and one,

and r is a constant that depends on the aggressiveness of pathogen, the susceptibility of the host, the environmental conditions, etc., averaged over the course of the epidemic. In this case, the slope, dx/dt, is proportional to x, and therefore disease progress increases with time at an increasing rate. In the integrated form the model is:

$x = x_0 e^{rt}$

Where x_0 is the proportion of disease at the start of the epidemic and e is the base of the natural logarithm. Vander plank (1963) called r the "apparent infection rate" because it is based on the appearance of disease symptoms, which lag behind the actual infections. It is defined as the rate of disease increase per unit of disease and has the units of proportion per unit of time. The parameter x_0 is sometimes carelessly called initial inoculum, to which it is quantitatively related, but strictly speaking it is the initial disease (a proportion).Graphically the model has the familiar form of the exponential model:

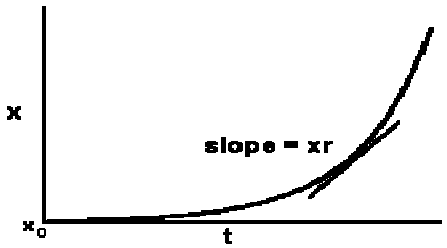

Fig 1: Exponential model for common rust disease

The above models assume unlimited growth of disease, which, of course, is impossible; the proportion of diseased plants or of diseased tissue cannot exceed one. We can adjust our models to address this issue by using a correction factor (1-x) to represent the proportion of healthy tissue remaining. A decrease in the remaining healthy tissue reduces the chance for new infections and therefore reduces the rate of disease progress. As x approaches one, there is no healthy tissue left, and the rate of the epidemic slows to zero.

The monocyclic model of disease progress, adjusted for the limit to disease is:

dx/dt = QR(1-x)

Graphically we see an epidemic that starts out looking linear, but as x approaches 1, the slope decreases to zero.

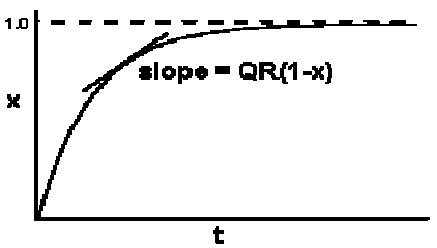

Fig 2: Upper limit of exponentialgrowth of common rust disease

In the polycyclic model we make a similar adjustment:

dx/dt = xr(1-x)

This model starts out approximately exponential, but its slope also decreases and approaches zero as xincreases and approaches one. The result is a sigmoid-shaped curve:

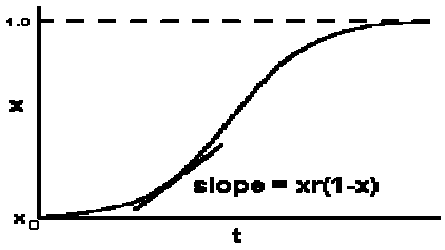

Fig 3: Sigmoid-shaped curve for common rust growth

In reality, it is rare that disease incidence or severity exceeds 50%, and when it does, the disease progress curve is usually not quite sigmoid.

The monocyclic model: In the case of the monocyclic model, if the observed x's are transformed to the natural logarithm of 1/(1-x), and these transformed values are plotted against t, we will get a straight line with a slope equal to QR.

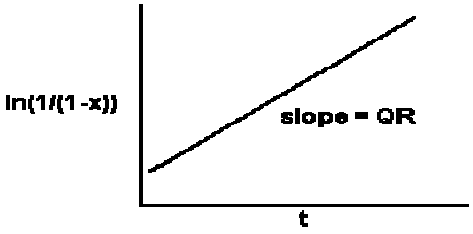

Fig 4: Linearized graph of common rust growth

Then with an independent estimate of the initial inoculum, Q,we can calculate R.

The polycyclic model: If the observed x's in a polycyclic epidemic are transformed to the natural logarithm of x/(1-x), and the transformed values are plotted against t, the result will be a straight line witha slope equal to r and an intercept equal to the natural log of $x_0/(1-x_0)$.

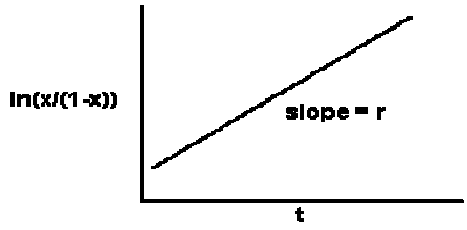

Fig 5: The polycyclic model for common rust epidemics

The Maize-Rust Pathosystem: Resistance of maize plant to common rust is also an important factor

that influences the rate of epidemic development in maize population at field level. There are various types and levels of maize plant resistance to this pathogen that may vary from small, where the rate of disease increase is slowed but only slightly, to large where incomplete pathogenesis occurs and pathogen reproduction is slowed to a greater extent.

Maize-rust pathosystem consists of a maize plant and the pathogen under prevailing environmental conditions. Determining the role that a specific pathogen plays in limiting yield necessitates quantitative knowledge of the interaction between host and pathogen as well as of the influence of biotic and abiotic factors on the host-pathogen interaction itself. Under favorable conditions for infection and disease development, rust pathogens constrain the development of maize plants in various ways, for instance by leaf tissue destruction, reduction of the photosynthetic rate, altering dry matter partitioning as well as accelerating dry matter loss through premature leaf senescence and defoliation (Boote et al., 1983; Robert et al., 2004; Agrios, 2005).

Conversely, during their growth and development, maize plants integrate all external factors from the environment and cultural practices, which in turn affect their interaction with pathogens through varying levels of susceptibility. The nature and levels of intrinsic host resistance and age-related resistance associated with specific host tissues is also a critical factor in determining epidemic development (Mersha and Hau, 2011). Quantitative description of the temporal disease progress on its host is an important step in elucidating the epidemiology of any plant disease. Here the pathosystem maize-common rust was investigated.

Production constraints of maize in Ethiopia: Maize (*Zea mays* L.) is an important component of farming systems and staple food crop in sub-Saharan Africa. In Ethiopia it is a staple food crop and one of the main sources of calories in the major maize producing regions. It is cultivated on about 2.135 million hectares of land. The national average yield of maize under subsistence production is 3667 kg/ha. This is too much below the world's average yield which is over 6520 kg/ha. This low yield is attributed to several factors among which foliar diseases and insect pests are the major once (CSA, 2017).

Common maize rust is one of the most dominant and economically important foliar diseases of maize in the world including Ethiopia. *Puccinia sorghi,* the causative agent of the disease is a macrocyclic, heteroecious rust and the most important spore stage is the uredospore, which is the repeating stage of the fungus. This pathogen has high variability in virulence, as numerous virulence phenotypes (races) identified on differential lines are known to exist. It is considered to be obligate parasite and survives only on living maize plants or the alternate hosts. Since the spores are windblown, transmission may occur over long distances, leading to rapid spread of the disease.This disease affects the photosynthetic efficiency of the crop through nutrient depletion and impairing radiation use efficiency leading to poor crop stand and yield loss(Bekeko, 2017).

Economic Importance of Common Maize Rust: Assefa and Tewabech (1993) reported common maize rust as one of the major diseases of the crop in Ethiopia. Assefa (1999) reported more than 60% incidence of common maize rust at Bako, Gudar and Melko. It has a serious impact on plant growth characters and is an important limiting factor to grain production wherever susceptible maize is grown. However, national yield losses due to the common maize rust are difficult to estimate because of the limited availability of precise information on prevalence, incidence and severity of the disease.

Average yield losses attributable to natural common maize rust infection can range between 7 and 35% on susceptible cultivars in U.S.A, while in western Ethiopia the losses were reported to reach up to 23% (Assefa and Tewabech, 1993). An experiment conducted at Awassa from 1995 to 1997 on common maize rust indicated a significant difference among disease parameters, grain yield and thousand-kernel weight.

Reductions of 22.6 and 8.4% in yield and thousand-kernel weight, respectively, were observed on naturally infected maize plots (EARO, 1999). The relationship between the disease incidence and yield loss was found to be linear and estimates of yield reductions ranged from 3 to 8% for each 10% of leaf area infected (EARO, 1999). At Bako, assessments of common rust for two years showed significant negative correlation (r= -0.93) between severity and yield (Assefa and Tewabech, 1993). Mean rust incidence of 69-75% and severity of 48-57% were also recorded in maize cropping systems of Hararghe region (Fininsa and Yuen, 2001). However, rust incidence and severity among the areas and between years varied, probably because of several factors such as the susceptibility of the plant variety (host), presence of a virulent disease-causing agent (pathogen), and a favorable air and soil environment (Grothe*t al.,* 1983).

Manifestation of Common Maize Rust: The most characteristic symptom of common maize rust is the oval elongated golden or cinnamon-brown pustules scattered over both surfaces of leaves.

Common rust pustules begin as small, circular, light green to yellow spots in the leaf tissue. These spots develop into circular to elongated, golden-brown to reddish-brown, raised pustules.

The pustules may be scattered across the leaf tissue or may occur in bands or concentrated patches while the leaf is still in the whorl or on the bend of the leaf. The pustules quickly rupture to reveal masses of rusty-red to brown spores and these on maturity turn blackish-brown because of replacement of uredospores by teliospores (Agrios, 2005). However, symptoms vary depending on the susceptibility of the variety, age and variety of the host, part of the plant infected and spore stage of the rust.

Experimental evidence indicates that older, mature tissue is more resistant; younger tissue or tissue with delayed maturation is more susceptible. In susceptible varieties, the fungus grows extensively, sporulates abundantly and the lesions increase in size and this is usually accompanied by the development of a yellow halo. In most resistant reactions, the fungus causes only chlorosis, but occasionally small pustules may also develop. In some resistant genotypes, rapid cell necrosis or hypersensitivity occurs and very limited fungal growth is seen (Van Dyke and Hooker, 1969a). In infected host cells, Golgi apparatus and endoplasmic reticulum increase in amount and rate of host respiration increases more significantly in resistant and susceptible maize plants than non-infected plants (Van Dyke and Hooker, 1969b). The increase in respiration may correspond to the increase in the number of organelles in infected host cells. It has been suggested that the fungus regulates or directs host metabolism for its own benefit (Van Dyke and Hooker, 1969b). The close association of haustoria to host nuclei and chloroplasts may enhance direct control of certain host metabolic activities by the fungus.

Epidemiology of Common Maize Rust: Maize is the major host of *Puccinia sorghi*. The alternate host of this pathogen is *Oxalis corniculata*. The uredial stage, which is responsible for repeated cycles of infection, is adapted from cool to moderate temperature conditions wherever early morning dews predominate. As a result, the disease is serious in areas where cool to moderate temperatures and high relative humidity prevail. It is reported that the maximum and minimum mean temperatures of 13 and 24 ^{o}C are within the optimum range for common maize rust development and spore germination during the main season (Fininsa, 2001). The optimum temperature for common maize rust infection ranges from 16 to 23 ^{o}C (Fininsa and Yuen, 2001). In the tropics, common maize rust tends to cycle endemically on higher altitudes.

*Pucciniasorghi*is macrocyclic, heteroecious rust. The most important spore stage is the uredospore, which is the repeating stage of the fungus. The pathogen has high variability for virulence, as numerous virulence phenotypes (races) identified on differential lines are known to exist. It is considered to be obligate parasite, i.e. it survives on living maize plants or the alternate hosts only (Agrios, 2005; Deyet al., 2012). Since the spores are windblown, transmission may occur over long distances, leading to rapid spread of the disease.

Viability and germinability of uredospores can be affected by temperature, light and humid conditions, in which the uredospores are produced, germinate and cause infection. About six hours of moisture (wetting period) is required for rust spores to germinate and infect plant tissues (Van Dyke and Hooker, 1969a). In Hararghe region, the prevailing weather conditions are conducive for common maize rust development. Recently introduced hybrid maize varieties are more susceptible to rust under Hararghe conditions than the locally adapted ones. The hybrid varieties may not have resistance gene(s) against prevailing *P. sorghi*races in Hararghe or the conditions are more favourable to the rust (Fininsa and Yuen, 2001). Although the race composition of *P. sorghi*is not known from this region, variability in virulence and pathogenicity cannot be ruled out.

Common Maize Rust Management: Several methods are available to manage common maize rust, but none has been totally satisfactory. Attempts have been made to manage common maize rust through cleaning up of alternate hosts, cultural practices, and use of chemicals. Breeding for disease resistance has been done in U.S.A. Although breeders have followed different approaches to achieve greater efficiency and higher yield, no disease resistance breeding program was executed in Ethiopia. However, materials exhibiting best yields were evaluated for disease resistance (Assefa and Tewabech, 1993).

Currently cultural methods depend largely on the use of early planting and intercropping of maize with other crops. Delayed planting may increase the disease pressure on maize as the late-planted hybrids are still in the susceptible whorl stages in the later part of the season. Consequently, late planted fields may develop heavy epidemic. However, onset of rainy season, which dictates planting time, is not the same every year in Ethiopia; hence, changing date of planting may or may not be helpful in controlling the disease. In intercropping systems, common maize rust spreads slowly from the infected to healthy plants (Fininsa, 2001).

Experimental results from Bako research site showed that intercropping of maize with haricot

bean resulted in low common maize rust severity as well as better economic return from the crops. Assefa and Tewabech (1993) also reported that maize intercropped with sweet potato showed reduced levels of common maize rust intensity when both crops were planted at the same time. Integrating early planting with reduced maize density in intercropping and educating the users about the scheme may offer an option to manage common maize rust. In addition, diversity in the cultivars grown can provide substantial benefits to farmers. Although adoption of intercropping can reduce the disease to some extent, it cannot fully control the disease.

Moreover, adoption of such technology on a large scale may not be feasible. In recent years, systemic plant immunization as an alternative method for disease control has been demonstrated successfully in various agricultural crops. Restricted inoculations with pathogens or non-pathogens and treatment with simple inorganic organic chemicals, which release immunity signals, have provided a potential technology for the management of some maize diseases caused by fungi.

Foliar application of essential nutrients has also been used to improve the quality and yield of maize. Chemicals such as phosphates, which might be used as foliar fertilizers, are the target agents for inducing resistance to common maize rust. According to Reuveni*et al*. (1994), spraying maize leaves with phosphate compounds at least 2-4 hours before inoculation induced systemic resistance to *P. sorghi*, and this induced resistance was not specific to the pathogen.

Chemical Control: The use of fungicides for management of common maize rust has been studied for many years and certain chemicals have been found to be effective. These fungicides include the C-14 demethylation inhibitors, isomerase and reductase inhibitors, succinate dehydrogenase inhibitors and complex III inhibitors, which are being used for managing rusts caused by *Puccinia*spp. in different crops. According to Assefa (1997), a combined application of mancozeb and propiconazole at the rate of 2 kg a.i. per ha each (2 to 3 applications at ten-day interval) effectively controlled common rust. Earlier zineb and maneb were recommended for the management of common rust and spraying these fungicides against common rust gave 28 g increase in thousand-kernel weight (Teclemariam, 1985).

Disease Progress and Maize Leaf Area Relationships: Generally, simulations of the disease progress and of the different leaf areas, i.e. healthy, diseased and defoliated area were considerably consistent with experimental observations (R^2 > 0.97). Specifically for common rust, a host growth rate *rH*of 0.045 to 0.053 day–1, defoliation rate *rD*of 0.250 to 0.53 day–1 and disease rate *rY*of 0.315 to 0.350 day–1 were estimated in two experiments while the rates in the other experiment clearly differed. Except for slight deviations, there were no considerable differences between progress curves of either host or disease dynamics under a constant or variable disease rate. Moreover, the models showed that the contribution of defoliated healthy area to total plant defoliation is insignificant.

The healthy leaf area duration (*HLAD*, cm^2 - days) was computed as:

$$HLAD = \sum_{j=1}^{m-1}\left(\frac{\left(H(t_j)+H(t_{j+1})\right)}{2}\right)\cdot\left(t_{j+1}-t_j\right)$$

Where *H* is the healthy leaf area on a whole plant basis,

The non-linear relation between yield (g plant-1) and *HLAD* was described by a

Monomolecular function using the combined data of NI, I + F and I treatment

$$AUDPC = \sum_{j=1}^{m-1}\left(\frac{\left(y(t_j)+y(t_{j+1})\right)}{2}\cdot\left(t_{j+1}-t_j\right)\right)$$

Similarly, to compare epidemics, the area under disease progress curve (*AUDPC*, proportion-days) was estimated using the trapezoidal integration method of Campbell and Madden (1990):Where *y* represents the disease severity (proportion) on a whole plant basis, *m* is the numberof assessments and (*tj+1* - *tj*) is the time interval between two consecutive assessments. From the early common rust of maize model, it was demonstrated that the diseased leaf area can increase up to 45% of the actual leaf area just within the early cycle of the epidemic (seven days after inoculation). Defoliation rates were 4.5 times higher in older plants (late inoculated) compared to the younger (early inoculated) plants. Similarly, the disease rate *rY*was three folds higher in the late inoculations (0.0380 and 0.0305 per day) when compared to the early inoculations (0.0151 and 0.062 per day).The progress curves of the cumulative total leaf area (cm^2/plant) were well described by the logistic and Gompertz growth functions with high values of coefficient of determination (R^2 > 0.92) in both experiments. The coefficient of determination (R^2) values indicated that the logistic function gave a better fit in 2013 while the Gompertz function was superior in 2014 (data not shown).

Simulation of Spatio-Temporal Epidemics of *Puccinia sorghi* from Hararghe Highlands: Simulations from the maize rust model showed that in the presence of the disease, the total host area production is significantly reduced and levels

off at proportions ranging from 0.05901 to 0.07668 of the maximum host area. Leaf defoliation rate is enhanced by more than five times in a diseased plant compared to the disease-free situation. The model also established that production of new healthy tissue is proportional to the healthy area, not the actual host area.

Given the good fit of models to the observations coupled with the biological realism of estimated parameter values, the models can be considered as satisfactorily describing the dynamic interaction between the disease epidemic and host growth in the pathosystems studied in this experiment. One of the important objectives in epidemiology is to understand such spatio-temporal dynamics via mathematical and statistical modeling. Maize growth occurs during the course of an epidemic and these changes in maize leaf area may influence the rate at which the disease increases and the shape of the epidemic curve. Thus, erroneous conclusions about the nature of the observed disease progress may be drawn if host growth is disregarded in the analysis of epidemics.

This limitation can be overcome by adopting methods of correcting for host growth when calculating the apparent infection rate, such as proposed by van der Plank (1963). Host plant resistance is also an important factor that influences the rate of epidemic development. There are various types and levels of host plant resistance that may vary from small, where the rate of disease increase is slowed but only slightly, to large where incomplete pathogenesis occurs and pathogen reproduction is slowed to a greater extent.

For example, the influence of environmental factors and human interferences on epidemics may be compared across different climatic conditions or agricultural practices either within the same disease host combination or for multiple disease epidemics. Ultimately, comparative epidemiology is of great importance in developing integrated and sustainable crop protection strategies.In this paper, we outlined common methodologies that are used to quantify and model spatio-temporal dynamics of plant diseases, with emphasis on developing temporal forecast models and on quantifying spatial patterns of common rust of maize.

Spatio-temporal epidemics of *Puccinia sorghi* under Ethiopian condition based on an empirical field research conducted at west Hararghe in 2013 and 2014 main cropping seasons. But detailed analysis of modeling maize growth dynamics was not conducted. Therefore, this information can be used by plant pathologists and physiologists in the future to model the relationship between the effects of the pathogen on maize growth dynamics and the spatio-temporal epidemic progression of the disease while undertaking an ecologically based management strategies against the disease.

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Compliance with Ethical Standards:

Author Contribution: The author set the experiment conducted the analysis and made the review based on the existing experiences and knowledge gap in Ethiopia on this pathogen.

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