Epidemiology of Wheat Rhizoctonia

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Abstract- *Rhizoniasolani* [*Thanatephoruscucumeris*] is a soil-borne pathogen occurs worldwide which causes economically important diseases to variety of vegetable and field crops, cereals especially wheat, ornamentals, and fruit and forest trees. The inflicted yield losses caused by this pathogen reaches up to 20% yearly worldwide on wheat crops.Environmental factors such as cool temperatures, wetter soils, and high humidity, planting date, winter temperatures, and disease levels in the previous wheat crop has been recorded to influence the occurrence on the field (Meyer, 2002). The objectives of this article is to present an overview of the epidemiology of Rhizonia on wheat.

Index Terms- Rhizoctonia, Epidemiology, Winter wheat, plant disease

I. Introduction

Winter wheat (*Triticumaestivum*) is an important staple grain crop found in cool and temperate countries. It acts as a food source for humans. It is usually planted in the autumn to germinate and develop into young plants and resume growth in early spring(Żółtańska, 2005). Wheat production is usually affected by some fungal pathogens (González García *et al.*, 2006). Among these pathogens is *Rhizoctonia spp*. causing various diseases affecting wheat production(Hamada *et al.*, 2011). In previously published literature, two anamorph species of Rhizoctonia which causes disease on wheat production are widely recognised and studied. These include *Rhizoctoniasolani* and *Rhizoctoniacerealis which are* common pathogens affecting various crops especially wheat(Sneh *et al.*, 1991).

R. solani [*Thanatephoruscucumeris*] is a soil-borne pathogen occurs worldwide which causes economically important diseases to variety of vegetable and field crops, cereals especially wheat, ornamentals, and fruit and forest trees. *R. cerealis*van der Hoeven (teleomorph: *Ceratobasidiumcereale*DIL Murray and LL Burpee) is a stem-base disease such as sharp eyespot on wheat and are present in temperate growing regions. The inflicted yield losses caused by this pathogen is averaged up to 20% yearly worldwide on wheat crops (Shu *et al.*, 2014). Although the severity of this disease caused by Rhizoctonia pathogen on wheat varies from year to year between regions under different environmental conditions at their time of planting (Zhang and Dernoeden, 1997). The objectives of this article is to present an overview of the epidemiology of Rhizonia on wheat.

II. The Fungus – Rhizoctonia

2.1 History and Taxonomy

The fungus, *Rhizoctonia*was first described by Julius Kuhn who observed a fungus on diseased potato tubers and named it *Rhizoctoniasolani*in 1858 (Ogoshi, 1996, Sneh *et al.*, 1991). This earlier work done by Kuhn in 1858 on epidemiology of diseases caused by *R. solani* has been reviewed by Baker and Martinson (1970). *Rhizoctonia spp. (phylum: Basidiomycota, class: Basidiomycetes, genera: Thanatephorus, Waitea, Koleroga, Ceratobasidium, Uthatobasidium, Athelia and Botryobasidium)* is one of the most important destructive soil-borne disease to most agriculturally important plant such as wheat*(Triticumaestivum)*(Ogoshi, 1987, Ogoshi, 1996). Rhizoctonia species are widely studied, particularly *Rhizoctoniasolani and Rhizoctoniacerealis*(Burpee*et al.*, 1980; Budgea e*t al*, 2009). *R. solani*occurs as a necrotrphic parasite which causes damping-off, root rot and stem based disease on numerous host plant species and alsoexist as thread-like growth on plants or in culture media (Figure 1)(Lewis and Lumsden, 2001). *R. cerealis acts* as causal agent of sharp eyespot disease. Areport by Hamada *et al*. (2011) suggested that the spread of disease caused by *R. cerealis* could be as a result of climate change, their means of dispersal, sclerotia formation, and environmental conditions that are favorable for the disease to grow (Robertse *et al.*, 1995). Sclerotia formation of *R. solani* are initially developed from the combination of small groups of hyphae which arise from irregular branching of neighboring hyphae(Otten and Gilligan, 1998).

Environmental factors such as cool temperatures, wetter soils, high humidity, planting date, winter temperatures, and disease levels in the previous wheat crop has been recorded to influence the occurrence on the field (Meyer, 2002). Kataria and Verma in 1992 found that the most virulent AGs of *R. solani towards* wheat were those that belonged to AG2-1 and AG4 causing pre- and post-emergence damping off, seedling root rot or basal stem disease.

2.2 Biology of *Rhizoctonia spp.*

The original concept that *Rhizoctonia spp*. causes rotting of roots and damping off of seedling was first observed by Ogoshi(1975), with an assumption that isolates of *R. solani*belong to basidiomycetous imperfect fungus. *R. solani*do not produce any asexual spores (also called conidia), despite the absence of asexual spores this fungus are recognized by some specific characteristic features such as their mycelia. *R. solani* possess these characteristics features, which include

i. Branching near the distal septum of cells in young vegetative hyphae(8-12 µm)
ii. Presence of hyphae and septum (Weber, 2009)
iii. Formation of septa from a point near the origin of hyphal branches (Parmeter and Whitney, 1970)
iv. Absence of clamp connections (Ogoshi, 1996, Sneh et al., 1991)
v. Absence of conidia sclerotia(Ogoshi, 1987)

The isolates of *Rhizoctonia spp.* tends to produce a simple or branched chain of cells called moniliod cells or chlamydospores. The moniliod cells chain is formed on or above the surface of a host plant or a media substrate aggregating to form sclerotia. It has been recorded that hyphae of the same AG group of *R. solani*are able to fuse and are therefore genetically isolated despite being morphologically similar(Ogoshi, 1996, Sneh *et al.*, 1991). *Rhizoctonia spp.* can be identified according to number nuclei present in each cell. The number of nuclei produced per cell of Rhizoctonia is then used for taxonomic identification purposes (Kuramae*et al.* 2003).*R. cerealis*has been recognised as a binucleate species having two nuclei and *R. solani*is a multinucleate species, with at least three nuclei in eachcell. Further classification of *Rhizoctonia spp.* are grouped base on their ability to fuse with one another through anastomosis (Sneh *et al.*, 1991, Ogoshi, 1996). However, some researchersrecognise *R. solani* as a group of filamentous structure which are based on their hyphal fusion, known as called Anastomosis groups (Anderson, 1982, Ogoshi, 1987).

2.3 The geographical distribution of *Rhizoctonia spp.*

Rhizoctonia spp. are plant pathogenic fungi that are distributed world-wide with a wide range of host plants. They cause a great loss in wheat quality and production due to this disease (Sayler and Yang, 2007). The global discovery of Rhizoctoniaoccurrence more than 100 years, existing as a thread-like growth on plants or in culture. Report on global distribution of *R. solani*on wheat production are limited to US Pacific, South Africa, Australia, Turkey, Canada (Demirci, 1998), United Kingdom (Dillion and Garrett, 1943), Africa and Asia (Hammouda, 2003; Tsuda*et al.*, 2000), Oceania (Cromey*et al*., 2002; Cromey*et al*., 2006), North America (Wiese, 1987), and Asia (Tsuda*et al.*, 2000) (Figure 1). The universal nature of *R. solani* over the last 30 years is yet to be recognised. The geographical distribution and development of plant disease which depends much on the climatic factors such as rainfall, humidity, temperature etc. The spread of *R. cerealis* has been suggested to be a climatic change, and the global dispersal of susceptible varieties or the large host variety of the pathogen (Hamada *et al*., 2011). Due to the global distribution of Rhizoctonia pathogens causing disease in wheat, there is an ongoing research on the taxonomic amendments of teleomorphs for these anamorphic fungi (Dillion and Garrett, 1943, Papavizas *et al.*, 1975)

Figure 1. Geographical distribution of *Rhizoctoniacerealis* in countries worldwide. (Hamada *et al.*, 2011)

2.4 Anastomosis (hyphal fusion)

In the last 35 years, the system of anastomosis grouping of Rhizoctoniahave been based on hyphal fusion among several fungi that consist the genus(Sneh *et al.*, 1991, Anderson, 1982). The anastomosis of *Rhizoctonia spp.* can be defined as the union of two fungal hyphae between different isolates of Rhizoctonia(Naito, 2006). These are classified with an isolate tester which are describe based on their anastomosis reaction between the fungal hyphae(Naito, 2006, Sneh *et al.*, 1991). In the previous studies, terminologies to define anastomosis has been divided into four different types of category to describe their reaction between paired fungal hyphae(Carling, 1996). This includes:

i. C0 - No fusion nor reaction (Neate and Warcup, 1985)
ii. C1- Contact fusion
iii. C2 - Imperfect fusion
iv. C3- Perfect fusion (Ogoshi, 1987, Sneh *et al.*, 1991)

C0 indicates that there is no fusion (contact) between different anastomosis groups (AGs) of the hyphae and are not related genetically (González García et al., 2006); C1 show that there was no clear penetration (Figure 2).

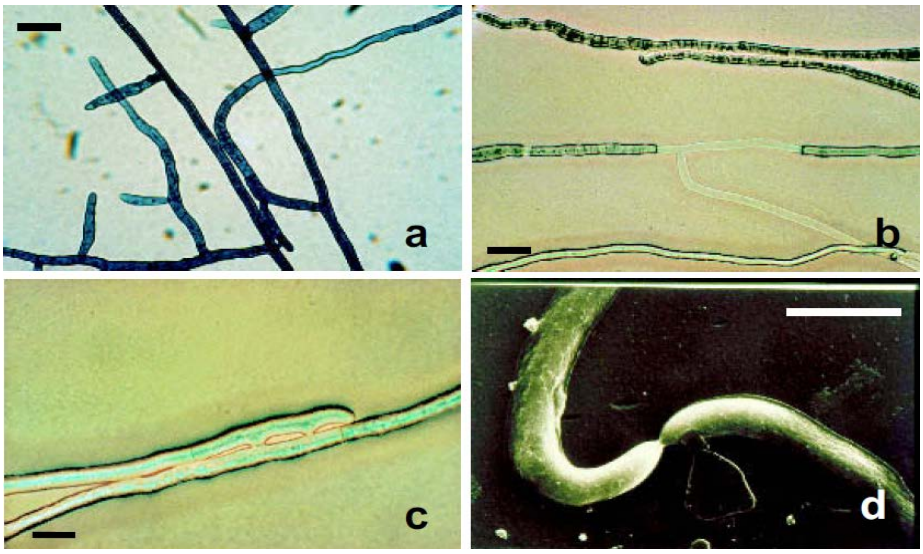

Figure 2. Anastomosis reactions of *R. solani*. strain (a) No reaction, when strains of different anastomosis groups show no hyphal attraction. (b) Incompatible reaction, when strains of the same anastomosis group but with different vegetative compatibility genes undergo hyphal fusion, (c)Imperfect fusion and d) Perfect fusion (Garcia *et al.*, 2006, Robinson & Jim Deacon]

2.4.1 Anastomosis groups of *Rhizoctonia spp*.

The anamorphic classification of Rhizoctonia isolates are based on characterization of the cell's nuclear condition, their vegetative hyphae and hyphal anastomosis. Research carried out by Cubeta and Vilaglys (1997) grouping of Rhizoctonia anastomosis haveindeed advanced our understanding of their genetic diversity within Rhizoctonia species.

The perfect fusion of AGs of *Rhizoctonia spp.* can be determined when C3 reaction (Figure 2) are observed between a pair of isolates indicating a genetic or nearly genetically related between Rhizoctonia isolates. Recent studies on the anastomosis reaction of Rhizoctonia isolates has shown variability within and between AG's (Neate and Warcup, 1985). Moreover, this C3 reaction has been considered insufficient for accurate identification and classification of the isolates, because some isolates lose the capability to self-anastomose (Ogoshi, 1996, Sneh *et al.*, 1991). Some will not anastomose with all isolates of the same AG and some anastomose with isolates of more than one AG (Ogoshi, 1987, Sneh *et al.*, 1991). Research in further studies has also shown that AG's have levels of host specificity and currently fourteen different AG's of *R. solani* are recognised (AG-1 to AG-13 and AGBI) including isolates from many crops. According to research work of Burpee *et al*., 1980; *Rhizoctonia*species were assigned to one of these seven AGs termed CAG-1 to CAG-7. This seven AG's have been subgroups into further division (AG-1, AG-2, AG-3, AG-4, AG-6, AG-8 and AG-9)(Burpee and Martin, 1992) to reproduce the differences in the occurrence of anastomosis, pathogenicity, and cultural appearance among isolates. However till date, 12 AGs of *R. solani* have been described (AG 1 to AG 11, plus AG BI) which are based on their hyphal fusion (Gill *et al.*, 2001a). The identification of these isolate are majorly on the basis of appearance of mature colonies on PDA in petri dishes whether *R. solani* or on *R. cerealis.* To test for the hyphal anastomosis, an isolate of the unknown AG will be co –cultured on water agar with a tester isolates of a known AG (Tsai *et al.*, 2012, Tu and Kimbrough, 1973). This anastomosis grouping generally helps with the understanding of diversity and sequence information amongRhizoctoniaspecies supporting the existence of genetical distinction in the AGs (Sharon *et.al*, 2008) of most widely studied species.

2.5 Host Range of Rhizoctonia

Rhizoctonia spp. are distributed widely in a great diversity on their host plant (Table 1) (Sayler and Yang, 2007, Ganeshamoorthi and Sunil, 2013). The host range of *R.solani* has been has been assumed to have at least 14 anastomosis groups (AGs) reported by Carling 1996 and Sumner *et al.*, 2003. Rhizoctonia species other than *R. solani* and *R. cerealis* are believed to have little or no role in causing disease towards various crop especially wheat (Zhang and Dernoeden, 1997).

2.6 The Disease: Infection and Disease Symptoms of Rhizoctonia on wheat

2.6.1 Disease symptoms

Rhizoctoniaprimarily affect the host's stem and root due to its saprophytic nature both below and above ground the soil surface (Bailey *et al.*, 2000). Rhizoctonia fungussurvives and develops to attack tips, hairs root and stem of wheat (Weller *et al.*, 1986; Mazzola *et al.*, 1996). In previous studies, multinucleate isolates belonging to anastomosis groups (AG) 2–1, 2–2 and 8 of *Thanatephoruscucumeris* were most pathogenic to wheat (Roberts and Sivasithamparam, 1986). It has been suggested that the bare patch disease is caused by a complex of root rot fungi composed of one or more anastomosis groups of *Rhizoctonia* spp. and other associated fungi. *Rhizoctonia spp.* are accountable for diverse types of infected diseases on wheat production (Fletcher *et al.*, 2010) under hugely differing environmental conditions. Wheat seedling infection caused by isolate of *R. solani* AG 2-1 are more favourable to cool weather at their sowing dates in spring whereas for AG-4 isolates, warmer environment are condusive for damping-off to grow

(Yitbarek *et al.*, 1988, Ogoshi, 1996, Sneh *et al.*, 1991). In a country like China, sharp eyespot is principally caused by the *Rhizoctoniacerealis*, the anamorph of *Ceratobasidiumcereale* D.I. Murray and Burpee. Although the disease pathogenicity of *R. solani* AG's such as AG1-IB, AG2, AG4 and AG5 and some other factor causes infection in wheat which eventually makes the plant very weak and stunts growth.

Among these important factors include the interactions between plant and pathogen (Strange and Scott, 2005) and the initial concentration of the inoculum (inoculum density), as well as the severity of Rhizoctonia. These infections caused by Rhizoctonia spp. on wheat crop are affected at the host's roots, leaves, and stems of the host due to their saprophytic nature.

Table 1. Host range summary of *Rhizoctoniasolani* and*Rhizoctoniacerealis* isolate based on their relationship (both multinucleate and binucleate) recognised with different disease and their host. Their biological species which are different from*Thanatephoruscucumeris* are names with question marks.

Anastomosis group	Diseases	Host Plant	Teleomorph
AG 1-IA	Leaf blight, summer blight, brown patch, sheath blight and sheath spot	Rice, corn, sorghum, bean, soyabean, turgass	*Thanatephoruscucumeris* (=*Corticiumsasakii; T. sasakii?*
AG 1-IB	Web blight, rot , bottom rot	Bean, rice, soybean, leguminous woody plants, cabbage, lettuce	*T. cucumeri, T. microsclerotius?*
AG 1-IC	Damping off and crown root rot	buckwheat, carrot, soybean, flax, pine	*T. cucumeris*
AG 2-1	Damping off, bud rot , leaf blight, root rot	Crucifers, strawberry, subterranean clover, wheat	
AG 2-2 IV	Root rot, leaf blight, and large patch, and black scurf	sugar beet, turfgrass	*T. cucumeris*
AG 3	Leaf blight, brown spot	potatoes, tobacco, tomato, egg plant	*T. cucumeris*
AG 4 (HG I, HGII and HGIII)	Fruit rot, stem rot, damping off and stem canker, root rot	Tomatoes, pea, potato, soyabean, onion, peanut,	*T. cucumeris*
AG 5	Black scurf, brown patch and root rot	Potato, turf grass, beans, beans	*T. cucumeris* (=*Pellicula**riapraticola*)
AG6(HG-I and GV)	Non-pathogenic group	-	*T. praticola*?
AG 7	Non-pathogenic group	-	*T. cucumeris*
AG 8	Bare patches	Cereals	*T. cucumeris*
AG 9	Weak pathogen	Crucifers, potatoes	*T. cucumeris*
AG 10	Non-pathogenic group	-	*T. cucumeris*
AG 11		Wheat, rye, oats, barley, corn, rice, grasses	*T. cucumeris*
AG BI	Non-pathogenic group	-	*T. cucumeris*
AG-D I, II, III (*Rhizoctoniacerealis*)	Sharp eyespot	Wheat, barley, oats, maize, rye and other *Poaceae* species	*Ceratobasidiumcereale*

(Carling *et al*., 2002,Sneh *et al.* (1991), Ogoshi*et al*., 1983)

2.6.2 Damping off caused by *R. solani*

Damping-off, a fungal disease caused by Rhizoctonia pathogen, tends to infect plant seedlings. This either kills the plant either before or after germinating from the soil (generally known pre-and post-emergence damping off). Infected seedlings not killed by the fungus often have cankers which occurs bydegenerating stem and root tissue at the stem and below soil line (Sneh, 1996). Most wheat seedlings are most susceptible to disease at their juvenile stage whenthe environment is favourable (wet weather and warm soil). Damping-off can be considered based on their invasion occurrence with symptoms of reddish-

brown lesions sometimes rotting completely or leaving behind a discoloured stump

2.6.3 Root disease caused by *Rhizoctonia spp.*

There are various diseases that affect cereals especially wheat. Among these are Rhizoctonia root rot(Sneh *et al.*, 1991), Rhizoctonia patch, and bare patch (Dillion and Garrett, 1943, Garrett *et al.*, 2006). Rhizoctonia root rot is more frequent and severe under reduced or conservation tillage(Oros *et al.*, 2013). With a close inspection of infected seedlings on the field, this shows brown discolouration or rotting of the roots indicate 'spearing tips' (Figure 3).*Rhizoctonia* is most evident as bare patches in a young crop plant (Figure 4). Close inspection of infected seedlings shows brown discoloration or rotting of the roots (Ogoshi, 1996, Sneh *et al.*, 1991). Diseases caused at the root region are majorly characteristics above the ground such as expansion of circular stunted patches and chlorotic plants. The maturation of crop are delayed for more than two weeks by the damages caused in the both absence and presence of patches of stunted plants (Mazzola *et al.*, 1996)

Figure 3. Spear-tipping symptom caused by Rhizoctonia root rot (bare patch) – with lesion at the tip of the root)

Figure 4. Brown bare patch (patches formed as a result of the sclerotia effect on the soil, debris and the plant) are caused by *R. solani*. (Macnish and Neate, 1996)

2.6.4 Sharp eyespot caused by *R. cerealis*

Sharp eyespot, are common stem based disease caused by *R. cerealis*, commonly found in wheat (*Triticumaestivum)*.However, sharp eyespot symptoms are easily identified at later growth stages GS 39 (flag leaf out)(Zadoks *et al.*, 1974). Plants that are affected shows lesions on the outer leaf sheaths near the base of the plant with a pale cream centre with a dark brown, sharply defined edge progressively extend through the leaf sheath layers (Figure 5) (Pitt 1964). Severe sharp eyespot infection of mature tillers may result in small, shrivelled grain, lodging and premature ripening. Sharp eyespot, generally known as stem based diseases are found throughout temperate wheat regions in the world (Lemańczyk, 2012). The average yield losses in wheat production over last five seasons in UK caused by *R. cerealis* is 5% moderate infection and 26% severe infections (Cromey et al., 2002). Wheat productions in China are majorly affected by sharp eyespot which has brought serious threat to crop production with estimated loss from the years 2005 to 2008 (Chen et al., 2008). Primarily sharp eyespot shows an oval-shaped lesion which grows on basal leaf sheaths, lower stem becomes brown in color cause stunting and reduction in tillers.

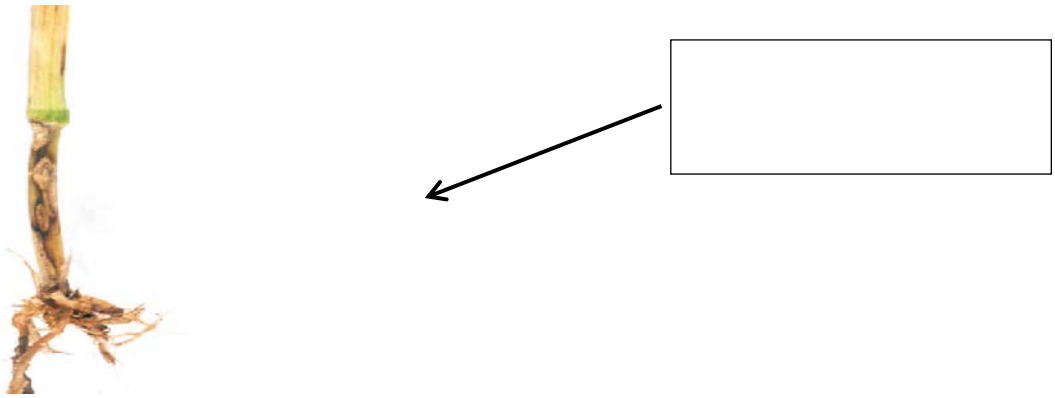

Figure 5. Symptom of the sharp eyespot disease caused by *R. cerealis.* (Source – R. V. Ray, pers. comm.)

2.6.5 Effect of disease caused byRhizoctoniain wheat

Root rot reduces the number of roots available for the plant nutrient and water uptake been disrupted. The record of disease incidence and severity caused by *R. cerealis* (sharp eyespot) (Colbach et al., 1997), has been a result of increase in the fungicide use, increase in the distribution of disease due to the environment. The wheat cultivars are more susceptible to R. *cerealis* (Cromey et al., 2005), and the occurrence of disease incidence tends to affect continual cropping of cereal crops (Clarkson, 1981, Clarkson and Cook, 1983). The major problem associated with damping-off is that this disease contributes to poor establishment of plant structure.Crop losses caused by the Rhizoctonia disease occurrence and development in plants are due to the interaction between the host, pathogen, environment, with man being the ultimate cause of disease, as conceptualized by the disease triangle. The effect of source, dispersal and reproduction of Rhizoctonia inoculum bring about loss in crop production. The yield and quality of wheat are reduced due to the impact of this disease pathogen.

2.7 Rhizoctonia life cycle

The disease cycle of *R. solani* is important in regards to management and control of the pathogen. *R. solani* can survive for many years by producing small, irregular-shaped, and brown to black structures known as sclerotia, in soil and on plant tissue. There are certain pathogens of *R. solani*which have evolved the ability to produce sclerotia with a thick outer layer that allows them to float and survive in water. *R. solani* also survives as mycelium by colonizing soil organic matter as a saprophyte, particularly as a result of the pathogenic activity of plant. Sclerotia and/or mycelium already present in the soil and/or on plant tissue germinate to produce vegetative threads (hyphae) of the fungus that can attack a wide range of food production (Figure 6).

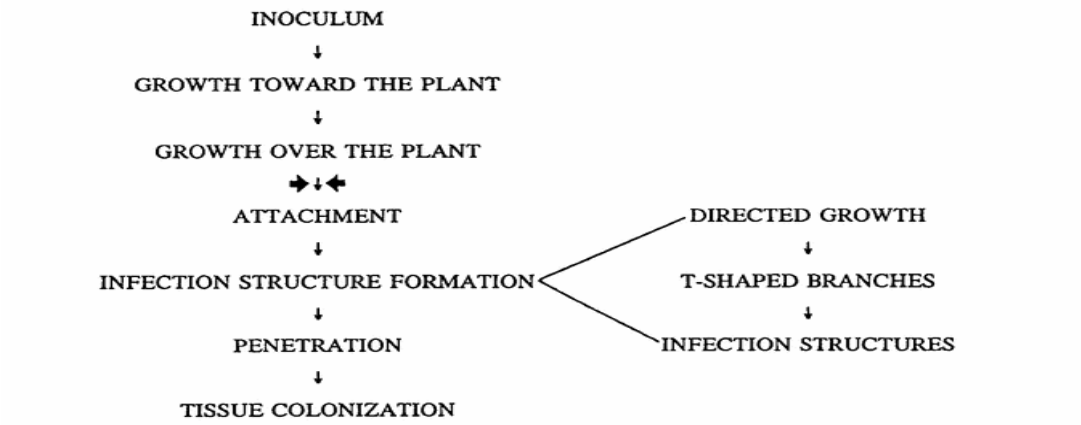

Figure 6. Disease cycle and the infection process of *Rhizoctoniasolani*. Initiation of the actual infection process is indicated by the two thick black arrows (Keijer1996)

The fungus, Rhizoctonia, is attracted to the plant by chemical stimulants released grown on plant cells and decaying plant residues(Naito, 2006). During the attraction process, the fungal hypha will come in contact with the plant and become attached to its external surface. After the fungus is attached to the plant, it grows continually on the external surface of the plant and then produces a specialized infection structure (either an appresorium or infection cushion) penetrating into the plant cell and releases nutrients for the fungal to survive, grow and develop (Naito, 2006). The infection processes are promoted by the production of many different extracellular enzymes that degrade various components of plant cell walls. As the fungus kills the plant cells, the hyphae then continue to grow and colonize the dead tissue of the plant cell, often forming sclerotia. The production of new inoculum is produced on or in a host tissue, and a new cycle are repeated when new substrates become fresh on the new plant emerging (Figure 7).

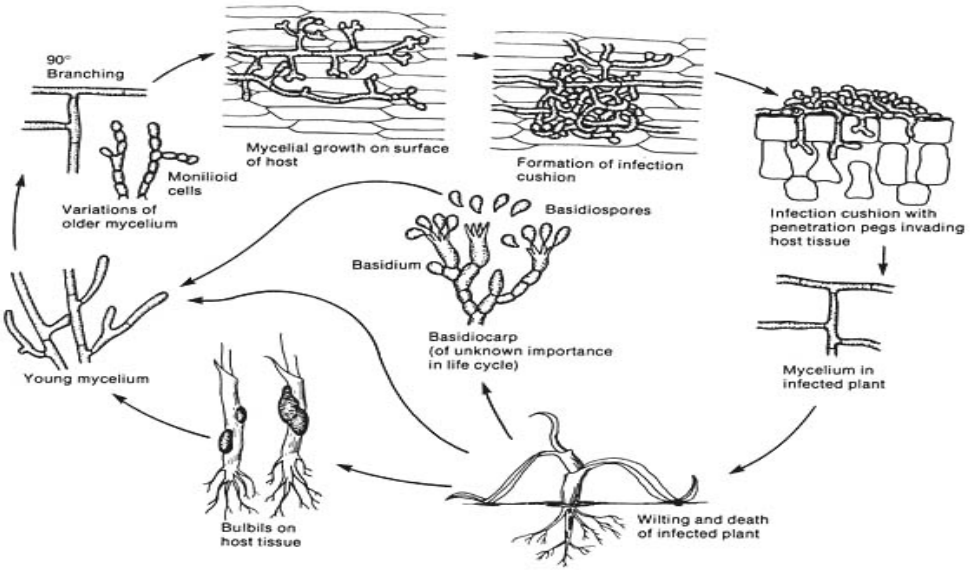

Figure 7.Disease cycle of *Rhizoctoniasolani* (Image taken from the Compendium of Turfgrass Diseases, APS Press)-(Tredway and Burpee), 2001)

2.8 Epidemiology of Rhizoctonia diseases

Epidemiology can be defined as the study of disease in the population of pathogen interaction with the population of host plant (Jeger, 2001, Cooke, 2006). In other words, epidemiologycan be defined as the interaction between a susceptible host (for example, winter wheat), a virulent pathogen (*R. solani*) under a favorable conditions. Rhizoctonia primarily causes disease and attacks majorly the stem and root regions (Ogoshi, 1987). Epidemiological studies of plant entails monitoring, experiments on loss assessment (Anderson, 1982), forecasting (Blumenthal *et al.*, 2001), analysis and modelling of disease progress (Waggoner and Aylor, 2000) to assess the disease risk and to formulate better management strategies (Zadoks, 2001). In the past years, the ecological and the epidemiological studies record of Rhizoctonia disease has resulted in a significant knowledge of their variation, variability, virulence, physiology mode of their infections (Sayler and Yang, 2007) which are largely determined by their soil or climatic condition. It has been suggested that infection by the fungus *R. solani*slowly increases with increasing soil moisture content (from 30 to 80%) and vice-versa (Verma, 1996). Scientific research based on the disease severity/complexity is greatly needed, particularly in areas where rapid field diagnosis, disease epidemiology and efficacy vector control are required for better management (Jeger, 2000).

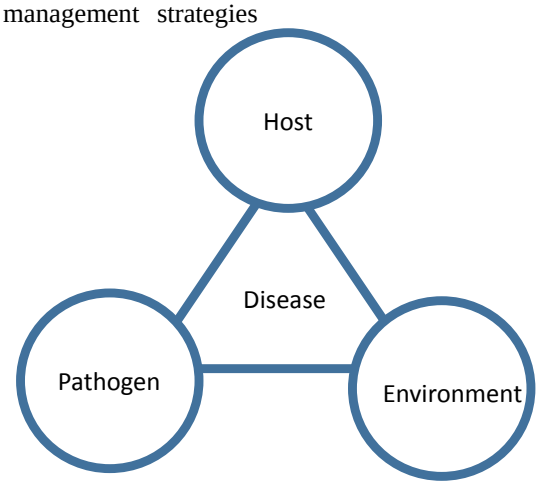

Figure 8. Plant disease triangle. The three necessary causal factors of disease. Disease triangle recognises the interaction of a virulent pathogen population with a susceptible host population in a conducive environment(Francl, 2001).

2.8.1 Source and survival of inoculum

The ability of this pathogen to establish disease depends on the susceptibility of the host to the pathogen. Environmental factors like moisture and temperature also play an important role in the survival of Rhizoctonia pathogen whilst establishing plant disease epidemics (Otten *et al.*, 2003, Otten *et al.*, 2004). The climatic condition is an important factor that relates epidemic development which varies widely from disease to disease. The formation of sclerotia (which act as source for the inoculum to survival), also permit the pathogen to survive without a host

nside the debris in the soil or organic matter (Papavizas *et al.*, 1975). Given the quantity of inoculum present in the soil, pathogens are easily transmitted to wheat seedlings and cause diseases (Jeger, 1987, Jeger and Van Den Bosch, 1994). Crop residue, stem, leaf debris and living plant are important sources of inoculum for the development of disease caused by *R. solani*and *R. cerealis.*

2.8.2 Dispersal of inoculum

Basidiospores serve as a means of dispersal of the *Rhizoctonia spp*. The dispersal of spores (known as basidiospores are initiated at high relative humidity and warmer temperature conditions (≥ 90% and ≥ 20°C respectively) (Naito, 1996). Under these conditions, basidiospores permit long distance dispersal of the fungus (Agrios, 2005). *Rhizoctonia spp.*produce basidiospores (reproductive spores) that lead to foliar diseases in some host crops. The role of basidiospores in disease epidemiology is yet to be clearly established since it can vary within AGs and between subgroups (Naito, 2006).

2.8.3 Reproduction of *Rhizoctonia spp*. Inoculum

Rhizoctonia spp. reproduces both sexually and asexually (Ogoshi, 1996; Roberts, 1999). Spores and fungal mycelia act as fruiting structures in inoculum production of pathogen of *Rhizoctonia spp.*, which consists of the spore-bearing formation (Naito, 2006). The fructification of *T. cucumeris* occurs when environmental factors, such as air and soil temperature, relative humidity including high level of oxygen (O_2) and carbon dioxide (CO_2) concentration are present (Naito, 2006). Asexual reproduction of Rhizoctonia is achievedby means of fungal hyphae (sclerotia), mycelium and by spore-formation (basiodiospore) (Anderson, 1982, Anderson *et al.*, 2004).

2.9 Factors affecting disease development caused by *Rhizoctonia spp.*

Rhizoctonia infections are established in the presence of virulent pathogen, susceptible host and availability of other environmental factors that either enhances Rhizoctonia pathogenicity or host susceptibility to infection (Figure 8) (Francl, 2001; Agrios 2005).

2.9.1 The pathogen

Factors that determine the occurrence of disease depend on the absence or presence of Rhizoctonia pathogen. The outbreak of disease in a plant community is initiated by the introduction of pathogen to that environment or in a specific area. The extent of disease which develop on winter wheat are mostly determined by the pathogenicity of the Rhizoctonia. The pathogenicity of Rhizoctonia are majorly related to the virulence (the ability of pathogen infection), and the vigour of the infection caused by this pathogen (inoculum density). The ability of the Rhizoctonia pathogen to adapt depends on their survivability in that environment, how it can dispersal over a long period of time. Rhizoctoniabeing a soil-based pathogen cansurvive in the soil for years and the amount of infective propagules which are available in the soil or crop debris tends to be a crucial factor affecting the growth and development of Rhizoctonia disease in *T. aestivum* (wheat). The survival of this infective propagules in the soil are influenced by the environmental factors (Section 2.8.3) that are suitable for the growth of disease pathogen.

2.9.2 The host

A susceptible host is another factor that causes disease development in a plant community. The important of planting of uninfected cultivar tends to reduce the effect of Rhizoctonia on winter wheat production. An extensively dense plantation can host epidemics, particularly if those anastomosis groups of Rhizoctonia are not present in that environment. The population structure and density will also affect the development of disease in a plant community. The density of the main host species and the proportion of other plants that are not hosts within the community will determine the rate and extent of epidemic development.

2.9.3 The environment

2.9.3.1 *Temperature and Weather condition*

Temperature is one of the major physiological factors that affect crop production(Gugel *et al.*, 1987, Yitbarek *et al.*, 1988). When the weather condition is humid and temperatures are stressful to the grass, Rhizoctonia Brown bare Patch tends to develop which is been recorded as is one of the most destructive Rhizoctonia pathogen. Gill, *et al.* 2001 viewed *R. solani*anastomosis group (AG8), with a conclusion that significant damage are caused when the temperature lower than 6 to 19°C at the root region, or when temperature which ranges from 16 to 27°C. Greater growth of *R. cerealis*occurs on soil at 20°C but slower growth occurs at 10°C indicating that temperature may be a factor of increased decomposition of soil organic matter (Herman *et al.*, 1992b). Disease severity was recorded at 10°C, advocating that cereal disease suppression occur in soil at higher temperatures. However, these Rhizoctonia pathogen disease are mostly favoured by temperatures of around 9-10°C (Wiese, 1987). Soil temperature and moisture play major roles in the development and spread of root diseases in wheat. A laboratory based studies was carried out by Herman (1992b) and found out that the influence of temperature majorly depended by the moisture content levels. There are various optimal ranges of temperatures affecting the AGs, both in disease growth and infection caused by *R. solani.* Change in climate constantly affect plants both in the natural and agricultural environments throughout the world (Anderson *et al.*, 2004, Garrett *et al.*, 2006). Temperature has a great influence on the growth and pathogenicity of *R. cerealis*, with pathogen prevalence and growth optimum at 16 to 20°C (Abdelshife and Jones, 1981).

2.9.3.2 Soil moisture content

Soil moisture levels on the incidence of *R. cerealis* were found to be similar in the results gathered by Gill *et al*. (2001b) in the laboratory and glasshouse. The disease incidence was observed to be higher in dry soils than in wet soils in his studies using non-sterilised soils(Gill *et al.*, 2001a). However, the authors found no difference in disease incidence at various soil moisture levels in sterilised soils. The soil environment tends to favour microbial spread of disease of *R. solani*and *R. cerealis*(Gill *et al.*, 2001b, Otten and Gilligan, 1998). Previous and ongoing research on the interaction between plant-soil-water relationships has not been successful given the inappropriate use

of quantity of water-holding capacity (Cook and Papendick, 1972). The presence of *R. solani*AG8 at soil moisture levels between 15 to 75% water holding capacity (WHC) has arguably been associated with high pathogen prevalence in the roots and subsequently taking its toll on wheat growth and development(Gill *et al.*, 2001a, Gill *et al.*, 2001b, Otten and Gilligan, 1998). The dry weight of root in an inoculated soil was related to soil moisture level, bringing a decline as WHC fell from 75% to 15%. This has led to a conclusion that at high moisture levels microbial activity is increased to a level that suppresses the pathogen.

2.9.3.3 Soil texture and pH

The spread of*Rhizoctoniasolani*on the soil surfaces tends to be faster when the soil profile is condusive to cause causing damping-off in wheat seedlings (Otten and Gilligan, 1998). Sandy soil has been recorded to be more favorable for *R. solani*(AG8) to spread, survive and grow compared to an heavier texture of soil resulting in greater loss in dry root weight. This was also supported by Otten*et al.* (2004a) who found that infection occurred quicker in sandy soil than othersoils. Gill *et al.*((2000) described the influence of soil texture in plant pathogen invasion. A wet soil has tendency of producing inoculum which causes a greater infection to crop (Gilligan and Bailey, 1997). Sharp eyespot caused by *R. cerealis*is favoured when the soil pH is acidic to some extent (Pitt, 1966, Pitt, 1964). The texture of soil has been the major factor influencing incidence of diseases such as sharp eyespot. Over 70% of the disease incidence of sharp eyespot has been recorded in the UK according to the observation by Pitt (1964) on light sandy soils. Further studies on the effect of soil was carried out by Daamen and Stol, (1990) indicated that the prevalence of sandy soils has been a major factor influencing disease occurrence.

2.9.3.4 Fertiliser use

The incidence of Rhizoctonia root rot can be reduced by the application of some nitrogen fertilizer such as ammonium sulphate, urea or sodium nitrate to fields (MacNish (1985).However, Pumphrey*et al.*, (1987) found that the applications of nitrogen fertiliser had no effect on Rhizoctonia root rot. This was later supported by MacNish (1988) who observed that N fertiliser failed to affect the incidence of Rhizoctonia root rot. Gill *et al.*, (2000) observed that the nutrient status of the soil had no influence on the pathogenicity or spread of the pathogen. Among the minor plants nutrients, the only one previously found to be inversely correlated to the incidence of root rot disease in wheat has been found to be zinc (Zn) (Thongbai*et al.*, 1993).The impact of applying high amount of nitrogen fertilizer to winter wheat production, particularly during spring and summer, favors development of Brown Patch by producing lush, succulent growth that is very susceptible to Rhizoctonia infection. Other factors increase disease severity by creating a humid environment favorable for growth of Rhizoctonia fungi. These factors include: overwatering, watering in late afternoon, poor soil drainage, lack of air movement, shade, a high mowing height, and overcrowding of seedlings. Excessive thatch, mowing when wet, and leaf fraying by dull mower blades also can enhance disease severity.

Table 2. General factors that affect Rhizoctonia disease development.

Pathogen	Host	Environment
Presence of pathogen	Crop susceptibility	Temperature
Pathogenicity	Growth stage & form	Rainfall
Adaptability	Population density & structure	Leaf wetness period
Dispersal efficiency	General health-Nutrient status	Soil properties
Survival efficiency		Wind
Reproductive fitness		Fire history
		Air pollution
		Herbicide damage

2.10 Control Measure

Control measure may be divided broadly into three- via crop management, biological and chemical means. Crop management is the considerable measure of crop manipulation for controlling the disease outbreak caused by Rhizoctonia.

2.10.1 Application of epidemiological principles to achieve plant disease control

There are several number of practices that illustrate particular epidemiological principles to reduce and control wheat disease caused by Rhizoctonia. Management of this disease effectively entails an application of integrated disease management approach and knowledge of each growth and development stages. The impact of source disease inoculum has emphasized the importance of strategies for controlling *R. solani.* Control of *R. solani*and *R. cerealis*which infect wheat requires the understanding of survival and dispersal of soil borne pathogen inoculum to provide information on disease management decisions.

2.10.2 Disease Control by Reduction of Initial Inoculum

Plant epidemics often result in loss of production, financial hardship to farmers and producers unless there is an appropriate control measures. The increasing knowledge of the interaction between host-pathogen-environment disease triangle has enabled us to apply certain principles to control disease invading wheat

production (Jones, 2004, Anderson *et al.*, 2004). Reducing the loss in wheat production caused by Rhizoctonia disease can generally be achieved if management efforts aim to eliminate the initial pathogen inoculum; discontinue the incidence and appearance of the inoculum production by slowing down the increase rate of disease and the time of exposure of the crop to the pathogen. These losses and damages caused by Rhizcotonia can be reduced by controlling epidemics using measures that minimize disease infection sources or suppress spread this disease. However, the 'economic threshold' varies with factors such as plant density, duration and pattern of infection, the extent to which infection decreases over yield and quality.

2.10.3 Cultural Method

MacNish (1985) suggested that ploughing or cultivation down to 8-10cm depth can be an effective cultural control option for Rhizoctonia. Timely elimination of volunteer plants and grass weeds, is seen as the most significant cultural control option for the control of Rhizoctonia root rot(Cook *et al.*, 1991, Cook *et al.*, 2002). Crop succession, soil tillage and sowing date are incidence factors that also influence the occurrence of sharp eyespot (Colbach *et al.*, 1997)

2.10.4 Biological control

Chen *et al.* (2010) showed that when agronomic practices such as straw mulching wereperformed,*Pseudomonad* spp. increased and a suppression of sharp eyespot occurred. Thisdemonstrated that it may be possible to induce bio-control agents through agronomic practices., as well as the biological control of *Rhizoctoniasolani* by indigenous *Trichoderma spp*. Isolates(Budge *et al.*, 2009).

2.10.5 Host resistance to *R. solani* and *R. cerealis*

The use of natural resistance expressed by the host is the most sustainable approach for soil-borne pathogens which are difficult to control chemically, with fumigation being an expensive and often temporary solution (Panella and Ruppel, 1996). There are however variations in the level of access to Rhizoctonia root rot-resistant wheat varieties among growers (Fletcher *et al.*, 2010). The level of resistance to sharp eyespot have differed among cultivars over the years (Hollins and Scott, 1983).

2.11 Identification of *Rhizoctonia spp.*

Molecular approaches used for the identification of the pathogen *R. cerealis*within infected wheat plants include PCR-RAPD (random amplification of polymorphic DNA) (Turner *etal.*, 2001), also suggested that molecular methods could be applied to discriminate infection by *R.cerealis*. Although RFLP, AFLP and RAPD-PCR are very effective molecular techniques for differentiating among *R. solani* AGs, conventional and real-time PCR are the preferred molecular techniques as they allow for the rapid detection of a particular *R. solani* AG present in plant tissue and in soil.

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