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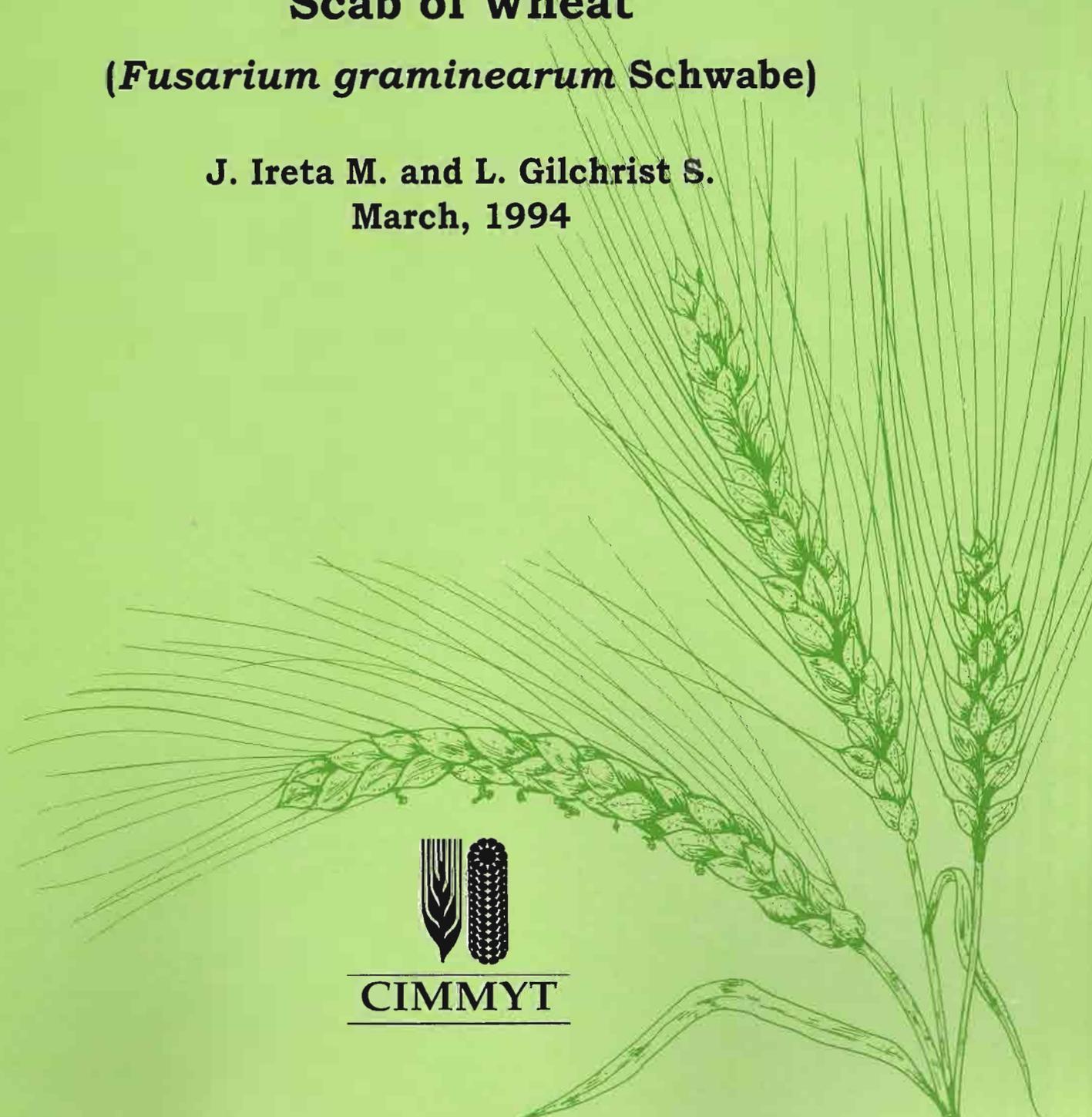
**Fusarium Head  
Scab of Wheat**  
*(Fusarium graminearum Schwabe)*

J. Ireta M. and L. Gilchrist S.  
March, 1994



**CIMMYT**

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**Wheat Special Report No. 21b**

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## **Preface**

E.E. Saari  
Leader, Crop Protection

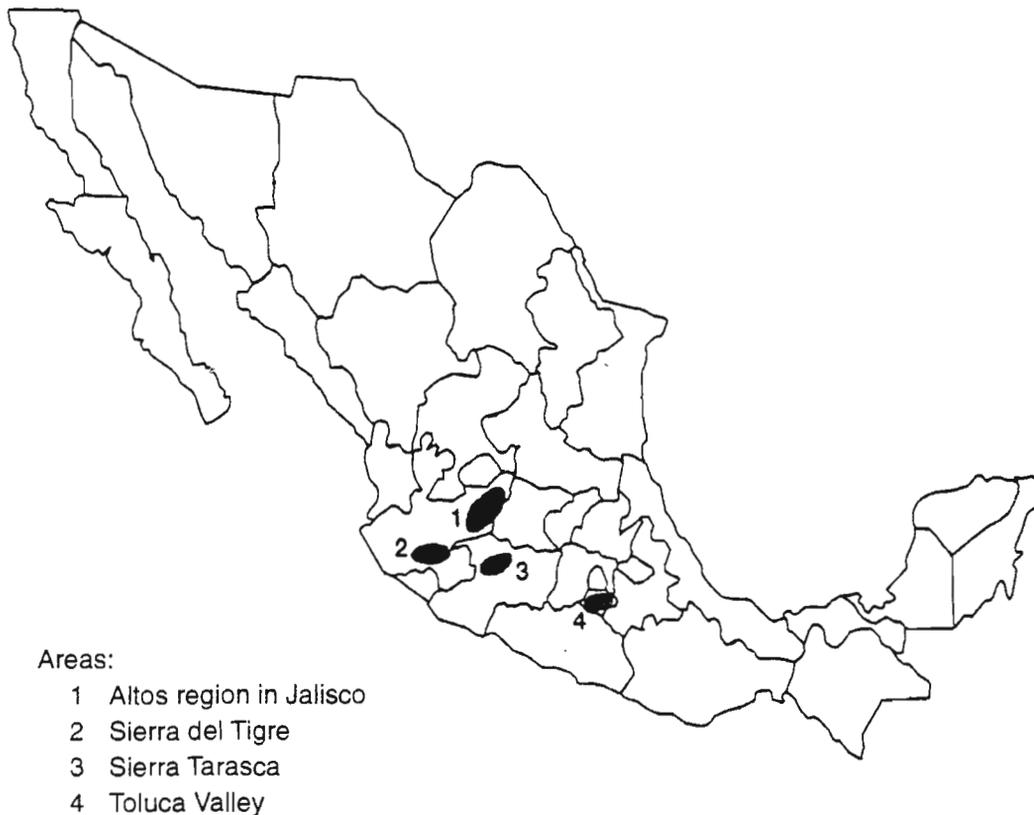
The scab disease of wheat has been an important factor in many areas of the world, especially where humid or moist conditions prevail from heading time to maturity. Not only can scab cause severe crop losses, but the role of mycotoxins it produces is now being recognized as a major factor in both animal and human health. Control of scab has not been an easy task, but a combination of genetic resistance and cultural practices may provide an adequate strategy. Better sources of resistance are thus being sought.

The shift in the area sown to wheat in Mexico toward the high valleys and the impact of scab on wheat production in that region should place the disease among high research priorities. This brief synopsis not only includes what is known about the disease in Mexico, but also contains essential information from all over the world. We hope the information presented here will help guide researchers facing the problem and pave the way for wheat scientists to refocus their efforts in trying to control this serious disease.

## Introduction

Scab or head blight of wheat, caused by the fungus *Fusarium graminearum* Schwabe, was first described in 1891 by Arthur in the United States (2); it was later reported in Japan in 1902. In Mexico the disease was detected in 1977 in the Altos region in the State of Jalisco, although the specific pathogen species was not identified (19). At present, scab of wheat, also known as head blight, is prevalent in warm, humid regions throughout the world, e.g., north central Europe, Asia, and, particularly, China and Japan, where the disease is reported to be endemic. It has also been detected in the coastal region of North Africa, northern USA and southern Canada, as well as in the Southern Cone of South America, specifically Brazil,\* Argentina,\* Paraguay, and Uruguay (2, 12, 15, 25, 41, 50, 59).

Up to now in Mexico scab has generally been restricted to areas where wheat is grown under rainfed conditions, such as the States of Jalisco, Michoacan, Mexico, Tlaxcala, and Hidalgo. Total affected area is about 170,000 ha (Figure 1) (21). However, the disease is not found exclusively in rainfed areas and has been reported on irrigated summer-sown wheat in the Bajio region in Guanajuato, although this type of cropping is very sporadic.



**Figure 1. Distribution of head scab (*F. graminearum*) incidence in Mexico.**

\* Sexual cycle reported.

## **Economic Importance**

Wheat scab causes severe production losses worldwide. In China and Japan losses as high as 50% have been reported. During 1978 production losses in Argentina were estimated at 30% in the Santa Fe and Córdoba Provinces, with a 10% loss in the western part of that region. Later, in 1985, losses in the west-central, east-central, and south-central regions of that country were 9.3, 13.3, and 5.4%, respectively (15). Severe epidemics occurred in 1950, 1967, 1977, 1978, and 1985; 30% losses were reported in 1978 and 10% in 1985. Durum wheat losses reached a high of 70% in 1985; since then the area sown to durums has virtually disappeared (15).

Similarly, in Paraguay weather conditions in 1972 and 1975 favored fusarium and septoria epidemics, which together accounted for losses of up to 70% (59).

In Japan losses as high as 45% were reported on severely infected crops. Scab is one of the most important yield constraints for wheat in the People's Republic of China and has been known for 35 years. Approximately 7 million ha in the Yangtze Valley are affected each year. Besides reducing grain quality, the disease causes an estimated 5-50% yield loss each year. Losses reach 50% in one out of every five years. In 1951-1985, the mean frequency of scab epidemics on wheat was 54.2%, disease incidence was 50-100%, and production decreased by 5-15% (10).

Poland, the Netherlands, the United Kingdom, Czechoslovakia, Russia, and Austria are some of the countries reporting scab incidence. During 1979-85, in the Netherlands scab prevalence in farmers' fields was about 66.6%, with 1.9% severity on infected spikelets. The disease has been known to reduce yields by as much as 50% in that country (45, 46).

In Mexico rainfed wheat-producing regions with very high disease incidence are Sierra Tarasca in Michoacan, the Altos region, Sierra del Tigre and Tapalpa in Jalisco, as well as the Toluca Valley and Jicotepec in the State of Mexico (Figure 1).

Some years there are relatively important incidence levels in the Valles Altos in Tlaxcala and Hidalgo. Disease incidence has increased in the past few years, due perhaps to the increase in the area cropped to cereals and close rotations with maize, wheat, and barley. Incidence of head scab as high as 60% has been observed in plant populations and levels of 10 to 15% on individual plants or spikes. These incidences have caused moderate yield damage; however, when the disease attacks susceptible varieties, it can reduce commercial production by as much as 17% (21).

## **Scab-producing Organisms**

Head scab of wheat is caused principally by *F. graminearum* (Schwabe), although the Netherlands and other areas of Central Europe report *F. culmorum* as the most prevalent species (45). In Poland *F. culmorum*, *F. graminearum*, and *F. nivale* have shown similar virulence levels (from severe to strong), while *F. avenaceum* has proved to be mildly to moderately virulent. However, in several studies aimed

at identifying causal organisms, as many as 18 *Fusarium* species were isolated and identified (Table 1) (60). Of the total samples, 98% proved to be *F. graminearum*; 8.2%, *F. poae*; 2.4%, *F. acuminatum*; 1.8%, *F. moniliforme* var. *subglutinans*; 1.6%, *F. equiseti*; and 0.1%, *F. culmorum*, *F. avenaceum*, and *F. nivale* (34, 39).

In Mexico surveys of *F. graminearum* infection on durum, bread wheat, and triticale found percentage frequencies of 59 and 40% in Toluca and 74 and 64% in Patzcuaro in 1988 and 1989, respectively. Other species causing wheat scab in Mexico are presented by location (Toluca and Patzcuaro) and crop (bread wheat, durum wheat, and triticale) in Tables 2 and 3.

**Table 1. Species isolated and identified as causing scab of wheat (Wang 1988).**

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<i>Fusarium graminearum</i> Schwabe
<i>F. culmorum</i> (W.G. Smith) Sacc.
<i>F. camptoceras</i> W&R.
<i>F. moniliforme</i> Sheld
<i>F. subglutinans</i> (W&R) Nelson, Tousson & Marasas
<i>F. longipes</i> W & R
<i>F. equiseti</i> (Corda) Sacc.
<i>F. compactum</i> Gordon
<i>F. sambucinum</i> Fuckel (W & R)
<i>F. graminum</i> Corda (W & R)
<i>F. avenaceum</i> (Fr.) Sacc.
<i>F. tricinctum</i> (Corda) Sacc.
<i>F. acuminatum</i> Ell. et Ev.
<i>F. nivale</i> (Fr.) Ces.
<i>F. sporotrichoides</i> Sherb.
<i>F. chlamyosporum</i> (W & R)
<i>F. semitectum</i> Berk & Rav.
<i>F. oxysporum</i> Schlecht. emend. Snyd. & Hans.
<i>F. solani</i> (Mart.) Appel & Wollenw.

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**Table 2. Percent frequency (%) of *Fusarium* species on durum wheat at Toluca and Patzcuaro during a two-year survey.**

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Species	Toluca		Patzcuaro	
	1988	1989	1988	1989
<i>F. graminearum</i>	59.2	40.7	74.4	64.5
<i>F. avenaceum</i>	32.7	19.8	9.6	20.6
<i>F. culmorum</i>	3.6	-	9.6	-
<i>F. nivale</i>	3.6	27.2	-	8.4
<i>F. equiseti</i>	0.9	12.3	1.4	6.5

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Source: Gilchrist, L., CIMMYT.

**Table 3. Percent frequency (%) of *Fusarium* species on bread wheat, durum wheat, and triticale in Patzcuaro and Toluca in 1989.**

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Species	Toluca			Patzcuaro	
	BW	DW	TCL	BW	DW
<i>F. graminearum</i>	28.2	40.7	91.5	95.1	64.8
<i>F. equiseti</i>	4.2	12.3	0.9	2.5	6.5
<i>F. avenaceum</i>	47.9	19.8	5.7	1.2	20.4
<i>F. nivale</i>	19.7	27.2	1.9	1.2	8.3

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Source: Gilchrist, L., CIMMYT.

BW = Bread wheat; DW = Durum wheat; TCL= Triticale

## Etiology

The main causal organism of *Fusarium* head scab is *F. graminearum* Schw. (syn *F. roseum* Lk. emend. Syd. & Hans. cv. *graminearum*, perfect stage *Gibberella zeae* (Schw.) Petch, syn *G. roseum* f.sp. *cerealis* cv. *graminearum*; *G. saubineti* (Mont.) Sacc.

Under lab conditions on PDA medium, *F. graminearum* shows color variations from pink to brown to dark red and purple. It produces abundant aerial micelia which turn white or red upon contact with PDA (Figure 2).

Optimum growing temperatures on culture media are 24-26°C. *Fusarium graminearum* produces short lateral phialides measuring 3.5-4.5µ x 10-14µ and canoe-shaped macroconidia measuring 2.5-5µ x 35-62µ with 3-7 septa. A distinctive characteristic of macroconidia of this fungus is the foot-shaped basal cells (Figure 3). In culture media macroconidia are produced on simple phialides which in turn may develop on branched conidiophores or group together to form a sporodochium. This species does not produce microconidia. Chlamydospores, if they are formed, are found midway or at the end of the micelium or in the macroconidia. Chlamydospores are balloon-shaped and can measure 10-12µ in diameter.

*Fusarium graminearum* is one of the few species that produce perithecia under field conditions. Perithecia, the sexual stage of the fungus (*Gibberella zeae*), develop on wheat glumes, protruding from glume tissue. Perithecia play an important role in the pathogen's survival from year to year (24), for together with micelia remaining on residues of the previous crop, they constitute the initial inoculum source for scab.

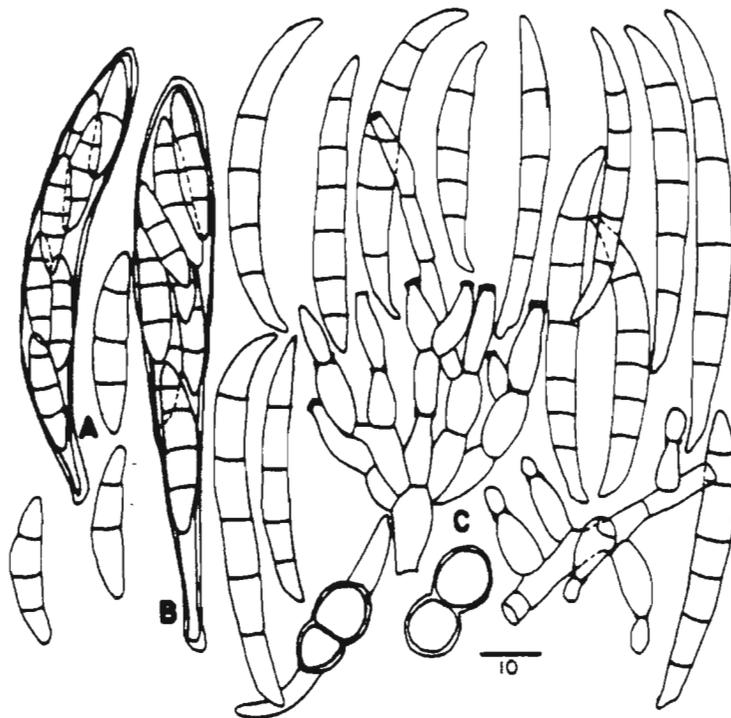


Figure 3.  
*Fusarium graminearum* (*Gibberella zeae*).  
A-B: Asci and ascospores; C: macroconidia and conidiospores (reproduced from Booth, 1971).

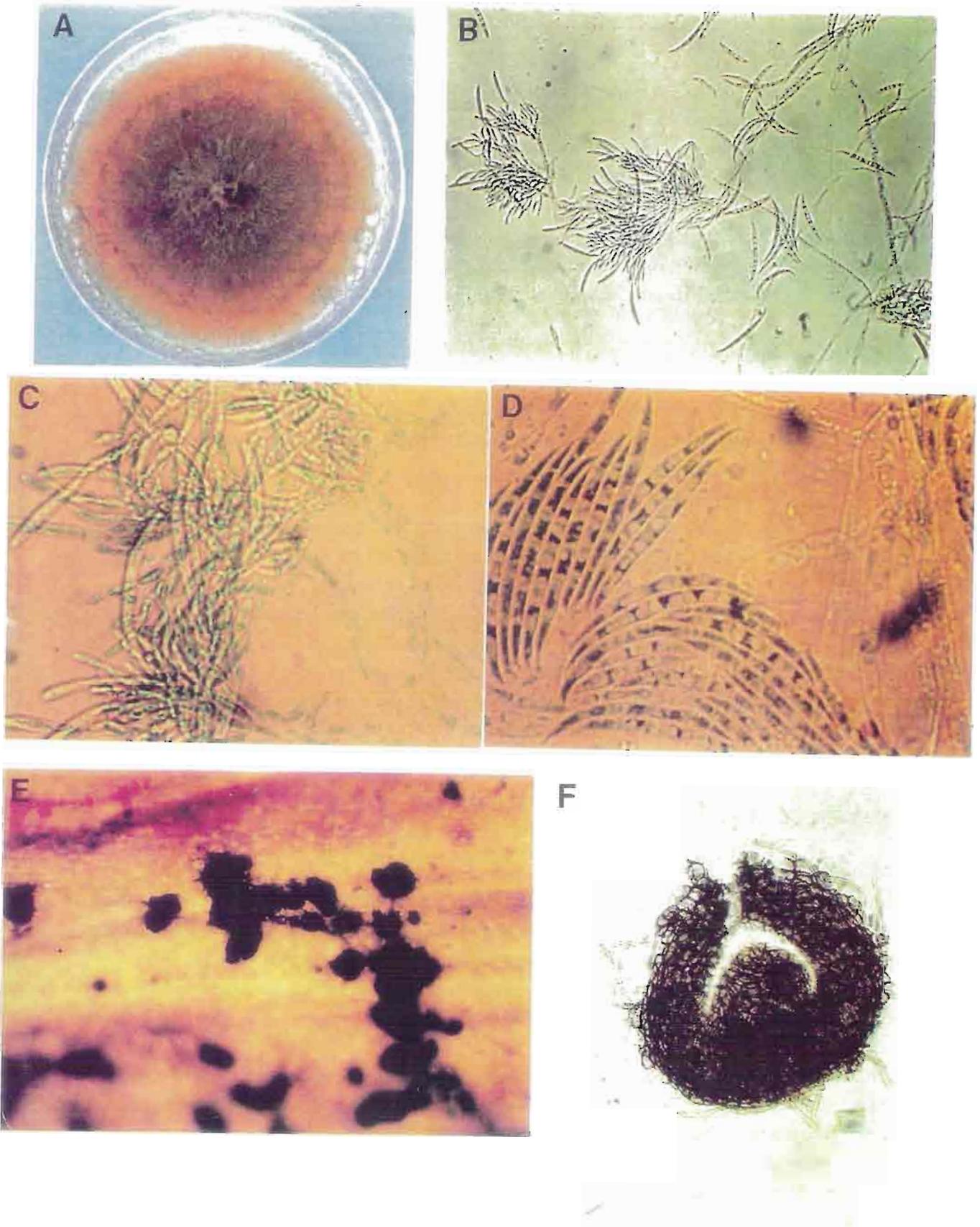


Figure 2. *Fusarium graminearum* (*Gibberella zeae*). A: micelium development on PDA; B, C, D: macroconidia and conidiospores; E: perithecia on glumes; F: perithecia and ascospores.

Perithecia are purple to black in color and develop on an inconspicuous stroma; they are oval shaped, papillate, and 150-350 $\mu$  in diameter. Each perithecium contains eight club-shaped asci which can measure 8-11 $\mu$  x 60-85 $\mu$ . Each ascus in turn contains eight ascospores measuring 3-5 $\mu$  x 17-25 $\mu$  and having 1-4 septa. Ascospores are subhyaline to light yellow-brown with rounded ends (7, 49).

*Fusarium graminearum* has developed a pathogenic specialization in relation to the point of infection on the wheat plant; this has given rise to the formation of two pathogenic populations called the Heterothalic Group or Group I and the Homothalic Group or Group II (26).

The basic difference between these two groups is that Group I doesn't develop perithecia on culture media and is pathogenic on roots and crown. It is associated with root rots in wheat (52) and is considered to be a genetically heterothalic fungus. Group II, on the other hand, does form perithecia on culture media, is associated with scab of above-ground photosynthetic organs both on wheat and on maize, and is considered homothalic (26, 39, 40).

Based on studies of isolates obtained from samples collected in the States of Jalisco, Michoacan, and Mexico, *F. graminearum* Group II has been identified as the prevalent pathogen in those regions; furthermore, damage symptoms have always been observed on spikes and, occasionally, on roots and crown. In some areas of the State of Mexico (Jicotepec, Ozumba, and Temamatla), it has been found on roots and crown in association with other fungi such as *F. equiseti*, *Gaeumanomyces graminis*, *Phytium torulosum*, *Rhizoctonia solani*, *Bipolaris sorokiana*, and *B. speciferum* (58). In addition, in places like Sierra del Tigre and the Altos region of Jalisco, perithecia have been observed on wheat glumes in the field (36).

## **Epidemiology**

### **Survival of the inoculum source**

Crop residues play an important role in the preservation of *F. graminearum*, since it survives in the form of micelia or immature perithecia on infected wheat spikelets, maize ear residues, or on maize and wheat stubble. Crop residues on the soil can be very important to pathogen survival, as evidenced by the fact that *F. graminearum* infections on wheat sown in a field of maize residues may be two or three times more severe (54).

Another important inoculum source is infected wheat grains remaining on the soil surface after harvest. Because of their low weight, the grains fall to the ground along with the straw and remain there until the next cycle (Figure 4). Cultural practices also play an important role in the survival of the inoculum source, e.g., if the residues are plowed under, perithecia survival decreases and reduces the primary inoculum source (Figure 5) (40).

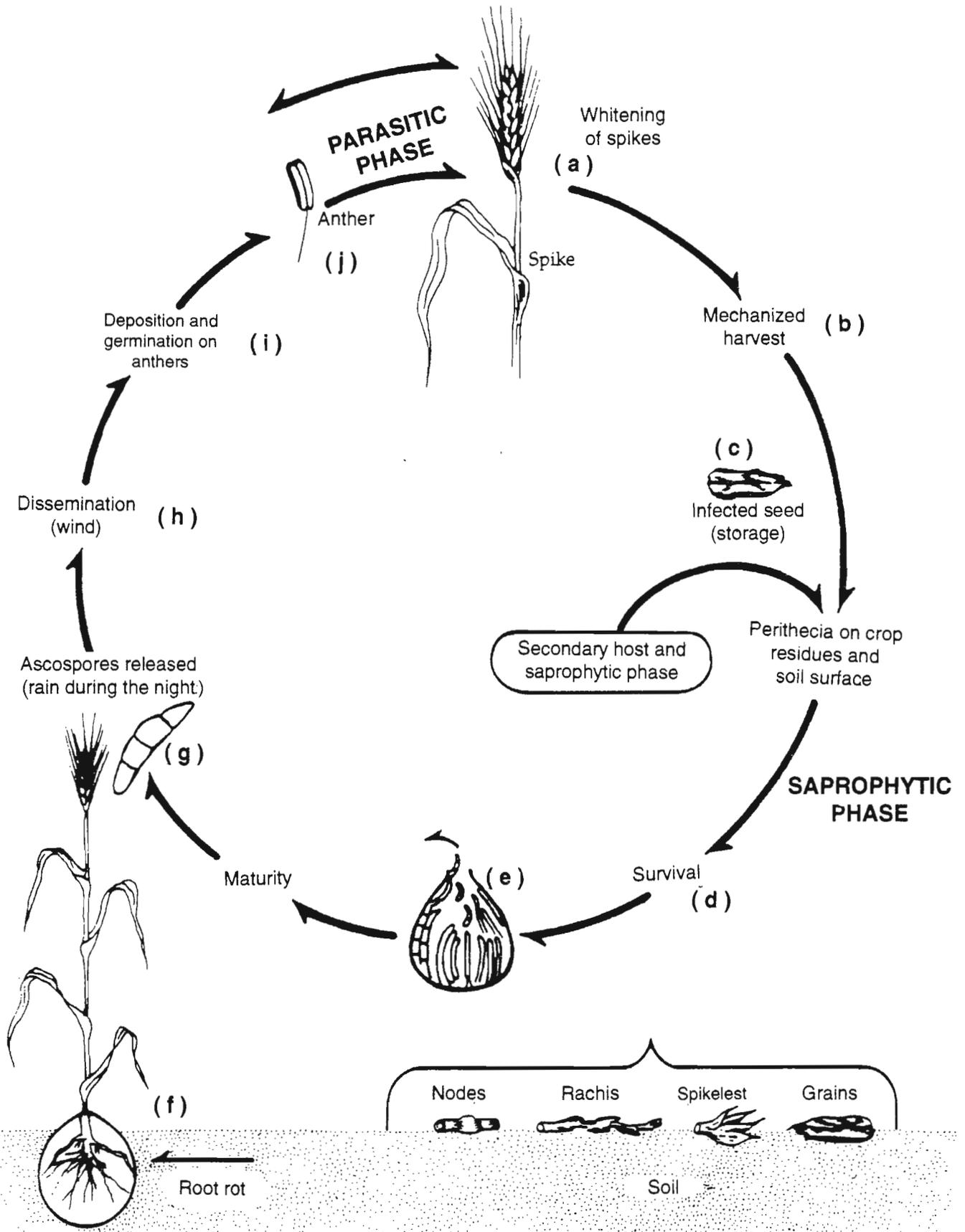


Figure 4. Life cycle of *Gibberella zeae* (*F. graminearum*) on winter cereals (Reis 1985) (adapted by J. Ireta).

## Types of inoculum

Scab infection process may be started by different types of inoculum: 1) macroconidia produced on sporodochia or individually; 2) ascospores produced inside perithecia (*G. zeae*); 3) chlamydospores surviving on soil surface or crop residues (this is less common); and 4) micelia surviving on maize or wheat residues.

## Inoculum production and dispersal

Conditions favoring inoculum production are high relative humidity and warm temperatures. The required temperatures for macroconidial formation are 16-36°C, and 32°C is optimal.

Perithecia formation in the field takes place four to five weeks after initial infection, if environmental conditions remain favorable. Ascospores are produced at temperatures of 13-33°C, though 25-28°C is optimal (1, 40). Ultraviolet light is also important for perithecium and ascospore production, since wave lengths of about 390 nm are required (40). Under Sierra de Jalisco and Michoacan conditions, high relative humidity seems to be more important than high temperatures for scab symptom production.

The principal means of inoculum dispersal are rain and wind, especially for Group II pathogenic populations. Since the thresher eliminates most infected seed at harvest, *F. graminearum* is not a significant pathogen on seed; however, infected grains that fall to the ground and remain on the soil

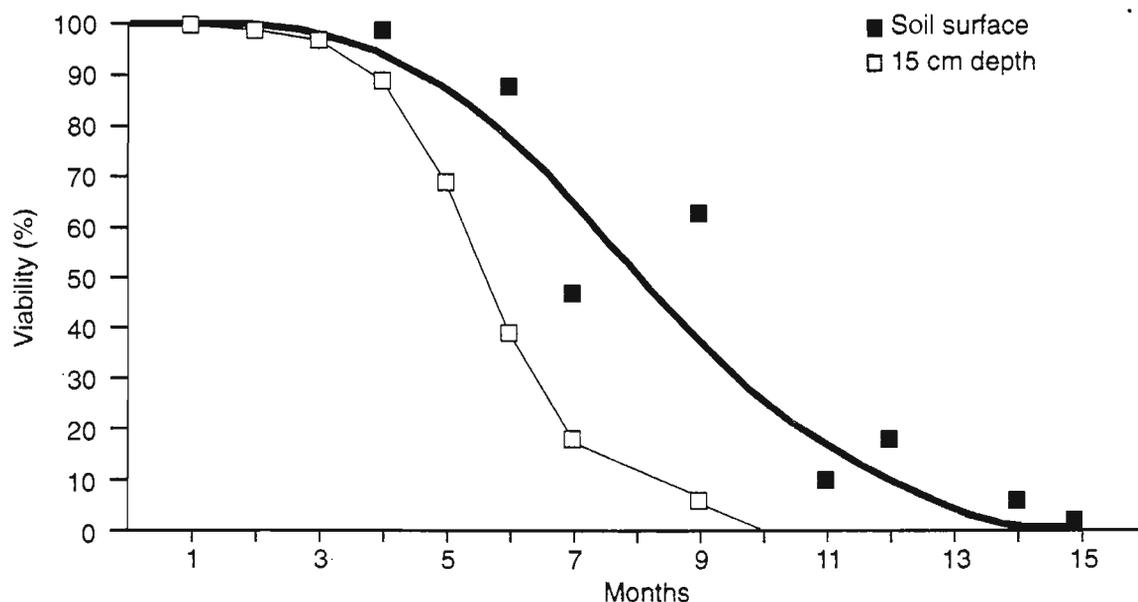


Figure 5. Survival of perithecia of *G. zeae* on naturally infected maize stalks maintained at two soil depths (Reis and Martinelli 1983).

surface are the inoculum source for the following year's crop. If infected grains are buried, they may damage the roots of the new crop, causing symptoms that are sometimes confused with those of *Helminthosporium sativum*.

## Hosts

The most common hosts of *Fusarium graminearum*, besides wheat, are barley, oats, rye, maize, alfalfa, and triticale. Some wild grasses such as *Brachiaria plantagia* (L.K.) Hitch., *Pennisetum purpureum* Chumach, *P. clandestinum* Chiov., *Digitaria sanguinalis* (L.) Scap., *Paspalum* spp., *Andropogon bicornis* (L.), and *Eryathus* sp. are either secondary hosts or saprophytic substrates (40).

## Spike Infection and Colonization

Primary *F. graminearum* infections have been observed on anthers extruded from the florets post anthesis, i.e., it is a floral infection (1). It has been suggested that *F. graminearum* requires anthers as a saprophytic base from which to penetrate the florets, since it is a facultative saprophyte and, under certain conditions, a parasite.

The presence of choline and betaine, two quaternary ammonium compounds produced by the anthers, seems to stimulate abundant growth of the pathogen on wheat florets. These substances occur naturally in the wheat plant, but under *F. graminearum* infection their concentrations increase and stimulate pathogen development. This process determines the pathogen's specificity to the wheat plant's floral organs (51).

Primary infections may arise from either ascospores or macroconidia deposited on glumes and extruded anthers. If the anthers have not yet emerged, spores can remain viable for several days until anthesis and later initiate penetration. However, preliminary evidence suggests that once they germinate, conidia can penetrate the glumes directly (21).

Temperatures of 10-30°C and relative humidity above 95% for 40-60 hours are usually enough for the spikes to be successfully infected by the macroconidia; in the Toluca Valley in the State of Mexico, however, symptoms appear after the first 72 hours post inoculation (4).

## Symptoms

The point of entry of *F. graminearum* is the wheat spike, especially the floral organs. This affects seed set and grain filling. Macroconidia or ascospores dispersed by the wind and rain are the principal inoculum.

Infected spikelets quickly lose chlorophyll and become pale in color. Later they turn pink or peach colored, especially at the base and edges of the glumes (Figure 6). If humidity continues to be high, diseased spikelets are invaded by saprophytic fungi and turn dark or black. For this reason, scab is sometimes mistakenly called “head smut.”

In the field, the first spikelets to become infected by the fungus are in the middle third of the spike because that’s where anthesis begins. If environmental conditions remain favorable, the infection advances toward the adjacent spikelets and, in some cases, may infect the entire spike, including the rachis and its peduncle.

When *F. graminearum* infection is severe, damaged grains are covered with micelia and take on the appearance of a pink cottony mass. If disease levels are moderate, the grain may be shriveled, low in weight, and whitish in color (Figure 7).

When the crop in general is at or close to maturity, perithecium formation (*G. zeae*) may occur. Masses of black perithecia that seem to be coming out of infected tissue develop on diseased spikelets. To date perithecia have only been observed on wheat grown in Sierra del Tigre and the Altos region in Jalisco of all the locations where the disease occurs.

## Toxin Production

Mycotoxins, which are toxic compounds produced by some fungi, belong to the trichothecene group (27, 48). *Fusarium* is one of the most prolific mycotoxin-producing genera, especially on such cereals as maize, wheat, rice, and sorghum. It is also one of the most dangerous because it produces toxic metabolites of diverse biogenic and toxic origins that may affect human and animal health, as shown in the following table.

**Table 4. *Fusarium graminearum* and *F. culmorum* metabolites obtained from strains of different origin.**

Metabolite	Canada/USA	Japan	China	England
Butenolide	+	+	+	+
Culmorin	+	+	+	+
Culmorone	+	+	+	+
Deoxysambucinol	+	+	+	+
Dihydroxyapotriconothecene	+	+	+	+
Sambucinol	+	+	+	+
7 Hydroxyisotrichodermin	+	+	+	+
Calonectrin	+	+	+	+
8 Hydroxycalonectrin	+	+	+	+
3 Acetyldeoxynivalenol	-	+	-	+
15 Acetyldeoxynivalenol	+	-	+	-
Dihydroxycalonectrin	+	+	+	+
Zearalenone	+	+	+	+
Fusarin C	+	-	-	-
Deoxynivalenol	+	+	+	+
Nivalenol	-	+	-	-

Source: Miller, 1989.



**Figure 6. Scab symptoms on wheat spikes; healthy and diseased grains.**



**Figure 7. White spikes; root rot causes early onset of maturity.**

Some of these compounds are mycotoxins, but the function of others is unknown. Under natural conditions, pathogenicity on maize ears and wheat spikes seems to be correlated to deoxynivalenol (DON) and 15 acetyldeoxynivalenol (15-ADON) production. Pathogenic or heterothallic isolates produce DON, 15-ADON, low concentrations of zerealenone, and are quick growing. In contrast, homothallic isolates aren't pathogenic on maize or wheat, don't produce DON or 15-ADON, maintain high levels of zerealenone, and are slow growing (60).

However, some non-DON and non-15-ADON producing strains may damage seedlings or cause root rots (32). Deoxynivalenol and 3-acetyldeoxynivalenol are thought to affect protein synthesis in the ribosomes and therefore may be phycotoxins as well as mycotoxins (62). Grain invasion by *Fusarium* destroys starch granules, protein storage, and cellular walls, thus reducing grain quality (48).

The T<sub>2</sub> toxin is approximately 10 times more toxic to mammals than deoxynivalenol; however, it's less toxic to wheat (60). Toxic effects of the metabolites vary, depending on the species. Swine seem to be the most susceptible, followed by cattle and, finally, poultry. Effects may range from decreased feed palatability, weight loss, malformations, gastrointestinal irritations, disorders of the nervous system, and, in extreme cases, death (Table 5) (56, 57).

At 0.7 ppm, deoxynivalenol is enough to cause digestive and reproductive disorders. There are reports from Russia, Argentina, Japan, and China on the effects of infected grain on human beings (3, 28, 45). In China epidemiological studies of esophageal cancer have revealed a link to exposure to high *F. moniliforme* concentrations; the pathogen has been shown to produce large quantities of such toxins as fusarin C, which is as mutagenic as the aflatoxins and fumonisines (17, 33). Chronic

**Table 5. Main toxic effects on swine and poultry of mycotoxins produced by *Fusarium* species.<sup>1</sup>**

Mycotoxins	Clinical signs
Zearalenone	Swollen red vulva, vaginal prolapse and sometimes rectal prolapse in swine; suckling piglets may show enlargement of vulvae; fertility problems
Vomitoxin (desoxynivalenol, DON)	Decreased feed intake and weight gain in pigs with DON at $\geq 2$ mg/kg feed; vomiting, and feed refusal at very high concentrations of DON ( $\geq 20$ mg/kg feed) <sup>2</sup>
Other trichothecenes: <ul style="list-style-type: none"> <li>• T-2 toxin</li> <li>• HT-2 toxin</li> <li>• diacetoxyscirpenol</li> </ul>	More toxic than DON; reduced feed intake; emesis; skin and gastrointestinal irritation; neurotoxicity; abnormal offspring; increased sensitivity to disease; hemorrhaging
Ochratoxin	Mainly affects proximal tubules of the kidneys in swine and poultry; kidneys are grossly enlarged and pale; fatty livers in poultry

<sup>1</sup> Source: Trenholm, 1989. <sup>2</sup> mg/kg = parts per million (ppm).

ingestion of small quantities of trichothecenes can cause significant secondary effects, e.g., suppression of the immune system or predisposition to infectious diseases (48). Care must be taken not to inhale *Fusarium* dust or spores and avoid direct skin contact with *Fusarium*-infected grain (48, 57).

One of the few countries regulating the handling of infected grain, Canada has established that DON levels in infected grain used in animal rations should not exceed 1.0 mgkg<sup>-1</sup>. Sweden allows a maximum of 0.5 mgkg<sup>-1</sup> in pig rations and 2.0 mgkg<sup>-1</sup> in cattle feed.

In humans the allowed maximum daily dose is 3.0 µgkg<sup>-1</sup> of DON per kilogram body weight in adults and 1.5 µgkg<sup>-1</sup> in children. Milling infected grain doesn't eliminate deoxynivalenol (DON), although cooking the flour may reduce toxins by as much as 40%. In the US, FDA (Food and Drug Administration) tolerance levels in wheat grain and flour are 2.0 and 1.0 mgkg<sup>-1</sup> respectively. In Europe, only Romania and Russia have specified DON tolerance levels, namely 0.005 mgkg<sup>-1</sup> for food products and 0.5 mgkg<sup>-1</sup> for wheat grain (48).

## Evaluating the Disease and Estimating Crop Losses

### Disease scoring

The way *F. graminearum* spreads within the crop poses serious problems for field evaluations of the disease because infection levels in the plant population and on the spikes are not uniform. In the individual plant, the pathogen penetrates one spikelet and may infect as many as two or three adjacent spikelets. If environmental and genotypic conditions remain favorable for two or three weeks, the disease may spread and cover the whole spike, including the rachis (not common in Mexico). Disease spread within the plant population isn't uniform and doesn't follow a specific pattern (48).

Due to these problems, several field and greenhouse evaluation scales have been devised and tested to better represent what happens under natural conditions. A 0-5 field scale has been adopted with the purpose of obtaining uniform data from both geneticists and pathologists (Table 6). It includes percent damaged spikes per unit area and percent damaged spikelets per spike. In other words, disease incidence and severity are evaluated at the same time (62).

**Table 6. Modified Japanese scale for evaluating scab (*F. graminearum*) of wheat in the field.**

Scale	Response	% infection
0	Immune	0
1	Resistant	1-5
2	Moderately resistant	5-25
3	Moderately susceptible	25-50
4	Susceptible	50-75
5	Very susceptible	> 75

Under controlled conditions (as in a greenhouse), disease scoring should be based on number of damaged grains per spike, for which the following scale is suggested:

**Table 7. Modified Japanese scale for evaluating number of grains infected by scab (*F. graminearum*) of wheat under greenhouse conditions.**

Scale	Response	% infection
0	Immune	0
1	Resistant	1-8
2	Moderately resistant	9-11
3	Moderately susceptible	12-20
4	Susceptible	21-50
5	Very susceptible	> 50

In other cases, the disease index is used as a parameter that expresses disease incidence and disease severity in a single value. This index is defined as percent infected spikelets on 50 randomly selected spikes (62).

**Table 8. Scale used for evaluating scab (*F. graminearum*) of wheat at CIMMYT.**

Scale	Diseased spikelets per spike	Resistance level
0	0	Immune
T	1	Highly resistant
1	1-2	Resistant
2	2-4	Moderately resistant
3	5-6	Moderately susceptible
4	> 7	Susceptible
5	Complete spike	Highly susceptible

Source: CIMMYT Report on Wheat Improvement, 1988.

### **Estimating crop losses**

Assessing or estimating production losses is essential for classifying the disease correctly among biotic stresses restricting wheat yields in rainfed areas. Losses are quantitatively estimated based on yield, test weight, and the presence or absence of toxic substances. The simplest way of estimating losses is to compare the yield of a healthy plant to that of a diseased plant; however, when percent infection is low, test weight and 1000-grain weight are better options (13).

When doing more precise calculations aimed at estimating production losses, it's a good idea to include the main yield components, such as the number and weight of grains per spike and the number of diseased spikes per m<sup>2</sup>.

Quantitative assessment of disease development is important for estimating losses; it is thus desirable to score genotypes based on disease development. This may be done when cumulatively evaluating disease levels taken at several readings. The recommendation is to do at least four readings per cycle at regular intervals, starting when infection begins. The most appropriate variable is number of diseased spikes per m<sup>2</sup>.

The ideal strategy would be to select the model that best describes the disease during each epidemic. Since this isn't always possible, a good alternative is van der Plank's formula, in which a general disease development model is used to estimate the development rate (*r*) of any disease:

$$r = \frac{1}{t_0 - t_1} \left[ \ln \left( \frac{Y_1}{1 - Y_1} \right) - \ln \left( \frac{Y_0}{1 - Y_0} \right) \right]$$

where:

*r* = disease development rate

*t*<sub>0</sub> = disease initiation

*t*<sub>1</sub> = disease termination

ln = natural logarithm

*Y*<sub>0</sub> = initial disease level

*Y*<sub>1</sub> = final disease level

The (*r*) parameter (disease development rate over genotype) is a good indicator of genetic resistance. In addition, development rate interacts with yield components to give the estimated quantitative losses caused by head scab.

There are several mathematical models that quantitatively describe scab development. In 1986 several models were screened using statistical parameters such as the coefficient of determination and the scattergram. Gompertz' model was the one that best described the disease (5, 6, 22).

In this trial, the Gompertz model had the best value of R<sup>2</sup> as well as the smallest value of *r* (Cuadro 9). It should be pointed out that the Bertalanffy-Richards model had the best values for both these parameters, but was not selected due the high degree of difficulty in implementing it.

**Table 9. Comparison of disease development models used for describing scab (*F. graminearum*) of wheat during an epidemic in the Toluca Valley, State of Mexico, 1986.**

Model	R <sup>2</sup>	∑ (Y-Y <sub>i</sub> ) <sup>2</sup>	r
Logistic	0.725	0.0005	0.535
Exponential	0.490	0.0260	0.524
Inverse	0.719	0.0008	-56.745
Gompertz	0.794	0.0004	0.130
Bertalanffy-Richards	0.836	0.0005	0.067

Source: Ireta, 1986.

The general equation for the Gompertz model is as follows:

$$\frac{dY}{dt} = rY [\ln 1 - \ln Y] = rY [-\ln Y]$$

where:

$dY/dt$  = change in disease levels over time

$r$  = disease development rate

$Y$  = disease severity

$\ln$  = natural logarithm

## Control Measures

Measures for controlling scab have been only partially effective because disease incidence is strongly influenced by environmental factors, especially relative humidity and temperature. Another difficulty is the pathogen's adaptable behavior. Despite these problems, efforts are underway to reduce scab's negative impact on wheat through genetic, cultural, and chemical means.

### Disease resistance

Wheat genotypes with good resistance levels have been identified. However, up to now it hasn't been possible to transfer this trait to agronomically desirable backgrounds. Varieties such as Toropi, Encruzilhada, K1, Atlas, and E. Young are late maturing and their resistance may be either partially genetic or the result of escape due to climatic conditions.

*Types of resistance.* Three types of genetic resistance to scab have been described:

- a) Type I or penetration resistance. The plant resists initial infection; the pathogen cannot penetrate host tissues.
- b) Type II or invasion resistance. The pathogen manages to penetrate the host plant, but its hyphae cannot invade cells adjacent to point of entry.
- c) Type III or biochemical resistance. Some genotypes are able to break down toxins (such as deoxynivalenol) produced by the fungus. The most resistant genotypes can tolerate high concentrations of toxic metabolites (46, 60).

*Screening genotypes.* Genotypic response to *F. graminearum* infection is screened under semicontrolled and field conditions (62). Identifying resistant materials is difficult because even small variations in the genotypes' vegetative cycles may cause differences in infection levels.

According to van Beuningen (7), there is negative correlation between the relative coefficient of infection (RCI) and scab with respect to days to heading and plant height; these combined parameters may cause nearly 50% of RCI variability. To avoid this problem, data should be taken at the critical growth stage.

**Heritability.** Heritability of *F. graminearum* resistance is thought to be polygenic (46, 47, 62). Chinese and Japanese researchers indicate that three major genes and some minor modifier genes are involved in resistance. The best sources of resistance, i.e., those with excellent resistance as well as good agronomic type, are of Chinese origin (25). Some reports indicate that scab resistance is controlled by a few genes; others maintain that 1) resistance is controlled by multiple (1-6) genes; 2) it is quantitatively inherited (low heritability and evidence of additive gene effects) (47); or 3) dominance predominates over recessiveness.

Since 1976 more than 10,000 wheat varieties and lines have been screened for scab resistance in China. Although 321 resistant varieties have been identified, only 40 of them have been incorporated into breeding programs, the most important of which are:

**Table 10. Wheat lines used as sources of scab resistance (*F. graminearum*).**

Name	Cross	Resistance	Origin
Su mai # 1	Funo/Taiwan mai	R	Jiangsu <sup>b</sup>
Su mai # 2	Funo/Taiwan mai	R	Jiangsu
Su mai # 3	Funo/Taiwan mai	R	Jiangsu
Wang shui bai		HR	Jiangsu
Zheng 7495	Fu sui-huang/You yi mai	MR	Jiangsu
Fan shan mai		MR	Fujian <sup>b</sup>
Xin zhong-chang		MR	Japan
Yan gang fang-zhu		R	Japan
Frontana		R <sup>a</sup>	Brazil

R = Resistant; HR = Highly resistant; MR = Moderately resistant.

<sup>a</sup> = Resistant to initial infection, not to pathogen spread within host tissue.

<sup>b</sup> = China.

CIMMYT conducts scab trials at three hot spots in Mexico: Toluca, in the State of Mexico, Patzcuaro, in Michoacan, and Sierra del Tigre in Atoyac, Jalisco. During 1985 CIMMYT's *F. graminearum* resistance breeding program identified 22 bread wheat varieties showing a mean resistance of 1 on the disease evaluation scale (Table 6). In 1986, 445 genotypes scoring between TR and 2 were identified at Toluca (11).

These successes indicate that progress is slow, but steady, especially in breeding programs working on developing *F. graminearum* resistance, such as the ones in China, Japan, Brazil, and Mexico (CIMMYT). Considerable advances have been made in Europe as well, although they're working on developing resistance to *F. culmorum*, the main scab-causing pathogen in that part of the world.

## Chemical control

The use of fungicides for controlling the disease is often impractical because it is difficult to protect a wheat field where heading isn't uniform and where flowering is staggered. Furthermore, varieties with adequate levels of resistance are not available in many countries and farmers have to resort to fungicide applications.

Chemical control of cereal diseases is not common practice in rainfed agriculture due to the high cost of chemical inputs. However, it is a good short-term control alternative, particularly when production levels must be maintained. Even when it's appropriate to use them, fungicides affording adequate levels of control are virtually unavailable on the market. For this reason, efforts aimed at developing better chemical control measures have been stepped up.

Satisfactory results have been obtained locally, as reported by Neto and Giordani (38), who obtained the lowest percent damaged grain with Thiabendazole (500 g a.i./ha) and Carbendazim (250 g a.i./ha). The combination of Propiconazole (500 g a.i./ha) + Prochloraz (450 g a.i./ha) was the most effective treatment in terms of grain yield and test weight.

In 1989 evaluation of fungicide effectiveness against scab and septoria diseases of wheat occurring together in Sierra del Tigre, Jalisco, indicated the best treatments were a mixture of San-619 (1.0 l/ha) + Chlorothalonil (1.0 kg/ha), followed by Propiconazole (0.5 l/ha), as shown in Figure 8. Effects of the treatments were observed on test weight and 1000-grain weight, but not on total plot yield (Table 11) (13, 18).

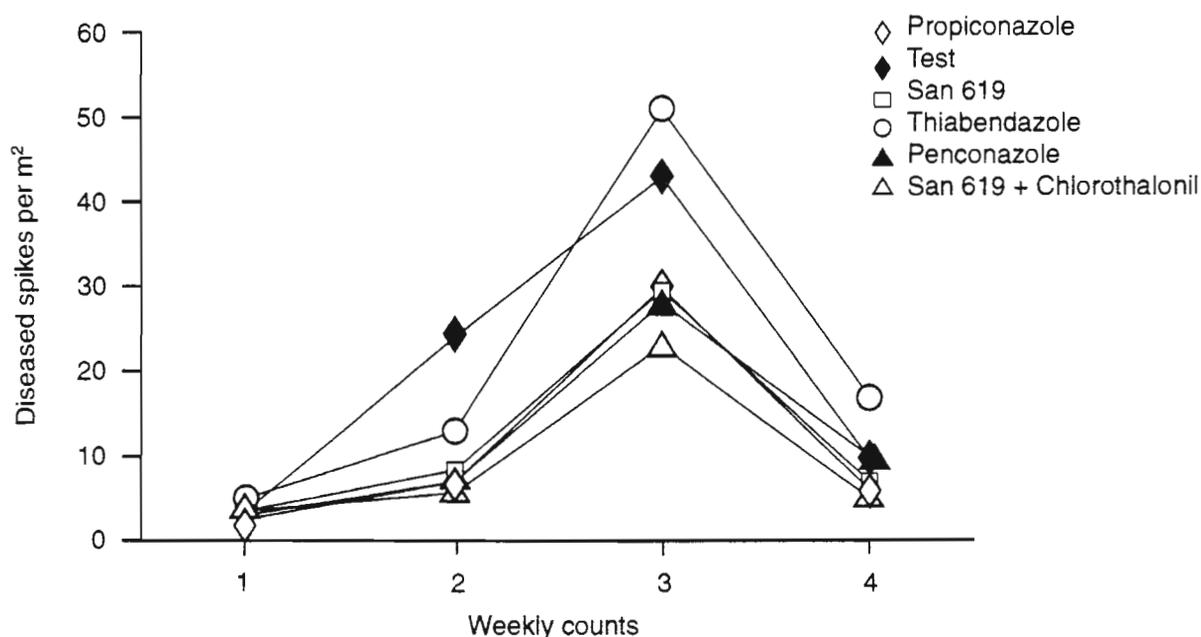


Figure 8. Effects of five fungicides on *F. graminearum* incidence, 1989 summer cycle.

## Cultural control

In Mexico wheat producers' socioeconomic circumstances make the adoption of quick, effective control measures, such as fungicides, very difficult. For this reason, the most economic alternative for maintaining a healthy crop is use of resistant varieties in combination with cultural control measures such as:

- a) *Removing crop residues.* Crop residues have been shown to act as a substrate for the survival of *F. graminearum* or *G. zeae* inoculum from one year to another, causing high disease incidence. Burying or removing crop residues decreases perithecium survival and eliminates or reduces the primary inoculum source for the following year.
- b) *Crop rotation.* It's important to break the pathogen's reproductive cycle by changing the host plants. In Canada a three- or four-fold increase in *F. graminearum* incidence has been observed on wheat sown after maize (54). This is because the pathogen attacks both crops.
- c) *Weeding.* Alternate hosts, such as weed species, are an important source of inoculum (40) and should be removed.
- d) *Use of certified seed.* Although seed transmission is not a significant disease spread factor, it's important to use certified seed to control the incidence of other pathogens and ensure seed vigor.

**Table 11. Response of wheat variety Gálvez M-87 to chemical control of the *Fusarium-Septoria* complex, Sierra del Tigre, Jalisco, 1989 summer cycle.**

Treatment	Yield (kg/ha)	Test weight (g/lit)	1000-grain weight (g)
San-619 +			
Chlorothalonil	1328.6	779.6	28.55
Penconazole	1208.3	728.3	24.53
Propiconazole	1204.6	765.6	26.96
San-619	1145.0	724.0	24.10
Thiabendazole	993.0	709.6	22.95
Test	990.0	715.0	22.86

Source: Ireta, J., Informe Anual de Investigación 1989. SARH, INIFAP-CEAJAL.

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