DISEASE: Common smut (Syn. boil smut, blister smut)

PATHOGEN: Ustilago maydis (Syn. Ustilago zeae)

HOSTS: Maize (Zea mays), teosinte (Zea mexicana)

Taxonomy of Ustilago maydis

Kingdom: Fungi – champignons, Fungo, fungi

Subkingdom: Dikarya

Division: Basidiomycota – basidio’s, basidiomycetes, club fungi
Common smut (Ustilago maydis) Of Maize

Subdivision: Ustilaginomycotina

Class: Ustilaginomycetes

Subclass: Ustilaginomycetidae

Order: Ustilaginales

Family: Ustilaginaceae

Genus: Ustilago (Pers.) Roussel

Species: Ustilago maydis (DC.) Corda – corn smut

Common smut of corn, caused by *Ustilago maydis*, is easily identified by tumor-like galls that form on actively growing host tissues and contain masses of dark, sooty teliospores. Throughout most of the world, common smut is considered to be a troublesome disease of corn, but in central Mexico, galls on ears of corn are considered an edible delicacy known as cuitlacoche (Syn. huitlacoche). In addition to the practical significance of causing a prevalent disease and being an edible fungus, *U. maydis* also has been used as a model organism to study a variety of interesting biological phenomena.

**Symptoms**

*Ustilago maydis* infects two hosts: maize (*Zea mays*) and teosinte (*Zea mexicana*). Like other smut diseases, common smut of corn derives its name from the sooty masses of teliospores found on infected host plants (Figure 2). The most obvious symptoms are tumor-like galls that vary in size from less than 1 cm to more than 30 cm (0.4-12 inch) in diameter. All meristematic tissues are susceptible to infection. Galls are found most frequently on ears, tassels, stalks, nodal shoots, and mid-ribs of leaves (Figures 3-6). Even though galls may form on many above-ground parts of the plant, infection is local (i.e., the host is not colonized systemically). Occasionally, galls develop beneath the soil surface when the apical meristem of a young plant is infected.

![Figure 2](image1)
![Figure 3](image2)
![Figure 4](image3)

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Smut galls consist of fungal and host tissues. Young galls are white, firm and covered with a semiglossy periderm. As galls begin to mature, interior tissue becomes semifleshy and streaks of black tissues occur as teliospores begin to form. With further maturation, galls become a mass of powdery teliospores and the periderm ruptures releasing the spores (Figures 2 and 3). Galls usually are obvious within 10 to 14 days after infection. On ears, galls mature about three weeks after ovaries are infected. Slightly discolored or disfigured kernels are obvious 3 to 6 days after infection. Small, white, firm galls develop 9 to 10 days after infection, and galls begin to enlarge resulting in a tumor-like growth 11-12 days after infection. Galls start to have a gray, silvery appearance as streaks of blackened tissues (teliospores) begin to form 14 to 15 days after infection. About 70% of the gall tissue is blackened while galls retain a semi-fleshy, mushroom-like integrity 16 to 18 days after infection. By 21 to 23 days after infection, the periderm ruptures and galls become a sloppy, wet, mass of teliospores. With dehydration, galls become sooty, powdery masses of teliospores.

The number, size and location of galls depend on the age of plants at the time of infection. Leaf galls differ greatly in size but usually are small when compared to stalk and ear galls. Galls 20-30 cm (8-12 inches) in diameter are common on the stalk. Rudimentary ear shoots below the fertile ear are commonly infected. Galls can replace most of the tassel or individual florets depending on the time and severity of infection (Figure 5). Ear galls usually result from infection of individual ovaries. Commonly, a few kernels at the basal or tip ends of the ear are infected (Figure 11), although nearly every kernel on an ear may be replaced by smut galls if most ovaries are infected.

Symptoms of head smut of corn, caused by *Sphacelotheca reiliana*, are somewhat similar to common smut (Figure 12). Head smut occurs when maize plants are infected systemically by *S. reiliana*. Floral structures (tassels and ears) are converted to sori containing masses of powdery teliospores that resemble mature galls of common smut. Vascular bundles of the host usually are present as thread-like structures in sori of *S. reiliana*. 
Reproduction

Mating and infection are under genetic control of two different loci, $a$ and $b$. Historically, the $a$ locus has been called the mating type locus and the $b$ locus has been called the pathogenicity locus. In order for $U.~maydis$ to infect, mating partners (i.e., haploid sporidia that form diakaryotic hyphae) must have different alleles at both the $a$ and $b$ loci (e.g., a sporidium with $a1b2$ alleles mates with one having $a2b1$ alleles). Alleles from both loci have been cloned and sequenced. The $a$ locus has two alleles, $a1$ and $a2$, and encodes components of a pheromone response pathway which mediates formation of conjugation tubes and cell fusion. When nutrients are limiting, haploid sporidia that have unlike alleles at the $a$ locus form slender filaments that grow towards each other following pheromone gradients. Ultimately, the filaments fuse. Multiple alleles occur at the $b$ locus which encodes DNA-binding proteins with regulatory functions. If the dikaryon that results from filament fusion has two nuclei with unlike alleles at the $b$ locus, a vigorous infection filament forms (Figure 17). The infection filament responds to an appropriate surface of the host by forming an appressorium that allows the fungus to penetrate the host (Figure 18). Appressoria form only on living cells that are rapidly elongating. Appressoria have not been induced in vitro. Mating and penetration occur within a 12-18 hr period on plants artificially inoculated with compatible sporidia.

In the first several days of infection, infected tissues may appear slightly chlorotic, but there are few macroscopic signs of infection. Microscopic examination reveals that in these early stages, the $U.~maydis$ dikaryon behaves as a biotroph. The hyphae grow mainly intracellularly, but both host and fungal plasma membranes remain intact. The plant cells undergo normal cytoplasmic streaming even though they are invaded by the hyphae. Regular haustoria are not observed, but the presence of an electron-dense matrix material in between the host and fungal walls is a likely site of nutrient transfer. The exact nature of
the nutrients transferred is not known, but most *U. maydis* strains that are amino acid auxotrophs are non-pathogenic, suggesting that a simple carbohydrate may be the primary nutrient used by the fungus.

Microscopic alterations such as abnormal enlargement of parenchyma cells may be seen as early as 24 hrs after inoculation. Tumors resulting from abnormal growth of host cells begin to develop as early as a week after infection. Tumors initially result from abnormal cell division and enlargement in host tissues, and fungal hyphae are relatively rare. Host cells remain alive during early stages of tumor formation, continuing to grow and divide, but the normal development of tissues is disrupted. For example, the regular pattern of venation is lost, and parenchyma cells may become extremely large. As the tumors enlarge, hyphae begin to proliferate in between the host cells rather than growing through them (Figure 21). Karyogamy (i.e., fusion of nuclei that occurs during sexual reproduction) appears to occur in early stages of tumor enlargement, and is followed by rapid hyphal proliferation in between host cells. At this stage, the hyphal walls become swollen and gelatinous, and the protoplasts lose their cylindrical shape and become spherical. By processes that are poorly understood, the spore initials enlarge and develop the pigmented, ornamented teliospore walls, which may be seen emerging from the remnants of the gelatinized hyphae (Figure 22). The fungus completes its life cycle each time teliospores form on an infected host.

**Disease Cycle and Epidemiology**

**Epidemiology**

Near the end of the 19th century, Brefeld worked out many of the details of the disease cycle of common smut which coincides with the life cycle of *U. maydis*. The fungus overwinters as diploid teliospores in crop debris or soil. Christensen estimated that a single smut gall of medium size may contain more than 200 billion spores (Figure 23). Teliospores can remain viable for several years. Teliospores can be disseminated directly by wind or splashing rain, or they can
germinate and undergo meiosis to form haploid sporidia which also can be disseminated by wind or splashing rain. Sporidia bud in a yeast-like manner, mate and form dikaryotic infection hyphae. Infection hyphae of compatible mating types (i.e., different alleles at the $a$ and $b$ loci) penetrate and infect the host. All infection is local. Any above-ground plant part can be infected, particularly young actively growing meristematic tissues. The fungus also may grow down stigmas (ear silks) and into developing ovaries (kernels), resulting in ear galls. Each gall on an infected ear is the result of infection of an individual ovary. Disfigured tissue which develops into galls may be noticeable within days after infection. Galls form within a week after infection and enlarge for up to 3 weeks after infection. About 1 to 2 weeks after infection, hyphae in galls begin to gelatinize, and hyphal cells become round and form cell walls of teliospores. As galls mature and dehydrate, the periderm ruptures releasing teliospores.

Figure 23

Teliospores produced in galls of infected tissues are probably unimportant as inocula in the growing season in which they are produced. However, they become the overwintering inocula that initiate infection on subsequent crops. There appears to be sufficient inoculum in nature to result in as much common smut as host and environmental factors will allow. In some instances, common smut has occurred in fields that were newly broken and have never been planted with corn previously.

There is no general agreement on weather conditions that are most favorable for common smut although most reports indicate that common smut is prevalent following rainy, humid weather. There also are reports linking increased infection rates with wounding of plants, as may occur during detasseling. Galls on leaves and stalks of seedlings often are observed following strong thunderstorms with heavy winds, especially when plants are injured by blowing soil. Obviously, germination and dissemination of teliospores and/or sporidia would be favored by wind and rain. However, anything that induces rapid localized cell division and elongation, such as wounding, could increase susceptibility to smut infection. Factors that reduce the production of pollen or inhibit pollination also increase the occurrence of ear galls of common smut.
because ovaries are protected from infection by *U. maydis* soon after they are fertilized, mostly likely because silks attached to fertilized ovaries die and are thus no longer susceptible to infection. For example, hot, dry, drought-like conditions often cause asynchronous pollen production and silk emergence which results in poor pollination. Ear galls of common smut may be prevalent if *U. maydis* is readily disseminated to stigmas of unfertilized ovaries during or immediately following these hot, dry conditions. Thus, some people associate the occurrence of ear galls with droughts although the droughts probably affect the prevalence of ear galls primarily by increasing the number of unpollinated ovaries with rapidly growing silks.

**Common Smut of Corn Disease Management**

Management

Many methods of controlling common smut have been recommended or evaluated, including crop rotation, sanitation, seed treatments, application of foliar fungicides, modification of fertility, and biological controls. In spite of these frequently mentioned control tactics, host resistance is the only practical method of managing common smut in areas where *U. maydis* is prevalent. Nevertheless, no corn line is immune from infection by *U. maydis*.

Resistance

The first significant advance in the control of common smut occurred when the first generation of corn hybrids were developed from inbred lines that where selected for lower incidence of smut. Although resistance to common smut has been studied for nearly 100 years, specific mechanisms of resistance and genes that condition resistance have not been documented clearly.

Early corn breeders observed that varieties of flint corn tended to be more smut-susceptible than varieties of dent corn. In 1918, just a decade after George Shull and Edward East independently discovered heterosis (hybrid vigor) in corn, Donald Jones demonstrated that inbred lines of corn developed by him and East at the Connecticut Agriculture Experiment Station differed strikingly in the number of smut infected plants. During the next ten years, early corn
breeders such as H. K. Hayes, R. J. Garber and F. R. Immer reported that self-fertilization of open-pollinated maize varieties rapidly led to the isolation of inbred lines with noticeably different reactions to common smut. Not only did inbred lines differ in the incidence of infected plants, susceptible lines could be differentiated by the plant parts on which galls formed. For example, some inbreds were uniformly infected on lower nodes of the stalk while others had a high incidence of ear or tassel galls. The rapidity with which selfed lines became homogeneous for smut reactions led Immer and J.J. Christensen to speculate that “a relatively small number of genetic factors are involved in determining smut reactions,” and that “composite crosses [of smut-resistant lines] will undoubtedly yield smut-resistant lines in a large percentage of cases.” Fortunately, the latter of these two hypotheses proved to be correct.