# Virus Diseases of wheat and their management

**Swati Yadav**

***Abstract*: in this paper described the virus disease of wheat and their management, in india many different factor can cause this, the most visible virus diseases in india are barley yellow dwarf virus ,soil borne disease , wheat streak mosaic virus, wheat spindle streak mosaic virus. Wheat streak mosaic virus is a virus that infects wheat and is transmitted by the wheat curl mite. This virus is responsible for wheat streak mosaic, a widely distributed disease of wheat that can cause economically important yield losses. The current viral taxonomy, vector biology, disease cycle, and management options of Wheat streak mosaic virus are reviewed in this paper and also reviewed the BYDV, WSSMV and SBWMV viruses diseases and their viral taxonomy, vector biology, disease cycle, and management options.**

***Keywords*: virus diseases of wheat, soilborne wheat mosaic virus, wheat streak mosaic virus, wheat spindle streak mosaic virus and their management.**

## DISEASES OF WHEAT

A number of different factors can cause these symptoms, including nitrogen deficiency, winter injury, and virus diseases. The most common virus diseases in Indiana are barley yellow dwarf virus (BYDV), soilborne wheat mosaic virus (SBWMV), wheat streak mosaic virus (WSMV), and wheat spindle streak mosaic virus (WSSMV). These diseases are difficult to distinguish from each other in the field, and laboratory testing will be needed for an accurate diagnosis. Al- though not typically severe, in certain years wheat viruses can substantially affect yield.

Wheat Spindle Streak Mosaic & Soilborne Wheat Mosaic Wheat infected with soilborne mosaic viruses (WSSMV and SBWMV) may exhibit yellow-green streaks on leaves and plant stunting and/or leaf tip dieback. SBWMV can cause a rosette symptom in susceptible varieties, which results in excessive production of severely stunted tillers. Researchers also have observed a reddish coloring on lower leaves on wheat plants infected with WSSMV and SBWMV. Plants infected with either virus may produce fewer stems and heads and have fewer kernels.

SBWMV and WSSMV infect wheat plants in the fall. The soilborne, fungus-like organism Polymyxa graminis transmits both viruses to wheat roots. This organism does not damage wheat by itself, but it enters wheat roots and transmits the viruses to wheat plants. Symptoms of virus infection are not apparent until spring, and the severity of symptom expression depends on varietal differences in susceptibility and weather. Prolonged cool conditions in spring (60°F, 15.5°C or less) enhance symptom development of both diseases in infected fields. As temperatures increase in the spring, symptoms often disappear and plants appear to recover. If virus symptoms and distribution within a field are limited, yield may be reduced, but severe or widespread infections can cause stunting and yield loss. You may need to replant severely infected fields. Consult exten- sion specialists to determine if replanting is necessary.

Wheat Viruses Wheat curl mite is an obligate parasite that can colonize wheat, corn, and a wide variety of other perennial grass hosts at any point during the growing season. When a large number of mites infect a single plant, they can cause the leaves to curl inward, giving the plant a rolled or twisted appearance. The mites acquire WSMV by feeding on infected plants, and they can then transmit the virus to other grasses for about one week. The mites do not have wings so they rely primarily on wind to move from one plant to another. The mites look like tiny grains of white rice when viewed under a hand lens or micro- scope, but they are not visible without magnification. When mites colonize wheat plants early in the growing season (especially closely after planting) there will be greater yield loss and more pronounced symptoms. However, a fall infection will not show symptoms until the following spring.

Since corn can also host wheat curl mites, try to avoid planting wheat within two weeks of harvesting late- maturing corn in adjacent fields. This will help inter- rupt the mite's life cycle and reduce the likelihood of wheat becoming infected with WSMV. Barley Yellow Dwarf Barley yellow dwarf (BYD) is caused by a number of different strains of Barley yellow dwarf virus (BYDV) and cereal yellow dwarf virus (CYDV). These viruses can infect more than 150 different grass species includ- ing wheat, oats, barley, rice, and corn. Several aphid species transmit the viruses. Once an aphid contracts the virus by feeding on an infected plant, it can transmit the virus for two to three weeks.

Symptoms usually appear in wheat two to three weeks after initial infection, but symptoms from fall infections may not appear until the following spring. Typical BYD symptoms include stunted tillers and root systems accompanied by discolored foliage. Discolor- ation typically begins at the leaf tips and progresses downward, which leaves a striped appearance at the interface of healthy and infected tissue. In wheat, leaves can appear yellow, red, or purple. The specific discoloration varies widely from one grass species to the next (from severe to negligible). Infected wheat plants may also exhibit reduced tillering, poor flowering, and sometimes kernel sterility or failure of the kernel to fill at all. Many factors influence how severe the symptoms will be including genotype, envi- ronmental stress, plant age at the time of infection, and the overall physiological condition of the plant. The healthier a plant is, the less severe the symptoms will likely be. Cool (50-68°F, 10-20°C), wet weather is favorable for aphid migration. Symptom expression is favored by cool, bright days.

## Septoria and Stagonospora Leaf Blotches

Septoria and Stagonospora leaf spots are common foliar diseases of wheat. Typical symptoms include round, elongated lesions, which, as they progress, develop small, black reproductive structures in their centers. Septoria leaf spot lesions tend to have wavy, poorly defined edges. Stagonospora lesions have clearly defined edges, often surrounded by a yellow halo.
**Barley yellow dwarf symptoms on wheat.Mixed Septoria and Stagonospora leaf lesions on a wheat leaf.**

**How to distinguish Septoria and Stagonospora leaf spots from wheat viruses**: Septoria and Stagonospora leaf spots are distinguished by small, brown-black fungal structures at the centers of their lesions. Also, as these lesions colonize leaves, the older lesions will turn necrotic, unlike viral discolor- ation, which simply remains discolored.
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Nutrient Deficiency Several nutrient deficiencies in wheat cause leaves to turn pale green or yellow, including nitrogen deficiency and potassium deficiency (Figure 9). Nitrogen and potassium deficiencies first appear on older leaves and are usually most pronounced at the tips. Symptoms usually begin in one or several patches in a field (those with lowest levels of the nutrient). How to distinguish nutrient deficiencies from wheat viruses: Viruses cause foliar discoloration that is usually mottled or mosaic-like. Nutrient deficiencies cause leaves to turn a uniform pale green or yellow. This coloration may have a gradient from tip to node, but it will not appear splotchy as with virus infections.
How to distinguish winter injury from wheat viruses: Winter injury can result in tip dieback and stunted plants, but winter injury will not appear to spread throughout the field or express itself as mottling or streaking on leaves (unlike a virus). Reference in this publication to any specific commercial product, process, or service, or the use of any trade, firm, or corporation name is for general informational purposes only and does not constitute an endorsement, recommendation, or certification of any kind by Purdue Extension. Individu- als using such products assume responsibility for their use in accordance with current directions of the manufacturer.

**Phosphorus-deficient wheat**.
Winter Injury Winter injury (or winterkill) typically occurs in aggre- gated areas throughout a field, often where conditions were conducive to prolonged periods of cold tempera- tures. Fields that were exposed to freezing and thawing cycles, and areas where ice developed on the soil surface, are particularly susceptible to winterkill. Winterkill symptoms include stunted plants and re- duced stand counts (Figure 10). This is most common after winters with prolonged periods of freezing weather.

## Wheat streak mosaic virus

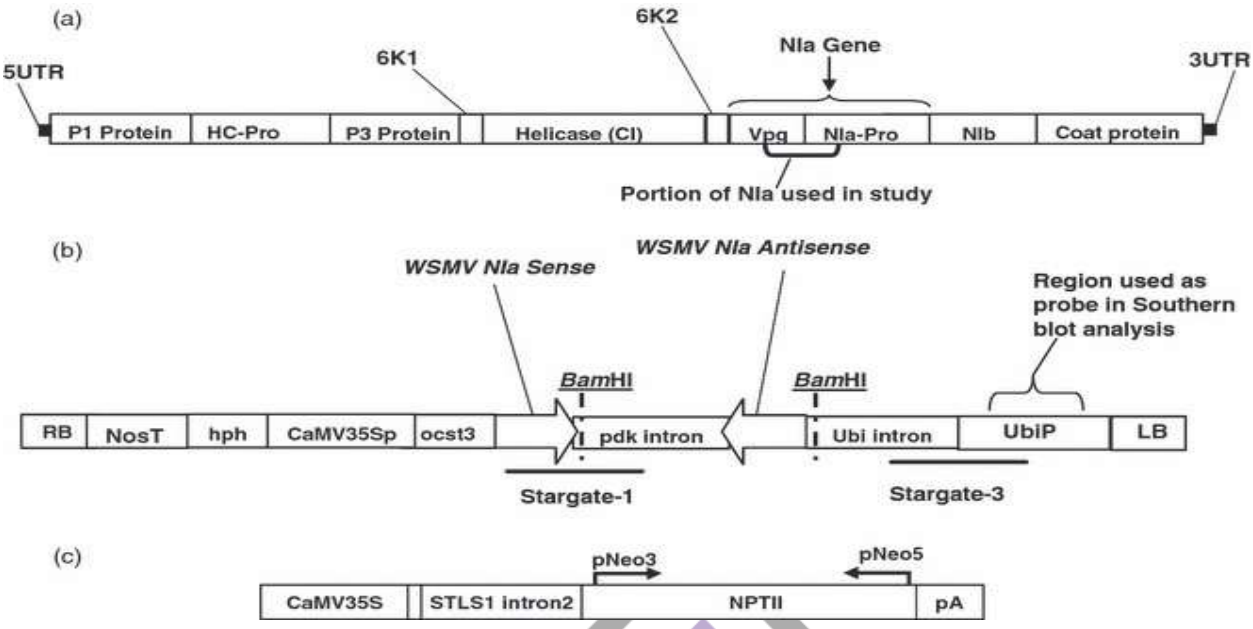
***Wheat streak mosaic virus*** (**WSMV**) is a plant pathogenic virus of the family *Potyviridae* that infects plants in the Poaceae family, especially wheat (*Triticum spp.*); it is globally distributed and vectored by the wheat curl mite, particularly in regions where wheat is widely grown. First described in Nebraska in 1922,[1] stunted growth and the eponymous "streaks" of yellowed, non-uniform discoloration are characteristic of WSMV infection. As it has been known to cause 100% crop mortality, WSMV is a subject of ongoing scientific research.

**Geographic Distribution and Yield Losses**

Wheat streak mosaic is a globally distributed disease of wheat. Initially described as "yellow mosaic" in Nebraska in 1922 (Hunger 2010), incidences of wheat streak mosaic have been reported in various states of the U.S., Canada, Mexico, Eastern Europe, Western Asia.
Reported yield losses in wheat because of Wheat streak mosaic virus infection can be substantial. In Kansas, reported yield losses ranged from 7 to 13% (Hansing et al. 1950, Christian and Willis 1993). An 18% yield loss was recorded during a Wheat streak mosaic virus epidemic in Alberta, Canada, in 1963 (Atkinson and Grant 1967). Wheat streak mosaic virus is reported to be a major limiting factor of wheat production in Texas panhandle (Velandia et al. 2010). Even though the average regional loss in yield potential seemed to be moderate, it is possible to find individual fields that suffer total loss because of Wheat streak mosaic virus infection.

**Taxonomy**

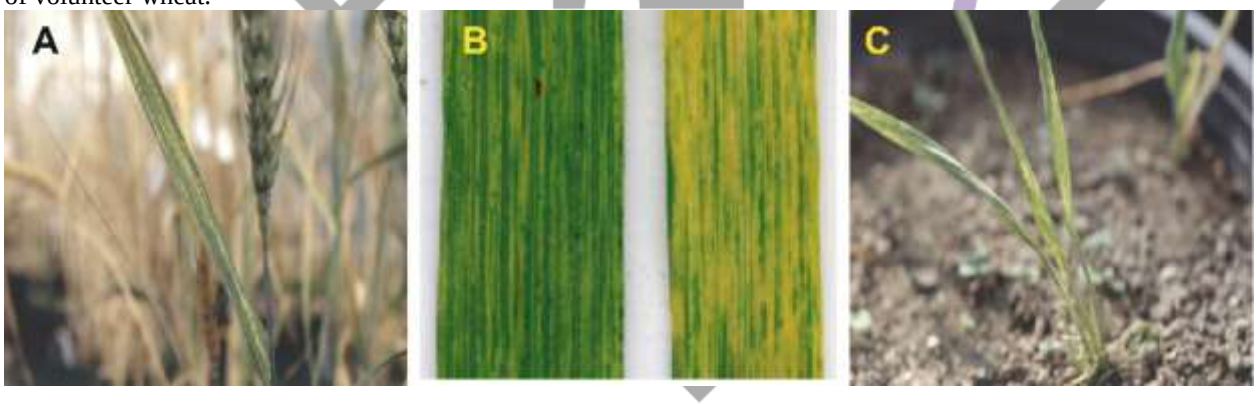
Wheat streak mosaic virus is a member of the family Potyviridae. The complete nucleotide sequence and the phylogenetic relationships of Wheat streak mosaic virus were reported by Stenger et al. (1998). Wheat streak mosaic virus was originally placed in the genus Rymovirus with other mite-transmitted viruses of Potyviridae. A phylogenetic analysis of Wheat streak mosaic virus using its completed nucleotide sequence demonstrated that it shares most recent common ancestry with the whitefly-transmitted Sweet potato mild mottle virus and not with Ryegrass mosaic virus, the type member of genus Rymovirus. This resulted in the proposal of a new genus within the family Potyviridae, genus Tritimovirus, of which Wheat streak mosaic virus is the type member (Stenger et al. 1998).




**Symptoms**

On individual leaves, the symptoms of wheat streak mosaic start as small chlorotic lines. As the symptom development progresses, chlorotic lines elongate to form discontinuous yellow to pale green streaks, forming a mosaic pattern in the leaves .In severe cases, the stripes may coalesce, forming large chlorotic areas and commonly result in the symptoms progressing into leaf tissue necrosis and plant death. Stunting is another prominent symptom of infected plants.

Infected plants often appear first on the field margins as the mites migrate from grassy areas and bordering crops (Hunger 2010). Wheat fields infected with Wheat streak mosaic virus exhibit yellowing and stunting in irregular areas, often at field margins or near weeds or volunteer hosts. As the season progresses, infection may spread and symptoms may appear within field. Wheat curl mites will colonize volunteer wheat growing after hail or heavy rain (Gibson 1957) and may serve as a source of wheat curl mite and Wheat streak mosaic virus for next wheat crops (Somsen and Sill 1970). In fields with heavy infestation of volunteer wheat, symptoms may not necessarily be found first on the field margins but instead be scattered throughout the field around the clumps of volunteer wheat.




**Damage**

Wheat streak mosaic virus infections reduce root biomass and water use efficiency, making it a serious concern in regions with limited availability of water (Price et al. 2010). Leaf chlorosis and necrosis because of infection by Wheat streak mosaic virus also reduce the photosynthetic capacity of the plant. Infection by Wheat streak mosaic virus may cause plant stunting (Langham et al. 2001b), reduced grain test weight (Atkinson and Grant 1967, Langham et al. 2001b), and reduced seed set (Atkinson and Grant 1967). Grains produced by Wheat streak mosaic virus-infected wheat have higher protein content but yield flour with lower water absorption compared with healthy wheat (Atkinson and Grant 1967). Yield loss because of infection by Wheat streak mosaic virus is correlated with the time of infection. Generally, infection by Wheat streak mosaic virus on early stages of the plant results in higher yield loss (Hunger et al. 1992).

Wheat streak mosaic virus has been reported most frequently on winter wheat yet it also occurs in spring wheat. Spring and winter wheat infected with Wheat streak mosaic virus exhibits similar symptoms including stunting, reduced yield and test weight, and increased protein content (Langham and Glover 2005).

**Transmission Mechanisms**

In the field, Wheat streak mosaic virus is transmitted by a mite vector, wheat curl mite, Aceria tosichella Keifer, the only known vector of Wheat streak mosaic virus to crops such as wheat (Slykhuis 1955) and corn (Sill and del Rosario 1959). The wheat curl mite is tiny (<0.3 mm or ≈1/100 inch) white, and cigar-shaped (Fig. 4) (Jeppson et al. 1975). It is invisible to unaided eyes. A heavy infestation of the wheat curl mite on a wheat plant induces curling of the leaf blades toward the midribs and distortion of the leaf lamina (Slykhuis 1955). Mites cannot fly, their dispersal is primarily wind assisted (Slykhuis 1955) but their long distance dissemination may be assisted by flying insects (Gibson and Painter 1957). As the host plants decline, the wheat curl mites crawl in large numbers to the tip of the plants. Sometimes piling on the top of each other and forming chains, ready for the next passing wind to carry them (Jeppson et al. 1975).

Figure 1. Wheat curl mites and symptoms of wheat streak mosaic.

Photo credit: Texas A&M Univ. Photo credit: Univ. of Kentucky

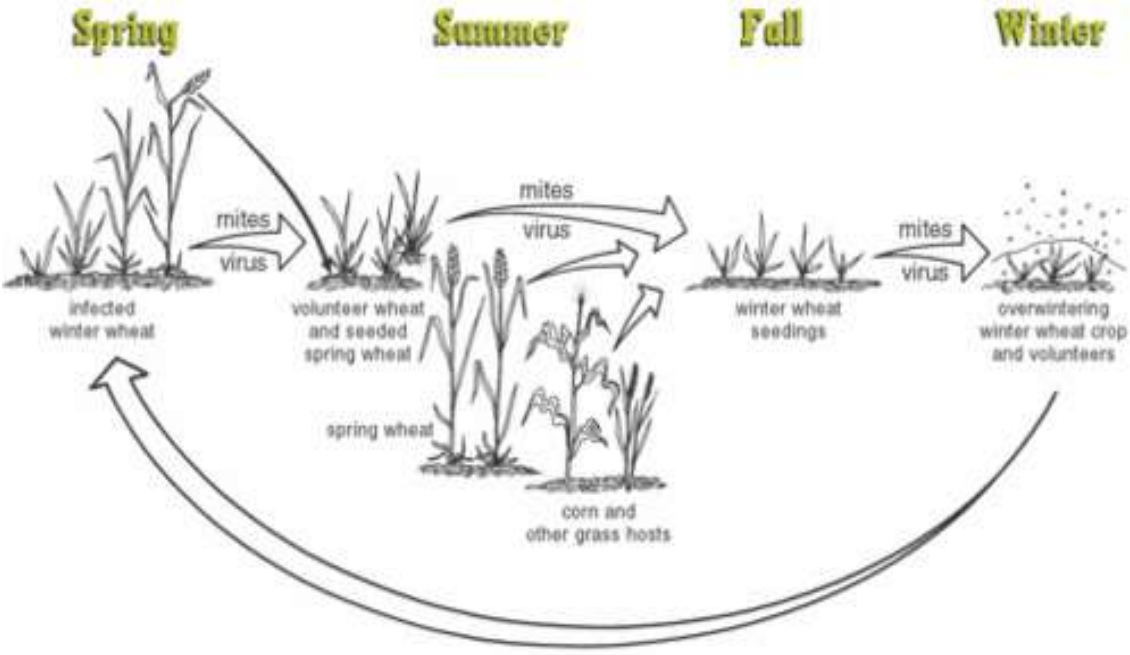
The life cycle of wheat curl mites consists of an egg, two instars of nymph and an adult. At 25°C (77°F), wheat curl mites complete their life cycle in an average of 7 days (Slykhuis 1955). The wheat curl mites live and feed on the hosts' green leaves, which are critical for mite survival. High humidity is another key factor in mite survival (Slykhuis 1955). Without food, wheat curl mites can survive for <8 hours at 24°C (75°F) and 30–40 hours at 3°C (36°F), indicating the mite's weak defense against desiccation (Jeppson et al. 1975). The mites overwinter as eggs, nymphs or adults living on the crown of winter wheat or other perennial grass hosts. Adult wheat curl mites can survive for several months at near-freezing temperatures but only 2–3 days at −15°C (5°F) (Slykhuis 1955). Eggs survived longer under sub-zero temperatures; over 25% of eggs exposed to −15°C (5°F) for 8 days were able to hatch when moved to room temperature (Slykhuis 1955).

Wheat streak mosaic virus is transmitted by wheat curl mite nymphs and adults but acquisition of the virus from diseased plant is restricted to nymphs (Slykhuis 1955). Virus acquisition and transmission occurs as the mite feeds. Wheat streak mosaic virus can be acquired from an infected host in as few as 15 minutes of feeding, however transmission efficiency is low (<2%) when acquisition time is short. Longer acquisition (feeding) periods result in higher transmission efficiency (Orlob 1966). At room temperature, Wheat streak mosaic virus is retained within the mite for up to 6 days after acquisition (Slykhuis 1955) but Orlob (1966) found that viruliferous mites kept under 3°C (37.4°F) remained infective for 2 months. Orlob (1966) suggested that slower mite development under near-freezing temperature may play a role in maintaining longer virus retention periods.

Recently, very low rates of virus transmission (0.5–1.5%) were reported to occur via seed produced by plants infected by an Australian isolate of Wheat streak mosaic virus (Jones et al. 2005).

**Disease Cycle**

Fall infections of Wheat streak mosaic virus on winter wheat occur when viruliferous mites move from Wheat streak mosaic virus-infected spring wheat, corn, volunteer wheat, and/or annual grasses onto winter wheat seedlings (Connin 1956, Christian and Willis 1993). Virus infection at this early stage is associated with the greatest yield losses on winter wheat (Slykhuis et al. 1957, Hunger et al. 1992). Infected wheat becomes a source of the virus for subsequent infection of adjacent plants. Winter wheat may become infected by Wheat streak mosaic virus in the spring, but typically spring infections result in insignificant yield losses (Somsen and Sill 1970).




In the spring, Wheat streak mosaic virus-infected winter wheat and perennial grasses serve as sources of virus and mites for spring wheat infection. Mite dispersal in the field is the highest as winter wheat matures, between May and June depending on the weather. Spring wheat planted near infected winter wheat fields during this time is at greater risk of Wheat streak mosaic virus infection (Langham and Glover 2005).

Wheat seeds shattered from spikes by hail may germinate as volunteer wheat. Gibson and Painter (1956) showed that volunteer wheat seedlings grown from mite-infested kernels were immediately colonized. Volunteer wheat may act as "green bridge" where mite and virus populations survive between wheat crops (Gibson and Painter 1957, Thomas and Hein 2003). Wet weather in summer that facilitates lush growth of volunteer wheat also supports high population of mites and Wheat streak mosaic virus for subsequent winter wheat infection (Connin 1956, Christian and Willis 1993, Thomas and Hein 2003).

**Management**

Volunteer wheat within wheat fields serves as a critical source of mites and early season Wheat streak mosaic virus infection (Thomas et al. 2004). Elimination of potential sources of mites and Wheat streak mosaic virus before planting also limits the risk of early Wheat streak mosaic virus infection (Slykhuis 1955). Destruction of volunteer wheat and wild grasses in a field two weeks before planting through conventional tillage or with herbicide applications has been shown to suppress the size of subsequent mite populations migrating into wheat (Thomas et al. 2004). In dry conditions, quicker results were obtained through tillage compared with glyphoshate herbicide in controlling the volunteer wheat and subsequent mite populations. Under wet weather, glyphosate herbicide and tillage were equally effective. As many nonselective herbicides may require several days or weeks to destroy all green leaf tissues, it is recommended that weed and volunteer wheat are destroyed at least three weeks before planting.In areas where spring and winter wheat season overlap, early planting of spring wheat reduces, but does not eliminate the risk of mite infestation, virus introduction and subsequent Wheat streak mosaic virus infection. Seeding winter wheat after the nearby winter or spring wheat crops mature and mite populations associated with these crops decline is recommended (Slykhuis et al. 1957, Hunger et al. 1992). Optimum window for sowing wheat differs depending on the region. Local Cooperative Extension Resources should be able to identify the locally relevant planting date range. Spring wheat should not be sown nearby infected winter wheat fields to avoid exposing the spring wheat seedlings to early Wheat streak mosaic virus infection. Spring wheat should also not be interseeded with winter wheat to limit movement of mites and viruses from maturing winter wheat to spring wheat seedlings (Langham and Glover 2005).

Wheat varieties with resistance to the wheat curl mites were shown to provide some protection against Wheat streak mosaic virus infection (Martin et al. 1984). However, biotypes of wheat curl mite that are able to overcome the resistant genes have developed in some regions (Hein 2010). Wheat with resistance against Wheat streak mosaic virus is another important tactic to manage the disease. An experimental wheat line 'KS91H184′ was shown to dramatically reduce the viral transmission efficiency of wheat curl mite (Harvey et al. 2005). Temperature-dependent resistance was reported on two experimental winter wheat lines, 'CO960293′ (Seifers et al. 2006) and 'KS03HW12′ (Seifers et al. 2007). Strains of Wheat streak mosaic virus failed to infect both wheat lines at 18°C (64.4°F) but the resistance broke down at 24°C (75.2°F). 'RonL' is a released hard white winter wheat variety with Wheat streak mosaic virus temperature-dependent resistance derived from experimental line CO960293. Because the resistance in RonL breaks down under high temperatures, planting is recommended in the months with cool temperature after the fall (Martin et al. 2007). Wsm1 is a gene conditioning resistance against Wheat streak mosaic virus identified from intermediate wheat grass (Thinopyrum intermedium) (Friebe et al. 1996). Wheat lines with Wsm1 gene showed resistance against Wheat streak mosaic virus in field conditions, as indicated by lack of symptoms and minimum yield loss (Seifers et al. 1995, Sharp et al. 2002). Mace is a released hard red winter wheat variety with Wsm1 gene. In prerelease field studies, winter wheat variety 'Mace' showed less

stunting and other Wheat streak mosaic virus-related symptoms compared with susceptible controls and comparable yield to Wheat streak mosaic virus-tolerant variety, 'Millennium' (Graybosch et al. 2009).

There is a variable degree of tolerance against Wheat streak mosaic virus among wheat varieties. Tolerant varieties infected by Wheat streak mosaic virus will generally suffer less yield loss than susceptible wheat varieties (Langham et al. 2001b). The hard red winter wheat variety 'Darell', for example, showed comparable severity of Wheat streak mosaic virus infection while sustaining much lower yield loss than wheat variety Millenium (Ibrahim et al. 2008).

Development of wheat varieties resistant to Wheat streak mosaic virus through genetic engineering has been attempted. Transgenic wheat lines have been engineered that include the virus' replicase gene (Sivamani et al. 2000) and the coat protein gene (Sivamani et al. 2002). Some of the transformed lines showed high level resistance in which fresh growth of inoculated plants showed no symptom development in laboratory studies (Sivamani et al. 2000, 2002). Yet, under field conditions, these transgenic lines did not show reduced Wheat streak mosaic virus severity and they generally produced lower yield than the parental line (Sharp et al. 2002).

Suppression of Wheat streak mosaic virus by chemical control of the wheat curl mite vector has proven difficult. Kantack and Knutson (1958) showed that spray application of several organophosphate and organochlorine pesticides, many of which are no longer registered in the U.S., did not provide long-term protection against wheat curl mite. In some cases, after an initial reduction in number, the mite population flared back to levels higher than untreated controls. Wheat curl mites feed in protected areas of the plant, such as within a curled leaf, leaf whorl or the recesses of the head. Because of this behavior, application of contact chemical control has not been successful (Hein 2010). Usage of systemic insecticides to control Wheat streak mosaic virus and wheat curl mite has been studied. Application of carbofuran, a carbamate pesticide, as a granule at fall planting resulted in low wheat curl mite populations and Wheat streak mosaic virus incidence (Harvey et al. 1979). Similar applications of carbofuran at spring planting did not result in wheat curl mite or Wheat streak mosaic virus suppression. Wheat curl mites survived equally well on plants with and without imidacloprid seed treatment (Harvey et al. 1998). The same study showed that an imidacloprid seed treatment generally resulted in no decrease of Wheat streak mosaic virus incidence.

## Soil-borne wheat mosaic virus (SBWMV)

**Soil-borne wheat mosaic virus (SBWMV**) is a rod-shaped plant pathogen that can cause severe stunting and mosaic in susceptible wheat, barley and rye cultivars.[1] The disease has often been misdiagnosed as a nutritional problem, but this has actually allowed in part for the fortuitous visual selection by breeding programs of resistant genotypes. Soil-borne wheat mosaic virus is part of the genus *Furovirus*. Members of this genus are characterized by rigid rod-shaped particles and positive sense RNA genomes consisting of two molecules that are packaged into separate particles that code for either replication, mobility, structure or defense against the host.[2] The virus is spread by a fungal-like protist, *Polymyxa graminis*, whose asexual secondary and sexual primary cycles help the virus spread. The disease produces secondary symptoms from the root cell infection. The disease is a serious contributor to loss in crop yield.

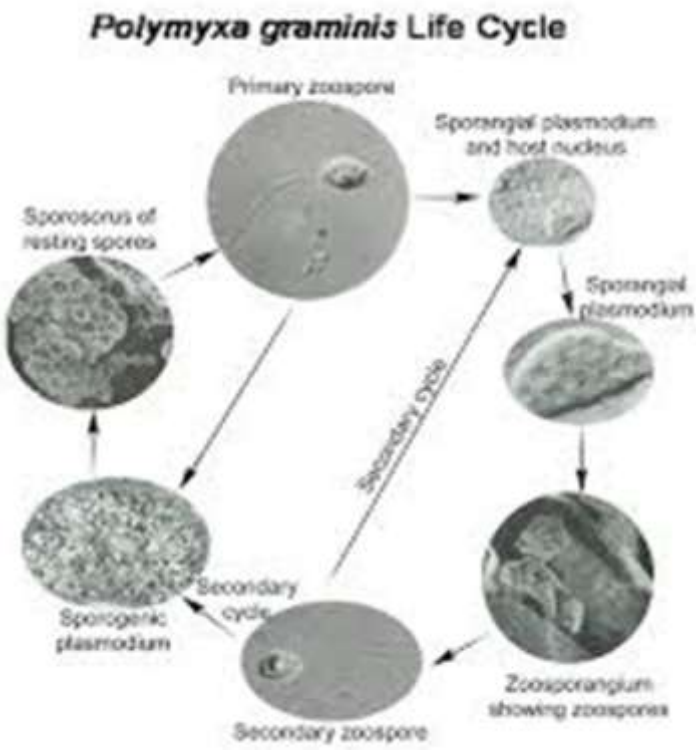
### Host and symptoms

The primary host for soil-borne wheat mosaic virus is the wheat plant, *Triticum aestivum*, although the virus can also affect rye, barley, and triticale.Symptoms of the disease are primarily found on the leaves. These symptoms include chlorotic leaf mottling or leaf mosaic, rosetting, stunting, streaking, and blotching of leaves. The mosaic and mottling symptoms may range from mild green to yellow, and leaves may sometimes also have dashes, parallel streaks, reddish streaking, and necrosis at the tips. Symptoms usually occur around the same time each year. This time is usually early spring, although in warmer climates it is possible that symptoms can emerge in late fall or early winter. Diseased fields are often uneven in appearance of symptoms especially in low wet areas. This is because the drainage pattern of water on the field is used by the virus to infect plants.

### Disease Cycle

The Soil-borne Wheat Mosaic Virus uses the fungal-like protist *Polymyxa graminis*, an endoparasitic slime mold as a vector. The *P. graminis* produces resting spores that contain the viral RNA and movement protein for up to 30 years. Dormant resting spores can germinate and produce an infection from the virus containing zoospore. The zoospores need water to reach the host so saturated soil conditions maximize dissemination. When the zoospore reaches the host plant, it encysts on the surface of a cortical root cell and develops a spear like bag which when mature will punch through the adjoining zoospore and host walls. Along with the bag, the zoospore contents as well as the virus particles are emptied into the host cortical cell. How the virus is attached to or carried by the zoospore and how the virus is transferred from the zoospore to the plant root is not fully understood although the actual virus and movement protein but not capsid protein have been found within *P. graminis sporosori*. After the cortical root cell puncture, one of two types of plasmodia of *P. graminis* may form inside. These plasmodia differentiate to give rise to either secondary zoospores (part of the asexual secondary cycle) or resting spores, the sexual primary cycle. The infection of the root cells causes substantial stunting and mosaic meaning a local infection on the root with secondary symptoms of stunting and mosaic formation.The virus itself contains two types of particles. The longer particle contains RNA 1, which is approximately 7100

nucleotides long and encodes three proteins. Two of these, measuring 150 kDa and 209 kDa, allow virus replication. The other protein is 37 kDa and allows cell-to-cell movement protein. The 150 kDa and 209 kDa proteins are translated directly from the message sense viral RNA, whereas the 37 kDa protein is expressed via a subgenomic mRNA. The shorter particle contains RNA 2 (approximately 3600 nucleotides), which also encodes three different proteins. The first is the 19 kDa coat or capsid protein (CP). Sometimes, the coat protein UGA termination codon is suppressed allowing translation of an 84 kDa CP-readthrough protein, which is believed to be required for virus transmission by its protist vector *P. graminis*.]The third protein is a 19 kDa cysteine-rich protein that is expressed via a subgenomic mRNA and may function as a suppressor of post transcriptional gene silencing countering the host resistance to the virus.[2] Optimal temperatures for *P. graminis* vary depending on where they are found: example 80-86°F in India varies in comparison to Belgium, Canada, Japan and France (59-64°F) and an optimal temperature for transmission of 59°F in New York state. Since no significant transmission occurs at 44°F, fall or spring in temperate climates are believed to be the times of the year the infections occur.




## Environment

The disease needs an environment that is conducive for infection by the swimming zoospores of the virus' vector, *Polymyxa graminis*. In dryer environments, infected plants occur in lower lying, wet regions of the field, and in humid climates or climates with more moisture, patches of infection are able to occur anywhere in the field While the disease is able to proliferate in overall dryer environments as long as there is some moisture, there is still a more optimal environment for the proliferation of the disease. The disease favors an environment with cool weather and temperatures near 60 degrees Fahrenheit, and in the US, Soil-borne wheat mosaic occurs mostly throughout eastern and central areas of the country.

## Management

Cultivar resistance to the virus is the most practical strategy to control the disease. Evidence shows it is likely that the resistance prevents the systemic movement to the foliage from the roots, although this resistance will not prevent any replication or movement of cells to roots.[3] Examples of resistant wheat cultivars include the Hawk and Newton cultivars.[8]However, information on the resistance mechanism is still lacking.[8] While there are two important aspects to Soil-borne wheat mosaic, the virus and the vector, resistance is directed more towards the virus rather than the vector. This is because the roots of susceptible cultivars and resistant cultivars can both still be colonized by *Polymyxa graminis*.[8] Resistance in the wheat line *Triticum aestivum* can be formed by crossing wheat with a wild diploid ancestor of wheat, *Aegilops tauschii*.[9] Other cultivars of resistance exist. Most cultivars are resistant to the common strains of the virus.[1] While cultivar resistance is currently the most effective form of resistance, there are a few other ways to help manage the disease. Chemical control in soil fumigants provide control against the vector *P. graminis*, but these fumigants are not feasible economically in use of small grains.[3] The sanitation of machinery is important to avoid the introduction of the virus into new areas by soil transport, and sanitation is a more economical option for the management of disease.[3] ELISA and RealTime PCR can be used to confirm diagnosis of infected plants.[10]

Importance

Soil-borne wheat mosaic virus is currently distributed over most of the eastern and central United States. Since the first European report in 1960, the virus has rapidly spread on the European continent and is abundant in France, Germany, Italy, and the United Kingdom. In the initial period of Soil-borne wheat mosaic virus research, host genotypes susceptible to rosette stunting were common and yield losses were recorded at over 50%. Today, while close monitoring for rosette phenotype has decreased the yield losses, plants still exhibit the mosaic phenotype leading to possible significant yield loss. Due to the fact that the viral symptoms are short lived and mimic nutritional deficiencies, the virus's economic significance is often overlooked. The virus contributes to lower kernel weight, tiller number, and test weight leading to lower grain yield leading to a loss in profit.[3]

Wheat Spindle Streak Mosaic Virus

Wheat spindle streak mosaic virus (WSSMV) is also known as wheat yellow mosaic virus. It was first reported in North America in 1960 from Ontario, Canada. It was first found in Kansas in Sedgwick County in 1984, but it may well have been present before 1984. Since then it has been detected in most counties in the eastern two thirds of the state. It is also present in many center pivot irrigated fields in the western counties. The disease is usually most intense in south central Kansas. The symptoms, life cycle, and field pattern of WSSMV are similar to those of wheat soilborne mosaic virus (WSBMV). This similarity probably prevented the recognition of WSSMV as a distinct disease until varieties with resistance to WSBMV became available. The similarity is only superficial, however, since they are unrelated viruses

**SYMPTOMS**

Symptoms typically appear in early spring right after green-up. In most cases, symptoms of WSSMV appear before those of WSBMV. The optimum temperature for symptom development is 50°F. Above 64°F new growth will be symptomless, but symptoms may persist on older leaves. The symptoms of WSSMV are yellow to light green streaks or dashes on a dark green background. Dashes are usually 1/8 to 1/4" long. The dashes and streaks are oriented parallel to the leaf veins and often are tapered, which gives the lesions a spindle shape. In contrast, WSBMV typically has a mosaic of green islands against a yellow background. Plants severely infected with WSSMV may have mottled leaves which resemble infection by WSBMV. In such cases, positive identification requires a serological test. The situation is confused further by the fact that many plants are found to be infected with both viruses. WSSMV also causes a mild stunting and reduced tillering. Like WSBMV, the field pattern of WSSMV tends to follow the lower, wetter areas of the field.

**DISEASE CYCLE**

WSSMV and WSBMV are both vectored by a fungus called Polymyxa graminis. The virus particles are carried on or in the fungal zoospores (swimming spores). The fungus invades root hairs of the young wheat in the fall during periods of high soil moisture. Apparently spring infections are possible but inconsequential to the wheat. The fungus forms dark clusters of resting spores in the wheat roots which are released to the soil when the roots decay. Since the fungus survives in the soil as resting spores, the disease is always associated with infested soil. Soils may remain infective for at least 8 years. Neither WSSMV nor WSBMV are insectborne or seed-borne. Wheat is the only known host of WSSMV.

**LOSSES**

We believe that losses in Kansas are usually low. Plants seem to recover well from this disease when temperatures increase in April. However, losses caused by WSSMV are difficult to measure. They are dependent both on the cultivar and the weather. Losses are roughly proportional to the length of time during which plants show obvious symptoms. A long, cool spring prolongs the symptomatic period and increases losses. In a warm spring, losses may be minimal. In Michigan, losses of soft white winter wheats

were reported to be 2 to 18%. In Canada, losses were reported to be 7 to 59%. In Pennsylvania, losses were estimated to be 7 to 24%. In Georgia, losses were estimated at 22 to 35%. In each case, loss was attributed mostly to reduced numbers of tillers.

**CONTROL**

Some of the most susceptible cultivars are Sierra, Mustang, and Thunderbird. These varieties should be avoided in fields infested with WSSMV. In addition, fields with WSSMV usually also have WSBMV, so avoid varieties which are susceptible to it as well. Although no cultivars are completely resistant to WSSMV, many wheat varieties express moderate resistance. See publication MF991, "Wheat Variety Disease and Insect Ratings," for current information on variety reactions. Late planting is sometimes effective in avoiding infection periods in the fall. Due to the longevity of the vector, crop rotation is not an effective control for WSSMV. Infection takes place at the time of wheat emergence in the fall, so avoid irrigation during this period if you have a susceptible variety.

## Barley yellow dwarf

| **Barley yellow dwarf** | |
|---|---|
| **Virus classification** | |
| Group: | Group IV ((+)ssRNA) |
| Family: | Luteoviridae |
| Genus: | ***Luteovirus*** |
| Species: | **Barley yellow dwarf virus** |

**Barley yellow dwarf** is a plant disease caused by the **barley yellow dwarf virus**, and is the most widely distributed viral disease of cereals. It affects the economically important crop species barley, oats, wheat, maize, triticale and rice.

Barley yellow dwarf virus (BYDV) is a positive sense single-stranded RNA virus; the viron is not enveloped in a lipid coating.]The virus is transmitted by aphids, and the taxonomyof the virus is based on genome organisation, serotype differences [2] and on the primary aphid vector of each isolate.

**Pathology[**

When aphids feed on the phloem of the leaf, the virus is transmitted to the phloem cells. Once inside the plant, the virus begins to replicate and assemble new virions. This process requires significant metabolic input from the plant, and causes the symptoms of barley yellow dwarf disease.

The symptoms of barley yellow dwarf vary with the affected crop cultivar, the age of the plant at the time of infection, the strain of the virus, and environmental conditions, and can be confused with other disease or physiological disorders. Symptoms appear approximately 14 days after infection. Affected plants show a yellowing or reddening of leaves (on oats and some wheats), stunting, an upright posture of thickened stiff leaves, reduced root growth, delayed (or no) heading, and a reduction in yield. The heads of

affected plants tend to remain erect and become black and discoloured during ripening due to colonization by saprotrophic fungi. Young plants are the most susceptible.

The virus is transmitted from the phloem when the aphid feeds. When the aphid feeds, virions go to the aphid's hind gut, the coat protein of the virus is recognised by the hindgut epithelium, and the virion is allowed to pass into the insect's hemolymph, where it can remain indefinitely, but the virus cannot reproduce inside the aphid.[4] The virus is actively transported into the accessory salivary gland to be released into salivary canals and ducts. The virus is then excreted in the aphid saliva during its next feeding.

The host range of BYDVs consists of more than 150 species in the Poaceae; a large number of grasses both annual and perennial are alternate hosts to BYVD and can serve as reservoirs of the virus.

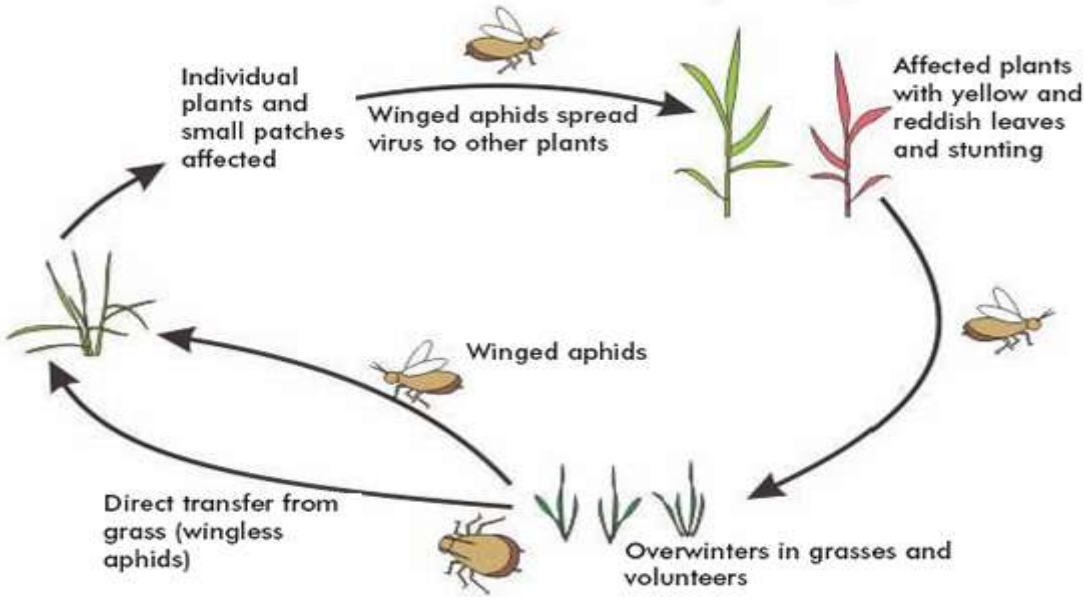
## Sources and spread

There are two main sources by which a cereal crop might be infected

1. By non-migrant wingless aphids already present in the field and which colonise newly-emerging crops. This is known as "green-bridge transfer".

2. By winged aphids migrating into crops from elsewhere. These then reproduce and the offspring spread to neighbouring plants.

**Life Cycle**




BYDV became very much more important and widespread with the increase in early-drilling of winter cereals. The virus exists as several strains and is transmitted by various species of cereal aphid. The bird-cherry aphid (Rhopalosiphum padi) is the principal vector in the south of England. In the north of England and in the Midlands the grain aphid (Sitobion avenae ) is usually more important.

In the autumn, BYDV can be introduced into cereal crops in two ways:-

1. Direct transfer by wingless aphids living on grass or on volunteer cereals which survive cultivation and move through the soil colonising the following cereal crop. This is much more common in coastal areas of the south west where cereals may follow grass and winters can be mild.
2. Indirect transfer by winged aphids migrating into newly emerged crops from grass or volunteer cereals elsewhere. BYDV introduced by winged aphids flying into crops is generally more common and important than BYDV resulting from direct transfer.

**Importance**

With the now common practise of sowing winter cereals very early, BYDV has increased in importance in many areas of the UK. The frequency of very mild winters has also meant that, for many farms, BYDV is now a regular problem. Early infections can kill patches of plants potentially resulting in large yield losses.

## Effect on yield

This is variable since it depends on viral strain, time of infection and rate of spread. Most severe losses are from early infections and can be as high as 50%.

Control

"Green bridge" sources must be ploughed in as early as possible. Alternatively, a desiccant herbicide should be applied 10 days prior to cultivation. Insecticide sprays may be used at crop emergence.

Drilling dates prior to mid-October favors attacks from winged migrant aphids. However, yield penalties may be experienced from late drilling. Insecticide sprays in this instance are therefore aimed at killing the aphids before significant spread can occur.

Products used

Synthetic pyrethroid insecticides

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