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AFRICA soil health consortium

Crop pests and diseases

A manual on the most important pests and diseases of the major food crops grown by smallholder farmers in Africa

August 2015

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Please cite this publication as: Pest and disease manual. Africa Soil Health Consortium, Nairobi, August 2015.

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About the publisher

The Africa Soil Health Consortium (ASHC) mission is to improve the livelihoods of smallholder farmers through adoption of integrated soil fertility management (ISFM) approaches that optimize fertilizer use efficiency and effectiveness.

ASHC books are available at special discounts for bulk purchases. Special editions, foreign language translations and excerpts, can also be arranged.

ISBN (e-book): 9781780648194

ISBN (paperback): 9781780648187

Typeset by Sarah Twomey

Acknowledgements

The preparation of this manual was supported by the Bill & Melinda Gates Foundation.

We would also like to thank:

Eric Boa, Erica Chernoh and Grahame Jackson who wrote the pest and disease factsheets this manual is based upon.

The many photographers who allowed us to use their excellent photographs. Individual credits and copyright status are given under each photograph.

The Africa Soil Health Consortium delivery team: Keith Sones, Dannie Romney, Victor Clottey, James Watiti, Abigael Mchana and George Oduor.

Contents

1. Introduction

This manual aims to provide clear, actionable information on the most important pests and diseases that affect the major food crops grown by smallholder farmers in Africa.

For each pest or disease, information is provided on how to recognise the problem, what to do to prevent it occurring and how to control it when it occurs. The management options for each pest or disease are divided into 'cultural approaches', such as use of resistant varieties, clean seed, crop rotations and good hygiene in the field, and 'chemical approaches', which involve the use of appropriate pesticides. In addition information is provided on the organism that causes the problem and its impact. Finally, a short list of publications and websites is given where additional information can be obtained.

A team of plant health experts has compiled this information; in doing so they have drawn upon the latest research and information available in 2015.

This manual is targeted at extension workers and anyone else who needs access to clear, actionable information on the most important pests and diseases affecting the major food crops grown by smallholders.

Crops covered are: cereals (maize, millet, sorghum, rice), legumes (groundnuts, beans and cowpeas) roots and tubers (cassava, sweet potato, yam) and banana.

This manual is an output of the African Soil Health Consortium (ASHC), a project managed by CABI. Funding for the manual was provided by the Bill & Melinda Gates Foundation.

2. Integrated pest management

2.1 What is IPM?

For each pest and disease covered in this manual, methods are first given for prevention of the problem. Then, if intervention is needed when a pest or disease occurs, the safest and most effective options available are listed.

The approach used in the manual is called Integrated Pest Management (IPM).

IPM consists of 4 main steps:

1. Identification: The section on *Key Signs* is designed to help identify the pest or disease.
2. Prevention: This includes cultural approaches, such as use of disease resistant varieties, disease-free seed and adoption of good practices in the field, such as removal of infected material that could carry the problem over to the next crop. It also includes use of pesticides where this method is appropriate.
3. Monitoring: The earlier a pest or disease is noticed, the earlier appropriate action can be taken to reduce losses and prevent its spread.
4. Control: This includes both cultural approaches, such as removal of infected plants, as well as use of appropriate pesticides.

IPM often involves the combination of several different options. Pesticides tend to be used when other approaches are inadequate for the problem at hand; they must always be used in accordance with the usage and safety information given on the pack.

2.2 Monitoring crops for pests and diseases

With most pests and diseases, the earlier the problem is noticed the better: it will be easier to take action to help prevent severe losses occurring or stop the pest or disease spreading throughout the crop and beyond to neighbouring fields.

The best way of achieving this is to regularly and systematically inspect the crop. One way to do this is to walk through the field or plot following an M-shaped pattern; this will ensure the farmer doesn't just look around the edges but also looks in the middle.

If any problems are noticed, the farmer should carefully examine the plants for signs of the problem and clues as to the cause:

Signs on the crop might include:

- Has the plant wilted – that is has the plant become less rigid than normal and is drooping?
- Are the leaves more yellow than usual?
- Have the leaves changed from green to some colour other than yellow?
- Are the plants smaller than usual?
- Have parts of the plant died?
- Are there unusual streak patterns on the leaves or stems?
- Do the leaves have spots on them?
- Are the leaves chewed – are there holes in them that look as though they have been eaten?
- Are there signs of the animals that might have done this?
- Are the leaves blistered or wrinkled?
- Are the leaves or fruits an unusual shape?
- Are the leaves smaller than usual and/or bunched closer together than usual?
- Do the leaves have patterns of lighter green and yellow giving a mottled or patchwork effect?
- Are there brown marks on the edges of the leaves?
- Is there an unusual growth on the surface of the leaves or other parts of the crop?
- Are there holes in the stem or grain?
- Are there lumps or swellings on the leaves or other parts of the plant?
- Are parts of the plant rotting – that is, becoming soft and slimy?

If any of these signs are seen the farmer should be encouraged to seek help from an extension worker, knowledgeable local farmer or staff at the local agro-dealer or research station.

3. Pesticide use and safety

3.1 What are pesticides?

Pesticides are mostly chemicals although some are biological agents, such as special types of fungi, that deter, weaken, kill, or otherwise discourage pests. They are commonly used worldwide to control a wide range of pests in agriculture.

Pests can be insects, rodents, birds and other animals, weeds, fungi, or microorganisms such as bacteria and viruses that have a detrimental impact on crops. Some kill the plants, others weaken them or reduce the yield in some way, while others attack the crop after harvest while it is being stored prior to use.

Some pests cause direct damage, such as quelea birds which feed on millet crops; other pests do not cause damage directly but can carry harmful micro-organisms such as fungi, bacteria or viruses. Fungi, bacteria and viruses can cause various diseases; for example: several species of aphid spread the virus that causes yam mosaic virus disease.

This manual includes pest and disease management options based on pesticides.

3.2 Safety and efficacy

Pesticides are considered to be easy to use and effective; however, pesticides are often misused and can cause human health effects and environmental contamination.

Furthermore, repeatedly using pesticides with the same mode of action (or method for controlling a pest) can lead to build-up of resistance, making the pesticides less effective. Where appropriate, recommendations are provided in this manual to avoid this problem.

Many countries have problems with counterfeit pesticide products being sold and used. These counterfeit products are often poorly labelled or mislabelled; they may contain too little active ingredient or even none at all. It is therefore important that farmers only buy pesticides from reputable suppliers, such as respected local agro-dealers; buying cheap products from travelling salesmen or at informal markets should be avoided.

Ideally, farmers should buy only as much pesticide as they need for the current season. This will avoid them having to store pesticides, which can represent a danger, especially for children. Fortunately, small packs of many pesticides are now readily available from agro-dealers.

It is also potentially dangerous if a friend or neighbour gives a farmer some leftover pesticide in a container other than the original packaging. In this case, the farmer will not know what the pesticide is, how it should be used or whether it is out of date.

3.3 Active ingredients and trade names

In the sections that follow management approaches based on the use of pesticides are given. In these cases the name of the active ingredient in the appropriate pesticide is given: for example, for the disease black sigatoka of banana the pesticide mancozeb can be used.

Pesticides containing mancozeb are sold under a wide range of different trade names and the amount of active ingredient in these different commercial products can vary. It is therefore essential that before any pesticide is used, the label and the leaflet provided with the product are carefully read and the instructions followed precisely. Farmers who are unable to read need to seek the help of a family member, neighbour, extension worker or the staff in the agro-dealer where they bought the pesticide.

3.4 Safe use of pesticides

When using pesticides, it is important to use them safely in order to protect both the farmer and the consumers, to reduce environmental contamination and to maintain the efficacy of the pesticides.

Farmers must use appropriate safety precautions when mixing and using pesticides. This includes reading and following the label recommendations for use, using the right personal protective equipment (PPE) and practising personal hygiene.

Information that can be found on the product label includes:

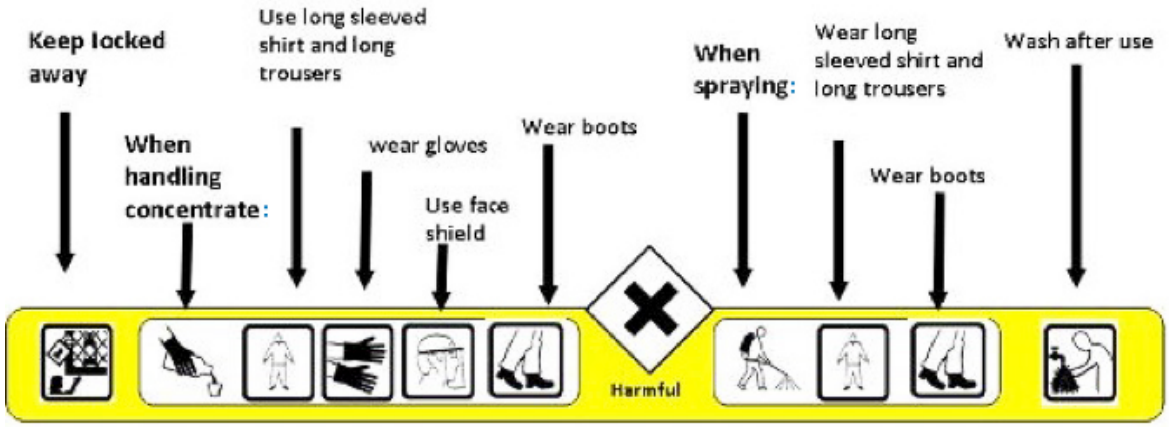
- PPE required
- What crops and pests the product can be used for
- Dosage rate
- Timing of application
- The time required before anyone can re-enter the field after spraying (REI)
- The number of days a product must be sprayed prior to harvest (PHI)
- Other precautions

It is important that farmers read and understand what is on the label prior to use. If they are unable to read it or do not understand it, then they should find someone to help them, such as a local extension agent or family member.

Farmers should be encouraged to use personal protective equipment (PPE) when mixing and spraying pesticides. At a minimum, a farmer should wear:

- Long sleeved shirt
- Long trousers
- Goggles, glasses or a face shield to protect the eyes
- Boots (preferably rubber or impermeable boots)
- Gloves (preferably rubber or impermeable gloves)
- Dust mask (for dry formulations) or respiratory protector
- Hat

Warning symbols are used on labels to indicate what type of PPE should be used for both mixing and spraying the product.



Pictograms on a sample pesticide product (Image: Stewardship Community[1])

A colour code system, based on the WHO pesticide toxicity classification, is used on labels to inform farmers about the level of toxicity of the product: red indicates the most dangerous pesticides.

WHO Class and Colour Code	
Ia	Extremely hazardous
Ib	Highly hazardous
II	Moderately hazardous
III	Slightly hazardous
U	Unlikely to present acute hazard
O	Obsolete as a pesticide

It is also important to practise good personal hygiene when applying pesticides. Farmers should never eat, drink or smoke when mixing or spraying pesticides. They must wash the equipment and bathe after each use. If any chemicals leak or splash into their eyes or onto their skin, they should immediately wash them off.

When not using pesticides, they should be kept locked away in a safe place that is inaccessible to children. Pesticide containers should never be reused for other purposes. Empty containers should be rinsed three times with water and punctured with a sharp object. Dispose of the containers following the local laws and regulations. Also, pesticides must never be repacked into containers that previously contained food or drink as this could lead to accidental consumption. There are different types of pesticides, including those which kill insects (insecticides), weeds (herbicides) and disease causing fungi (fungicides). There are no pesticides that kill viruses and very few that kill the bacteria that can infect plants.

1 www.stewardshipcommunity.com

4. The major pests and diseases of cereals

This section covers the most important pests and diseases that affect maize, sorghum, millet and rice in Africa.

Table 4.1 summarises the main signs associated with the most important pests and diseases of maize in Africa.

Table 4.2 summarises the main signs associated with the most important pests and diseases of millet in Africa.

Table 4.3 summarises the main signs associated with the most important pests and diseases of sorghum in Africa.

Table 4.4 summarises the main signs associated with the most important pests and diseases of rice in Africa.

In the following pages, more details on the signs, management options, cause and impact are provided.

Table 4.1: Key signs of the most important pests and diseases of maize in Africa

Key signs	Possible pest or disease	See page
Small holes and small dark caterpillars in leaf funnel. Small holes in straight lines on the young leaves. Larvae droppings on leaves and in stems. Central leaves become dry and withered. Weak stems that break. Top of the plant wilts and turns yellow then dries out and dies.	Maize stalk borer	13
Tunnels and irregular shaped holes in stored grain and large quantities of dust.	Larger grain borer	15
Small caterpillars: yellowish-white to reddish-brown, dark brown to black head, black rows of short hairs running along backs. Fully grown caterpillars 35-40 mm, dark brown, yellow-green, to red-brown, with grey-black stripes along the side. Bore holes in cobs.	Cotton bollworm	17
Spots surrounded by yellow haloes on lower leaves. Spots expand parallel to the leaf veins: light brown to grey, rectangular, up to 70 mm.	Grey leaf spot	27
Yellow-green, brown or black powdery mould on maize ears.	Aspergillus fungus	29
Pink or red, woolly mould between the ear and the husk, usually starting at tip of ear and advancing downwards.	Fusarium fungus	29
Scattering of small yellow areas on leaves which merge; leaf becomes paler before the edges go brown and dry inwards. Sometimes young leaves die before they expand.	Maize lethal necrosis disease	31
Stunted, yellow, scorched and wilted plants when the soil is still moist. Attractive, brightly coloured (often purple) flowering weeds.	Striga or witchweed	47

Table 4.2: Key signs of the most important pests and diseases of millet in Africa

Key signs	Possible pest or disease	See page
Small caterpillars: yellowish-white to reddish-brown, dark brown to black head, black rows of short hairs running along backs.	Cotton bollworm	17
Base of leaves turn yellow. Plants stunted. Lack flowers or parts of the flower head become leaf-like. At high humidity, downy spores appear on yellow areas, particularly on the lower surface.	Downy mildew of pearl millet	33
Small green to yellow slightly raised spots on leaf surface, more on top side than underside. Develop into reddish orange pustules, round to elliptical, with yellow haloes. Leaves start to die from tips towards base. If spots occur on the stems, plants fall over.	Rust of pearl millet	35
Huge flocks of small brown birds with red beaks feeding on millet and other small grains.	Quelea (weaver finch birds)	45
Stunted, yellow, scorched and wilted plants when the soil is still moist. Attractive, brightly coloured (often purple) flowering weeds.	Striga or witchweed	47

Table 4.3: Key signs of the most important pests and diseases of sorghum in Africa

Key signs	Possible pest or disease	See page
Small caterpillars: yellowish-white to reddish-brown, dark brown to black head, black rows of short hairs running along backs. Fully grown caterpillars 35-40 mm, dark brown, yellow-green, to red-brown, with grey-black stripes along the side.	Cotton bollworm	17
Shrivelled or flat grains that result in empty or chaffy spikelets and blighted or blasted looking panicles (heads).	Sorghum midge	21
Feeding marks on the funnel leaves, ‘dead heart’ and holes in the stems.	Sorghum stem borers	23
Pale leaves with white stripes. Leaves narrower and more erect than usual. White areas dry up and go reddish-brown. Leaves shred. Plants stunted and usually produce no grain. Downy or woolly appearance on underside of leaves.	Sorghum downy mildew	37
Huge flocks of small brown birds with red beaks feeding on millet and other small grains.	Quelea (weaver finch birds)	45
Stunted, yellow, scorched and wilted plants when the soil is still moist. Attractive, brightly coloured (often purple) flowering weeds.	Striga or witchweed	47

Table 4.4: Key signs of the most important pests and diseases of rice in Africa

Key signs	Possible pest or disease	See page
Maggots burrow into young shoots, which then stop growing. Swollen area (gall), where the maggot transforms into pupa, up to 1.5 metres long: these silver shoots are associated only with gall midge damage. Plants stunted and bushy.	Rice gall midge	25
Grey-green streaks starting from the tips and margins on seedling leaves; later the streaks join together, turn yellowish-white with wavy edges, dry up and die. Leaves wilt, dry up and die. Older plants have, pale yellow steaks with wavy margins from leaf tips towards base.	Bacterial leaf blight	39
Small, oval or diamond-shaped spots, often with yellow haloes, with dark borders on leaves. Under moist conditions large number of spores on both sides of the leaves. Spots lengthen, centres whitish-grey and borders red-brown; spots join together and leaves die. Various rots. Severely infected fields have scorched appearance.	Rice blast	41
Newly emerged leaves show yellow (or orange) and green stripes. Plants stunted. Few tillers, panicles fail to emerge completely, grain is unfilled or sterile.	Rice yellow mottle disease	43
Huge flocks of small brown birds with red beaks feeding on rice and other small grains.	Quelea (weaver finch birds)	45
Stunted, yellow, scorched and wilted plants when the soil is still moist. Attractive, brightly coloured (often purple) flowering weeds in upland rice.	Striga or witchweed	47

Maize stalk borers

Busseola fusca; Chilo and Sesamia species

Photo: CIMMYT, Flickr, CC BY-NC-SA 2.0, http://bit.ly/1amGK8S

Maize stalk borer larvae and frass inside a young maize tassel.

Photo: International Institute of Tropical Agriculture, Flickr, CC BY-NC 2.0, http://bit.ly/1FEC61c

Larva feeding on maize stem.

SUMMARY: Maize stalk borers are pests of maize, sorghum and other crops throughout many countries in Africa. The caterpillars bore into the stem of maize, feed on the internal tissues and cause the plant to wither and die. The pest can be controlled through a combination of cultural practices (most notably intercropping and the 'push-pull' system) and chemical insecticides or neem powder (but only at the early stage, before the larvae have bored into the stem).

KEY SIGNS

Maize stalk borers are common pests throughout sub-Saharan Africa. There are three main species of stalk borers which attack maize: the African maize stalk borer (also attacks sorghum); the spotted stem borer (also attacks sorghum, bulrush millet, sugarcane and rice) and the African pink stem borer (also attacks finger millet, sugarcane and rice). The behaviour and development of all three is very similar. The larvae (caterpillars) first attack young plants, feeding on the leaves and entering the stem, damaging and reducing grain production or killing the plant.

The eggs of the African maize stalk borer are round, flat on top, creamy-yellow in colour and about 1 mm diameter. The larvae have no distinct features: they are creamy-white in colour often with a grey or sometimes pink colour and the head is dark brown. The larvae grow up to 40 mm long. Pupae are up to 25 mm long and shiny yellow-brown to dark brown in colour. The adult moths have a wing-span of about 25-33 mm. The forewings are light to dark brown with dark patterns and the hindwings are light to greyish-brown. Colouring varies somewhat depending on location and season.

The spotted stem borer larvae are creamy white to yellowish-brown with dark conspicuous spots and four purple stripes lengthwise across the back. The pupae are shiny, light yellow-brown to dark red-brown and 15 mm long. The adult moths are 7-17 mm long with a wingspan of 20-25 mm. The forewings are light yellow-brown with darker horizontal patterns, and the hind wings are white. The eggs are creamy-white, scale-like and are laid in overlapping batches on the underside of leaves near the midrib.

The African pink stem borer eggs are creamy-white when laid, but get darker as they develop. The larvae are smooth and shiny, creamy-white with a distinctive pink colouring, a brown head, and 30-40 mm long when mature. The pupae are yellowish-brown and 18 mm long. The adult moths are slightly smaller than the other two species of stem borers. They have yellowish-brown forewings, white hindwings, and a wingspan of 20-30 mm.

On young plants that have been attacked small holes or small dark larvae in the leaf funnel can be seen. An early sign is small holes in straight lines on young leaves. Larvae droppings (frass) are often visible on the leaves and in the stems. Larvae also attack the growing points, forming 'dead hearts' (meaning the central leaves become dry and withered). Older larvae feed inside the stems, weakening them and causing them to break. The top of the plant wilts, turns yellow, and eventually dries out and dies. If plants show symptoms, cut open the stem and look for larvae, pupae and frass.

MANAGEMENT

Prevention – what to do before signs are seen

Cultural approaches: Resistant or tolerant varieties are available in some countries and should be used. Applying nitrogen, either as a mineral fertilizer, or as manure or compost, enhances the crops ability to sustain an attack.

To prevent the stalk borer, a 'push-pull' system can be implemented in which *Desmodium*, a repellent plant, and Napier grass, a trap crop, are intercropped with maize to push and pull the insect away from the maize. Plant Napier grass (the Bana variety is the best) along the border around the maize field and plant one row of *Desmodium* (silverleaf or greenleaf varieties) between every three rows of maize. The *Desmodium* should be planted first, as soon as the rains begin, so that it begins to repel the stalk borers before the maize emerges. At least three rows of Napier grass should be planted around the borders of the maize field. The *Desmodium* produces a smell that the adult moths do not like; this pushes the moths away from the maize. The stalk borers are more attracted to Napier grass than maize and so the border of Napier grass will pull the moths away from the maize to lay their eggs on the Napier grass. When the larvae bore into the Napier grass, however, the plant produces a sticky glue-like substance that traps them and they die. An additional benefit of this system is that *Desmodium* is a legume that fixes nitrogen into the soil; it also acts as a ground cover that supresses Striga, a parasitic weed. Disadvantages of this system include the space taken up by the Napier grass; the cost and lack of availability of *Desmodium* seed; and the difficulty in establishing the *Desmodium* crop.

Intercropping with non-host plants, such as cowpeas or cassava, will also reduce the damage. Adult moths will lay eggs on the non-host plants, but the larvae are unable to feed on them and will die.

Dispose of crop residues after harvest to reduce stalk borer populations and limit the pest the following season. The crop residues can be burned, used as feed for livestock, or left on the ground exposed to the sun's heat for one month to kill the larvae and pupae.

Rotate maize with a non-host plant, such as a legume (for example groundnut), to increase the nitrogen in the soil. This will make the next maize crop hardier and less susceptible to an attack and break the cycle of the stalk borer.

Control – what to do after signs are seen

Chemical approaches: Chemical control can be used early in the season by applying appropriate insecticides, such as trichlorfon, as granules or dusts to the leaf funnel of young plants. Neem powder can be effective and should be applied as a 1:1 mixture with dry clay or sawdust to the funnel of the plant: 1 kg of neem powder can treat 1500-2000 plants. Once the larvae have bored into the stem of the plant, insecticides are no longer effective.

CAUSE

Busseola fusca is the scientific name of the African maize stalk borer. Common names include maize stalk borer, maize stem borer and sorghum stalk/stem borer. *Chilo partellus*, the spotted stem borer, is the most common of the *Chilo* species found in Africa, while the African pink stem borer, *Sesamia calamistis*, is the most common species of *Sesamia*. *Sesamia* are quite similar to B. fusca during the larval stage, but can be distinguished during the pupal and adult stages. *Busseola fusca* can also be confused with other African species of *Besseola, Poeonoma* and *Manga,* which have similar wing patterns but are rarely found on crops.

The adults lay batches of 30-100 eggs under the edges of leaf sheaths or in long columns up the stem. They prefer young plants or the youngest unfolded leaves. The larvae hatch a week later and move all over the plant, eventually entering into the leaf whorls (or funnel) to feed and tunnel into the stems. They feed inside the stems for 3-5 weeks. Before pupating they make an exit hole, through which the adult moth will eventually emerge, and then pupate in the tunnels they created in the stem for 9-14 days. When the adult moths emerge from the stem in the late afternoon or early evening, the females immediately release a pheromone to attract a male and mate, starting the cycle again. The adult moths are active at night, resting on plants and plant debris during the day. The total life cycle is 7-8 weeks long, but during dry or cold periods it can be up to 6 months (as they stop developing and remain in stems or plant debris).

IMPACT

Yield losses up to 10-12% have been reported for maize. The African maize stalk borer is primarily a pest of maize and sorghum; other hosts include pearl millet, finger millet and sugarcane. Many wild grasses are also hosts, including Johnson grass (*Sorghum halepense*), elephant grass (*Pennisetum purpureum*), wild Sudan grass (*Sorghum verticilliflorum*) and Guinea grass (*Panicum maximum*).

DISTRIBUTION

B. fusca is more common at high altitudes (above 1000m), and Chilo and Sesamia species at lower altitudes (below 1500m). The African maize stalk borer is native to sub-Saharan Africa and is present throughout the region from sea level to 2,000 meters altitude. The stalk borers are often spread through the transport of dry stems, grains and grasses that contain resting (diapausing) larvae.

FURTHER READING

Plantwise Knowledge Bank www.plantwise.org/knowledgebank
BioNET-EAFRINET http://keys.lucidcentral.org/keys/v3/eafrinet/index.htm

Larger grain borer

Prostephanus truncatus

Photo: Pest and Diseases Image Library, CC BY-NC 3.0 US, www.bugwood.org

Adult larger grain borer.

Photo: CABI

Large gain borer adult boring into maize grains.

SUMMARY: The larger grain borer is a serious pest of stored maize throughout Africa. The beetle eats its way into the grain leaving a hole and empty shell. Having a clean storage facility is important to prevent the pest from becoming unmanageable. Shelling the grains from the cob prior to storage can minimize the damage.

KEY SIGNS

The larger grain borer (LGB) is a major pest of stored crops, such as maize, dried cassava roots and yams. Although it is considered a post-harvest pest, the LGB attacks maize both before and after harvest. The adult beetles bore into maize cobs, husks and grains leaving tunnels and empty grain shells, and producing large quantities of grain dust. The adults prefer grain that is still attached to the cob rather than loose-shelled grain; damage is often greater on unshelled maize. The pest often bore into the wood used to construct the stores in which the grains are kept.

Adult females lay eggs inside the tunnels or near the food source. The larvae bore into the grains or feed on the dust produced by the adults, and pupate in the grain, cob and dust. The adults are about 3-4.5 mm long, cylindrical in shape, shiny, dark brown-black in colour with their heads turned down. The larvae are white, grub-like, thinly covered with hairs and have three pairs of legs.

Early detection of the pest is difficult unless the populations are high because they develop inside the cob or grain and are therefore not visible. Flight traps (e.g. funnel, delta or wing traps) baited with pheromones can be used by plant protection officers and researchers to monitor for large infestations of adult beetles. Farmers can look for tunnels and irregular shaped holes in stored grain and large quantities of dust.

MANAGEMENT

Prevention – what to do before signs are seen

Cultural approaches: Use maize varieties with a good husk cover that provide protection and are less likely to be attacked.

Having a clean storage facility is important to prevent or limit LGB infestations. The storage facility should be cleaned thoroughly between harvests. Any old stock should be removed and burned before bringing newly harvested grain into the storage facility.

Sacks used to harvest or store grain should be immersed in boiling water to kill any remaining infestations.

Do not put crops that show signs of infestation inside the storage facility.

If using a wooden storage facility, inspect the wood for tunnels and remove infested boards. Wood is a host of the LGB; if possible, farmers should be encouraged to construct storage structures using brick or other non-wood materials such as metallic containers, old oil drums, mud or cement-plastered basket cribs.

Roofs should be constructed with iron sheets instead of grass-thatch to prevent the pest from harbouring and breeding in the roof. If using grass-thatch, the roof should be thick and cone shaped.

Shell cobs before storing and dry to a moisture point below 12% (or when it is too difficult to bite through the grain with your teeth).

Sell maize stock within three months after harvest when infestation of the LGB is lower.

Destroy crop residues after harvest and burn cob cores after shelling.

Control – what to do after signs are seen

Cultural approaches: Harvest shortly after the maize has matured, before the LGB has an opportunity to enter the grain.

Mixing the stored grain with a dust, such as ash or clay, causes the beetles to desiccate and can help reduce populations.

At the regional or national level, releasing the predatory beetle *Teretrius nigrescens* can maintain the populations at a tolerable level.

CAUSE

The LGB is currently classified as *Prostephanus truncatus* but used to be classified as *Dinoderus truncates* or *Stephanopachys truncates*. It is commonly referred to as the larger grain borer (LGB), but is also called the greater grain borer (GGB) in some countries to distinguish it from the lesser grain borer (*Rhyzopertha dominica*). It is easy to confuse the LGB with other species such as the lesser grain borer (*Rhyzopertha dominica*) and the maize weevil (*Sitophilus zeamais*). The lesser grain borer is smaller, narrower, rounder and dark brown-red in colour; the maize weevil is rounder at the end of the body.

The entire life cycle from egg to adult is completed in about 25 days under favourable conditions (27-32°C and 70-80% relative humidity), a little longer under cooler or drier conditions. The LGB prefers high temperatures and relatively high humidity, but it is a hardy pest that can develop in grains with low moisture content.

IMPACT

LGB can cause significant losses in stored grain, ranging from 19-30% after 6 months storage. LGB causes a weight loss of the grain, as well as reduced quality and nutritional value, leading to economic losses for farmers and traders. Furthermore, there are strict quarantine measures against the pest; detection can result in rejection of traded goods. Host plants include yams, cassava, sorghum, wheat, maize and other materials such as wood products, gourds, bamboo and even plastic and soap. LGB can develop in forested areas therefore complete eradication is nearly impossible. In heavily infested areas, wooden storage structures may provide a host to the pest from which it can develop and attack the next harvested crop.

DISTRIBUTION

The LGB is native to Central and South America. In East Africa, it was first introduced into Tanzania in the 1970s. Since then it has spread to Burundi, Kenya, Malawi, Mozambique, Namibia, Rwanda, Zambia, South Africa and most likely other countries in the region. It was introduced separately to West Africa, where it was first detected in Togo in 1984, and has spread to Benin, Burkina Faso, Ghana, Niger and Nigeria. The LGB is distributed across countries and regions through the import and export of infested grains. Locally, the LGB is distributed through local trade of grain (e.g. in contaminated sacks and lorries) and by adult beetles flying from one location to another.

FURTHER READING

Plantwise Knowledge Bank http://www.plantwise.org/knowledgebank/datasheet.aspx?dsid=44524

FAO: A guide for marketing maize for extension officers http://www.fao.org/docrep/005/x0530e/x0530e05.htm

Infonet-Biovision http://www.infonet-biovision.org/default/ct/91/pests

Cotton bollworm

Helicoverpa armigera

Photo: Donald Hobern, Wikimedia Commons, CC-BY-2.0, http://bit.ly/1a8PJuf

Adult moth of cotton bollworm.

Photos: Gyorgy Csoka, Hungary Forest Research Institute, CC BY 3.0 US, www.bugwood.org

Cotton bollworm caterpillars in two different colours.

SUMMARY: The cotton bollworm is a major pest of many important food, oil and cash crops worldwide, including cereals, legumes, fruits and vegetables. A severe infestation of caterpillars of this moth can cause a complete loss in yield. Chemical control needs to be carefully timed as the caterpillars bore into the grains or fruit of the plant and are then protected. Resistance to pesticides, such as pyrethroids, has been reported in many countries. *Bacillus thuringiensis* (Bt) and neem extracts provide effective control against the caterpillars while minimising damage to natural enemies. Important cultural controls include removal and destruction of post-harvest crop residues, ploughing the soil to expose the pupae and uniform planting times.

KEY SIGNS

The cotton bollworm is considered to be one of the most important pests worldwide, attacking over 200 species of plants. The caterpillars feed on leaves and developing grain heads, and bore into the fruiting parts of plants.

The eggs are 0.4-0.6 mm across, round and yellowish-white, but change to a dark-brown colour before hatching. The eggs are typically laid singly on plants that are flowering or about to flower. The young caterpillars are yellowish-white to reddish-brown, with a dark brown to black head and black bumpy rows of short hairs running lengthwise on their backs. The fully grown caterpillars are 35-40 mm long and vary in colour from dark brown, yellowish-green, to reddish-brown, with greyish-black stripes along the side of the body and a thin light stripe. The pupae are 14-16 mm long, brown, and smooth on the surface. The adults also vary in colour, but typically the forewings are yellowish-brown with black kidney shaped markings in the middle of the wings and 7-8 small spots in a line along the border of the wing. The hindwings are white with a darker coloured band along the edge. The wingspan is about 35-40 mm at its widest point, and the body is 14-18 mm long. The adult moths are active at night and are good at flying, so they can easily move from plant to plant and field to field.

The caterpillars typically feed on the buds, flowers, grains, fruits and pods, but will also feed on the leaves and stems. In sorghum and millet, they feed on the developing grains during the milky stage and hide in the head during the day. In beans and tomato, they bore into the fruit. In maize, they feed on the developing seeds and bore into the cobs. The bore holes left by the caterpillars provide an entry point for bacterial diseases.

Monitoring is extremely important to prevent a severe outbreak. Monitoring should be done one or two days per week once the sorghum flowers bloom. Caterpillars can be seen on the plants, or shake the panicle heads over a bucket or sweep net. Bore holes and caterpillar dropping (frass) can often be seen on the plant. Pheromone traps and light traps can also be used to monitor the adult moths and provide some control.

MANAGEMENT

Prevention – what to do before signs are seen

Cultural approaches: Sorghum varieties with tight compacted heads are more likely to be attacked; plant varieties with loose open heads to reduce infestations and damage.

Resistant varieties of cotton, pigeonpea and chickpea have been released. Talk to local agro-input suppliers to see which varieties are available.

Planting a whole field at the same time (uniform planting) prevents movement of the moths from the older plants to the younger plants and reduces the damage.

Two post-harvest practices are important for preventing the population of bollworms from building up and becoming a severe problem the following season. First, it is important to remove and destroy crop residues immediately after harvest. Second, plough the soil to expose the pupae to direct sunlight and natural enemies.

Rotating with non-host crops can also prevent the population from building up, but because the bollworm can attack and harbour on many species of plants, rotation must be used together with other prevention and control practices.

Control – what to do after signs are seen

Cultural approaches: For small plots, it is possible to hand pick and destroy the eggs and young caterpillars.

There are many natural enemies of the bollworm, including *Trichogramma* spp. (egg parasitoids), several species of wasps and flies (caterpillar parasitoids), as well as many predators such as ants, lacewings, ladybugs, assassin bugs, minute pirate bugs and birds. Providing habitat to attract and preserve these natural enemies is an important strategy for controlling the bollworm.

Chemical approaches: The older caterpillars feed inside the fruiting part of the plant, protecting them from pesticide sprays. Therefore, if pesticides are used, they should be applied before the caterpillars bore into the pods or fruit of the plant. It is important to monitor the field regularly to look for eggs and young caterpillars before they bore into the plant. The decision to use a pesticide needs to be based on the severity of the problem, the presence of natural enemies and the economic value of the crop.

The cotton bollworm has developed resistance to several pesticides, notably synthetic pyrethroids.

Two microbial pesticides, *Bacillus thuringiensis* subspecies *kurstaki* and *Bt aizawai*, and *Helicoverpa armigera* nuclear polyhedrosis virus (HaNPV) can be used to control the young caterpillars with minimal harm to natural enemies. There are commercial products of these microbial pesticides available in many countries. Neem extracts, made from the neem seeds and leaves, and neem oil are also reported to be effective against the larvae and eggs.

CAUSE

Helicoverpa armigera has also been classified as *Heliothis armigera*. It is commonly known as the cotton bollworm, but is also called the African bollworm, tomato fruitworm, corn earworm, gram pod borer, old world bollworm and tobacco budworm. In French it is called chenille des epis du mais, noctuelle des tomates, or ver de la capsule.

Adult females can lay hundreds of eggs in a lifetime. They lay eggs singly on the tender parts of plants including the leaves, flowers, or fruit, and the eggs hatch in 3 to 5 days. The caterpillar period lasts 17 to 35 days, and the pupa period 17 to 20 days. The caterpillars drop from the plant to pupate inside a silk cocoon, 3-15 cm deep in the soil. The adult moths live between 1 and 25 days. The total life cycle can last up to 60 days; if conditions are favourable, then the total life cycle can be completed in 30 days. The length of time for development is mostly dependent on availability of food and the temperature: development time is shorter at higher temperatures. In warmer areas there can be numerous cycles per season.

IMPACT

The cotton bollworm is a major pest of many crops, including sorghum, soy, cotton, maize, common beans, chickpeas, groundnut, sunflower, potatoes, winter grains, citrus, wheat, barley, oats and a wide range of vegetable crops, including okra. The larvae can develop on many wild plant species, including *Amaranthus*, *Cleome* and *Acalypha*. The bollworm has a preference for feeding on the flowering and fruiting parts of plants, which causes high economic losses. If the infestation is severe, then it can result in a complete crop loss.

DISTRIBUTION

Native to Africa, the cotton bollworm has spread to Asia, Europe, Australia and the Pacific Islands. There have also been recent reports of infestations in South America. The pest can be spread through infested plant propagation materials. The adults are strong flyers and can move easily from field to field in areas where host plants are available; they can also be spread by strong winds.

FURTHER READING

Plantwise Knowledge Bank www.plantwise.org/knowledgebank

Millet stem borer

Coniesta ignefusalis

SUMMARY: Stem borers are a major pest of millet in the Sahelian and sub-Saharan regions. The larvae (caterpillars) of this moth tunnel into the stems causing them to fall over (lodging), 'dead hearts' and poor grain development. The use of chemicals is rarely justified due to difficulty in timing the application and cost. A combination of cultural practices, such as early planting, practicing intercropping or the 'push-pull' system, and managing crop residues are the most effective approaches to controlling the pest.

KEY SIGNS

The stem borer is an important pest of pearl millet, especially in West Africa. The larvae of the stem borer attack the growing points and leaves of millet, and bore into the stem of the crop causing 'dead hearts'. In wet regions, there are three generations or cycles of the pest per year; two cycles in the drier regions. Millet that is planted early is susceptible to attacks by the first generation of larvae, which cause damage to the young plants and dead-hearts. Late-sown millet is susceptible to the second and third generations of larvae, which attack the seedlings producing tunnels in the stems that can kill the plant. In older plants, tunnelling into the stem can also cause lodging, panicles covered in chaff and inhibition of grain formation.

The eggs are laid in batches of 20-25 between the leaf sheaths and stem or on lower leaf blades. The eggs are 1 mm long, elliptical and yellowish-white in colour. Larvae are about 20 mm long when fully grown, have a reddish-brown head and a creamy white body, with distinct black spots along the body during the wet season. These spots disappear during the dry season when the larvae enter into diapause (a resting period). The pupae are yellow to reddish-brown and grow up to 15 mm long. The adult moths have a wing span of about 8 to 15 mm, golden-brown forewings and white silky hind wings. The moths are active at night; during the day they hide on leaves, stems and plant debris, with their heads pointing down towards the ground. The total life cycle takes about 30 to 40 days during the wet season.

Pheromone bait traps have been successfully used to monitor and mass trap the millet stem borer in many countries. ICRISAT has published a research brief describing how to build and use the pheromone traps (see *Further Reading*).

MANAGEMENT

Prevention – what to do before signs are seen

Cultural approaches: Use resistant varieties if locally available.

Plant early, with the first rains, to avoid a heavy infestation of the stem borer.

Intercropping millet with non-host crops, such as cowpeas, will confuse the moths, causing them to lay eggs on the non-host crops. The larvae are then unable to eat the non-host crops and will die.

The 'push-pull' method is effective against stem borers. In this method, *Desmodium* is intercropped with millet and acts as a repellent that 'pushes' the pest away from the millet. Around the border of the field a trap crop, such as Napier or Sudan grass, is planted to attract or 'pull' the stem borer moths away from the millet. The moths lay eggs in the trap crops; when the larvae bore into the Napier grass, the plant produces a sticky glue-like substance that traps the larvae and they die.

There are several natural enemies of the stem borer that attack during different stages of the life cycle. *Telenomus busseolae*, a parasitic wasp, is present in several countries in West Africa. The females lay eggs inside the eggs of the stem borer and develop within the egg. Another group of wasps, species of the *Syzeuctus* genus, are larval parasites and are present in many countries. Ants are also predators of stem borers. Planting strips of flowering plants in or around the field will attract and conserve the natural enemies.

Following harvest of the grain, the stem borer larvae can survive until the next rainy season in the stems and stubble of millet. It is important therefore to destroy crop residues after harvest, to prevent the pest from re-emerging in the field the following season. If the stems are to be used for building structures, they should be partially burned to kill the larvae inside the stem.

Rotate millet with a non-host crop, such as a legume, to break the pest's life cycle.

Control – what to do after signs are seen

Cultural approaches: Locally made inexpensive pheromone bait traps can be placed in and around the field to disrupt the mating cycle of the stem borers and reduce the populations. The traps are particularly beneficial if placed along fences and granaries, or other places the stem borer may hide.

Chemical approaches: The use of insecticides, such as dimethoate, is impractical, and rarely justified due to the costs and difficulty in use. The eggs of the stem borer are often covered by the leaf sheaths, and the larvae bore into the stems, therefore the pest is often protected from chemical sprays.

Neem can be effective against stem borers if used early in the season before the larvae bore into the stem of the plant; it should be applied in the evenings.

CAUSE

The moth *Coniesta ignefusalis* is the stem borer that causes the most damage to millet. The majority of the damage is caused by the larvae feeding in the stem and preventing the flow of nutrients from the roots to the upper parts of the plant. Pearl millet is the primary host plant, but sorghum, maize, sugarcane and several species of wild grasses are also hosts. Sorghum is especially susceptible when intercropped with maize.

IMPACT

There is little data that reports on crop losses from farmers' fields. Some studies have reported that yield losses can average 15%, although severe cases can result in total crop failure.

DISTRIBUTION

The millet stem borer is a major pest of millet, especially in the Sahel and sub-Saharan region of Africa. *C. igenfusalis* has been recorded in Benin, Burkina Faso, Chad, Gambia, Ghana, Guinea Bissau, Mali, Mauritania, Niger, Nigeria, Senegal, Sudan, Ethiopia and Angola as well as some parts of West Africa. It can live at altitudes up to 1000 m above sea level. Stem borers are mainly distributed from country to country or region to region by diapausing (dormant) larvae in the stems and other crop residues. Millet stems are often used for roofs, fences and other building uses; it has been reported that attacks are more severe near villages where the stems are used for this purpose.

FURTHER READING

Youm, O., Harris, K.M., and Nwan'ze,K.F. 1996. Coniesta ignefusalis, the Millet stem borer: a handbook of information. Information Bulletin no. 46. Patancheru 502 324, Andhra Pradesh, India: International Crops Research Institute for the Semi-Arid Tropics. http://pdf.usaid.gov/pdf_docs/pnaby140.pdf

ICRISAT. Pheromone-based monitoring system to manage the millet stem borer Coniesta ignefusalis (Lepidoptera: Pyralidae) http://www.icrisat.org/what-we-do/agro-ecosystems/aes-rb-monitoring-system.htm

FiBL (2013): African Organic Agriculture Training Manual. Version 2.0 July 2013. Edited by Gilles Weidmann and Lukas Kilcher. Research Institute of Organic Agriculture FiBL, Frick. http://www.organic-africa.net/fileadmin/documents-africamanual/training-manual/chapter-09/Africa_Manual_M09-03.pdf

AgriGuide http://www.agriguide.org/index.php?what=agriguide&id=148&language=wo

Sorghum midge

Stenodiplosis sorghicola / Contarinia sorghicola

Photo: Alton N. Sparks, Jr., University of Georgia, CC BY 3.0 US, www.bugwood.org

Adult midge on sorghum bloom.

Photo: Eric Boa, CABI, CC BY 4.0

Damaged sorghum heads.

SUMMARY: The sorghum midge is one of the most important pests of sorghum. The larvae of the midge feed on developing seeds causing malformation of the grain and empty or chaffy heads. Cultural controls are the best approach; chemical sprays have to be carefully timed since the pest spends the majority of its life cycle protected inside the spikelets. Using resistant varieties, planting early and planting varieties that flower uniformly are the most important methods that can be used to reduce damage to the crop.

KEY SIGNS

The sorghum midge is one of the most important pests of sorghum worldwide. The adult midge lays eggs into the flowering spikelets of sorghum. When the larvae hatch they feed on the developing seeds, causing poor grain development and resulting in empty grains or chaffy spikelets.

Adult midges have a mosquito-like appearance, are small (about 3 mm long), with a bright orange body, transparent wings and very long antennae. The eggs are very small, cylindrical and a transparent light-red colour. The young larvae are colourless, but turn dark-orange as they develop. The larvae feed on the young soft ovary of the developing grain, which causes the grain to shrivel and prevents normal seed development. Populations of midge begin to build-up at the start of flowering. A long flowering period (due to staggered planting dates or planting varieties that flower at different times) can significantly increase the midge population in an area. Two or three generations of midge can develop during a season, resulting in higher populations and therefore more damage to late flowering sorghum.

Symptoms include shrivelled or flat grains that result in empty or chaffy spikelets and blighted or blasted looking panicles (or heads). Damaged spikelets can be squeezed between fingers to see if they produce red ooze (the larva or pupa being crushed). A small empty, clear or white pupa casing (or skin) is left attached to the tip of the spikelet and can be seen with the naked eye. Adult midges can be observed in the mornings during flowering.

MANAGEMENT

Prevention – what to do before signs are seen

Cultural approaches: Resistant varieties are one of the most effective ways of controlling the insects and keeping the population of midge below the threshold level. Use resistant varieties if they are available in the region. If resistant varieties are not available, select varieties that flower early or all flower at the same time.

Timing of planting is also an important measure for reducing the damage. Uniform planting (i.e. planting on the same day and at the same depth) to ensure even flowering, and planting early, will reduce exposure to high infestations and reduce the damage caused by the sorghum midge.

Remove alternative hosts, such as wild sorghum, Johnson grass and Sudan grass, from in and around the field to prevent midge population build-up early in the season.

It is reported that midge populations are higher in fields with a low plant density. Planting at a high density reduces the number of insects per plant or area and can reduce damage.

Damage can also be reduced by intercropping sorghum with a legume.

Rotate sorghum with non-host crops or leave the field fallow the next season to break the build-up of the pest in the field.

Destroy crop residues after harvest to prevent the pest from carrying over to the following season.

Control – what to do after signs are seen

Cultural approaches: Chaffy spikelets should be removed and destroyed to stop the spread of the pest.

There are four groups of parasites that are natural enemies of the sorghum midge: the *Eupelmus, Eupelmidae, Tetrastichus* and *Aprostocetus* families – all are small black parasitic wasps. Efforts should be made to conserve habitat (e.g. flowering plants on the borders of field) to build-up populations of these natural enemies.

Chemical approaches: In the field, chemical control of the midge can be difficult because the larvae, pupae and eggs remain protected inside the spikelets. Pesticide use has to be carefully timed to occur when the adults emerge in the morning during flowering or else it will be ineffective.

After harvest, sorghum grains can be fumigated with phosphine to kill the larvae in the spikelets. This will reduce the chance of the pest spreading to new areas.

CAUSE

Stenodiplosis sorghicola is also classified as *Contarinia sorghicola*. It is commonly known as the sorghum midge, but is also known as dura gall midge and jola earhead fly. It was formerly classified as *Allocontarinia sorghicola* Solinas, *Contarinia andropogonis* Felt and *Contarinia palposa* Blanchard.

Sorghum is the main host, but wild varieties of sorghum (*Sorghum arundinaceum* and *Sorghum dochna*) as well as Johnson grass (*Sorghum halepense*) and Sudan grass (*Sorghum sudanense*) are also hosts. The symptoms caused by the midge are sometimes confused with poor seed setting, bad weather, or other insects such as the head bug (*Calocoris angustatus*).

The adults emerge from diapause (a resting period) in the morning and mate within an hour. The females lay 1 to 5 eggs in each spikelet, with each female laying a total of 50-100 eggs in their 1 day lifespan. The eggs hatch 2 or 3 days after being laid and the larvae begin feeding on the young soft ovary of the developing grain. The larvae continue to feed on the grain for 10-15 days, after which they pupate inside the grain for 3 to 5 days before emerging from the flower as an adult to start the cycle over again. The total life cycle is 15-20 days. Following harvest, larvae that are still in the grain enter into diapause where they can remain resting for up to 3 years. When the temperatures and humidity rise, usually triggered by the beginning of the rain season, the adults come out of diapause to emerge from the grain and mate.

IMPACT

Sorghum midge is considered to be the most important pest of sorghum in Africa, destroying 10-15% of the sorghum crop each year. It is reported that one adult midge can destroy 1.4 grams of grain. Where susceptible varieties are used, high populations can completely destroy the crop.

DISTRIBUTION

Sorghum midge has been recorded in almost all sorghum producing regions of the world. It is present in Africa, Asia, the Americas, the Pacific Islands, Australia and Europe. It is typically found in tropical and subtropical areas, and is more prevalent during the rainy season. The midge is spread between countries and regions by the transport of sorghum grain that contains larvae.

FURTHER READING

Plantwise Knowledge Bank www.plantwise.org/knowledgebank

Studebaker, G., Lorenz, G., and S. Akin. Grain Sorghum Insect Control. University of Arkansas, Cooperative Extension, FSA 2066. http://www.uaex.edu/publications/pdf/FSA-2066.pdf

Sorghum stem borers

Busseola fusca, Chilo partellus, Sesamia calamistis

Photo: Georg Goergen/IITA Insect Museum, Cotonou, Benin

African maize stalk borer moth.

Photo: Georg Goergen/IITA Insect Museum, Cotonou, Benin

Spotted stem borer moth.

SUMMARY: Stem borers are major pests of sorghum throughout Africa. The stem borers tunnel into the stem of the plant feeding on the internal tissues and causing the plant to weaken. The pest can be controlled through a combination of cultural practices, most notably intercropping and the 'push-pull' system. Pesticides can also be effective, but must be applied in the early crop stage before the larvae bore into the stem.

KEY SIGNS

There are three types of stem borers that attack sorghum: the spotted stem borer, the African maize stalk borer (also called the sorghum stem borer) and the African pink stem borer. Stem borer adult moths mate and the females deposit eggs on the underside of leaves near the funnels. The young larvae feed on leaf funnels leaving holes, and also the growing points preventing flowering. The older larvae bore into the stems of the sorghum, weakening them and causing chaffy heads. The central shoot withers and dries (known as 'dead heart'). The pink stem borers do not usually feed on the young leaves, so are less likely to cause the holes and scars symptoms that are typically associated with the spotted stem borer and African maize stalk borer.

The African maize stalk borer eggs are round, flat on top, creamy-yellow in colour and about 1 mm diameter. The larvae have no distinct features, lacking notable hairs or markings. The larvae are creamy-white in colour often with a grey or sometimes pink tint, the head is dark brown, and they grow up to 40 mm long. Pupae are up to 25 mm long, and shiny yellow-brown to dark brown in colour. The adult moths have a wing span of about 25-33 mm; the forewings are light to dark brown with dark patterns and the hind wings are light to greyish-brown. There is some variation in the colouring depending on location and season.

The spotted stem borer larvae are creamy white to yellowish-brown with dark conspicuous spots and four purple stripes lengthwise across the back. The pupae are shiny, light yellow-brown to dark red-brown and 15 mm long. The adult moths are 7-17 mm long with a wingspan of 20-25 mm. The forewings are light yellow-brown with darker horizontal patterns, and the hind wings are white. The eggs are creamy-white, scale-like and laid in overlapping batches on the underside of leaves near the midrib.

The African pink stem borer eggs are creamy-white when laid, but get darker as they develop. The larvae are smooth and shiny, creamy-white with a distinctive pink colouring, a brown head and are 30-40 mm long when mature. The pupae are yellowish-brown and 18 mm long. The adult moths are slightly smaller than the other two species of stem borers. They have yellowish-brown forewings, white hindwings, with a wingspan of 20-30 mm.

Monitor fields to look for symptoms of stem borers: feeding marks on the funnel leaves, 'dead hearts' and holes in the stems. Cut open the stems of a few selected plants that show symptoms to look for larvae and pupae.

MANAGEMENT

Prevention – what to do before signs are seen

Cultural approaches: Plant early to avoid a serious infestation of stem borers.

Applying nitrogen, either a commercial product or manure or compost, enhances the crops tolerance to an attack.

Implement a 'push-pull' system in which *Desmodium*, a repellent plant, and Napier grass, a trap crop, are intercropped with maize to lure the insect away from the maize.

Intercropping with non-host plants, such as cowpeas or cassava, will also reduce the damage. Adult moths will lay eggs on the non-host plants, but the larvae are unable to feed on them and will die.

Destroy crop residues after harvest to reduce populations and limit the pest the following season. Stems should be burned, fed to livestock or dried on the ground under full exposure of the sun's heat.

Rotate sorghum with a non-host plant, such as a legume, to prevent the build-up of the pest in the field and to increase the nitrogen in the soil which will make the next sorghum crop hardier and less susceptible to an attack.

Control – what to do after signs are seen

Cultural approaches: Two parasitic wasps, *Cotesia flavipes* and *Xanthopimpla stemmator*, attack the spotted stem borer. *C. flavipes* attacks the larvae while they are feeding in the stem, and *X. stemmator* attacks the pupae. Both parasites have been released in several countries. In coastal Kenya, studies have shown that *C. flavipes* has reduced the populations of spotted stem borers by 70%.

Several parasitic wasps attack the African pink stem borer, the most effective of which are *Cotesia sesamia, Descampsina sesamiae, Pediobius furvus, Sturmiopsis parasitica, Telenomus busseolae* and *T. isis. C. sesamia* attacks the larvae, *D. sesamiae* and *S. parasitica* attack both the larvae and pupae, *P. furvus* attacks the pupae and *T. busseolae* and *T. isis* are egg parasites. *C. sesamiae* has been released and established in Mauritius and Madagascar to control the African pink stem borer.

Ants and earwigs are also important natural enemies of stem borers and efforts should be made to maintain habitat to conserve parasitoids and predators.

Chemical approaches: Chemical control should only be used when the infestation is severe. Since sorghum is more densely planted compared to maize, applying granules to the leaf funnels is too labour intensive. Spraying a pesticide early over the plants might be more effective; however, once the larvae have bored into the stem of the plant, pesticides are no longer effective. Neem products can be effective against stem borers.

CAUSE

Busseola fusca, the African maize stalk borer is also commonly known as the maize stem borer and sorghum stalk/stem borer.

Chilo partellus is commonly known as the spotted stem borer.

Sesamia calamistis, the African pink stem borer, is also known as the African pink borer of sugarcane, Mauritius pink borer of sugarcane, pink stalk borer of sugarcane, southern pink borer of sugarcane. The pink stem borer is similar to the purple stem borer (*Sesamia inferens*).

IMPACT

The African maize stalk borer and spotted stem borer cause more damage to cereal crops than any other pests in sub-Saharan Africa. Stem borers can build up from year to year in fields where sorghum or maize is continually cropped, making the problem progressively worse and resulting in serious crop losses for sorghum and other grain crops. The African pink stem borer is not as important as the spotted stem borer or African maize stalk borer, but is still present throughout the region.

DISTRIBUTION

The occurrence of one species over another is dependent on location. The spotted stem borer is native to Asia, but spread to East Africa in the 1950s and then into Central Africa. The spotted stem borer is predominant in warmer, lower altitudes, or at high altitudes where rainfall is below 500 mm per year.

The African maize stalk borer is native to the region of sub-Saharan Africa and is present throughout the region from sea level to 2000 meters altitude.

The African pink stem borer is not as common as the other two species, but is present throughout sub-Saharan Africa.

Stem borers are often spread through the transport of dry stems, grains, and grasses that contain diapausing larvae. Primary hosts of stem borers are cereals, notably sorghum, maize and pearl millet, and sugarcane and several grass species, as well as wheat and rice.

FURTHER READING

Plantwise Knowledge Bank www.plantwise.org/knowledgebank

BioNET-EAFRINET http://keys.lucidcentral.org/keys/v3/eafrinet/index.htm

African rice gall midge

Orseolia oryzivora

Photo: IRRI

'Onion' or silver shoots are an unmistakeable symptom of damage by rice gall midge larvae.

SUMMARY: Larvae of the African rice gall midge feed on young shoots (tillers) of rice, causing them to stop growing and yields to therefore be reduced. 'Onion' or silver shoots are the most noticeable symptom of infestations and this is unique to the gall midge. A combination of natural control, through encouragement of parasitic wasps, and planting of resistant or tolerant varieties is the most effective method for managing this damaging pest.

KEY SIGNS

The rice gall midge is a flying insect pest, the larvae of which feed on the growing tips of new rice shoots. The adult midge is mosquito-like and small, up to 5 mm long. In the few days it is alive, the female can lay 200-400 eggs at or near the base of shoots.

The eggs produce larvae (maggots) which move between the leaf sheath and up the stem to the growing tip. There they burrow into young tillers (shoots), which then stop growing. A gall is formed, a swollen area where the larva transforms into a pupa. At first the galls are small, about 3 mm in diameter, and oval but they can extend up to 1.5 metres long. These galls are the distinctive 'onion' or silver shoots which are associated only with gall midge damage. The adult midge eventually emerges from these long, white hollow tubes about three weeks after the initial burrowing by the larva into the tiller.

The death of young tillers stimulates the production of more tillers, which often become infested. Attacks by the gall midge larvae at the early stages of rice plant growth cause stunting and bushiness. Gall midge larvae only feed on rice when it is tillering or producing new shoots. The infested tillers do not produce grain.

MANAGEMENT

Prevention – what to do before signs are seen

Cultural approaches: The use of resistant varieties together with preventative measures and natural control will help to reduce damage and increase yields.

Resistant varieties are available for African rice (*Oryza glaberrima*): TOG 7106, 7206 and 7442. For Asian rice (*Oryza sativa*), resistant varieties include: 'Cisadane' (Nigeria), BW 348-1 (Burkina Faso, Nigeria) and TOS 14519 (Gambia). Check if these are available locally. NERICA L-25 is moderately resistant to rice gall midge in Nigeria.

For prevention, neighbouring farmers should ideally plant at the same time to reduce the availability of new plants for emerging adult midges to infest and persist. Sow and plant early to avoid peak insect populations. However, these measures require, respectively, good coordination and cooperation by many farmers, and suitable conditions for early planting.

Be careful with fertilizer application. Too much nitrogen will increase shoot and leaf production and provide more food for the gall midge. High yielding varieties of rice, which respond well to fertilizer application, are also at risk.

There are several parasitic wasps which attack the rice gall midge. Paspalum grass grows naturally with rice: it is attacked by a different type of midge which does not attack rice but is an alternative host for the wasps. Conservation of Paspalum grass (and therefore more parasitic wasps) is encouraged.

The gall midge will survive on wild rice (*Oryzae longistaminata*) and be carried over to the next season in any remaining rice plants – either re-sprouting from cut plants (ratoons) or natural re-seeding (volunteers). Removal of these plants is encouraged.

Both of these approaches may be difficult to achieve on a large scale.

Chemical approaches: Insecticides are costly and unlikely to be effective.

CAUSE

The African rice gall midge is *Orseolia oryzivora*. The closely related Asian species, *Orseolia oryzae*, also attacks rice but is not found in Africa.

African rice gall midge is mainly a pest of rainfed and irrigated lowland rice. The insect prefers high humidity and in wetter years the risk of infestation is higher.

IMPACT

The gall midge only became a significant problem in the 1970s, when major losses occurred in Burkina Faso. Elsewhere, localized outbreaks were damaging but not seen as a major threat, for example in Zambia. In 1988 things changed dramatically with 40 to 85% of tillers infested in rice grown in the savannah zone of Nigeria, leading to major losses in yield. Around 50,000 hectares were affected, With further outbreaks and closer investigation of gall midge attacks since the Nigeria outbreak, observations in several countries indicate yield losses of between 25 and 80%.

DISTRIBUTION

In West Africa the gall midge is a major pest in Burkina Faso, Nigeria, Mali and Sierra Leone (where it is known locally as yabas leaf). The pest has also been recorded in many other countries: Senegal, The Gambia, Guinea Bissau, Guinea, Côte d'Ivoire, Ghana, Togo, Benin, Niger, Chad, Sudan (and presumably South Sudan, though this is not confirmed at the time of writing), Uganda, Tanzania, Malawi and Zambia.

FURTHER READING

Check the Africa Rice website for papers, extension leaflets and up to date information. The following are all available at www.africarice.org

Nwilene FE, Nwanze KF and Okhidievbie (2006). African rice gall midge: biology, ecology and control. Field guide and technical manual. Available from AfricaRice website.

Africa rice gall midge (no date). Reference 22.

Plantwise Knowledge Bank (www.plantwise.org): Africa rice gall midge: technical fact sheet.

Plantwise Knowledge Bank (www.plantwise.org)

Infonet-Biovision (www.infonet-biovision.org)

Rice Knowledge Bank (www.knowledgebank.irri.org)

For information on Paspalum go to: http://www.cabi.org/isc/datasheet/38955

Grey leaf spot of maize

Cercospora zeae-maydis & Cercospora zeina

Photo: Noah Phiri

Maize growing in Zambia showing symptoms of grey leaf spot.

Photo: Noah Phiri

Spots join together resulting in long grey streaks and a blight of the leaf.

SUMMARY: Grey leaf spot of maize has emerged as a yield-limiting disease throughout USA and southern Africa in the last 25 years. Although thought to be one species of fungus, *Cercospora zeae-maydis*, molecular tests have shown that another fungus, *C. zeina*, is dominant in eastern USA and southern Africa. Spores, surviving in the remains of previous crops, are rain-splashed onto lower leaves causing long spots, merging on susceptible varieties, to cause a blight. Stems are weakened and fall over (lodge) during epidemics. The disease can be managed by destroying plant debris after harvest, crop rotation, using more tolerant varieties and, where economically justifiable, by fungicides.

KEY SIGNS

Symptoms first appear on the lower leaves as pinpoint spots surrounded by yellow halos, best seen when held to the light. The spots expand parallel to the leaf veins and, on susceptible varieties, are light brown to grey, rectangular, up to 70 mm long and 4 mm wide. The spots may join together on the leaves to cause a blight. Spores form on the spots and the leaves begin to look silvery-grey.

Sheath and stalk infections may also occur, although they are not as clearly seen as those in the leaves. The infections can lead to rots, which result in the plants falling over.

MANAGEMENT

Prevention – what to do before signs are seen

Cultural approaches: Grey leaf spot is best managed by using resistant varieties and a number of hybrids are now available for Africa.

Choice of varieties is extremely important. There are no varieties which are totally resistant (immune to infection), but many are 'tolerant' where leaf spots are small, disease comes late and the impact is low. Check with local seed companies or retailers if open-pollinated varieties with grey leaf spot tolerance are available. Otherwise, consider the use of improved commercial hybrids, which have to be purchased each year. As an example, a recent study (2014) has shown that ZMS616 is the most popular variety with farmers in Zambia because it is suited to a range of environments, drought tolerant and disease resistant. Other varieties that are disease tolerant are SC407 and SC411.

If smallholders want to try a new variety, instead of planting traditional open-pollinated types, it is recommended that only small plots are planted at first, before scaling up only if the new variety is suitable.

If direct seeding (no-tillage or zero tillage) is the preferred method of cultivation, grow legume cover crops before planting the maize; suitable crops are lablab, velvet bean or sunhemp. Clear the legume just before planting the maize by making furrows without cultivating the land. At first weeding plant a short-term food legume cover crop such as beans, peas, mung bean or soybean as an intercrop to cover the soil. Note that continuous maize, and no tillage or

reduced tillage, are high-risk practices, creating conditions for disease development; this is because of the amount of maize debris that remains on the soil surface.

After harvest, if not using conservation tillage practices, plough in or collect and burn crop debris. This is very important as the spores on the leaves and stalks allow the fungus to survive from one season to another.

Control –what to do after signs are seen

Cultural approaches: Even if a tolerant variety is used, it is still important to use a 1-2-year rotation between crops of maize on the same land if grey leaf spot is established in the field. Note that grey leaf spot only infects maize, so other crops in the rotation will not become infected.

Chemical approaches: If fungicides are affordable and available, apply just before tassels (male flower) emerge. In South Africa mixtures of benzimidazoles and triazoles are used, and more recently strobilurins. These, though, are expensive and so unlikely to be economically viable except in large-scale commercial operations.

CAUSE

Grey leaf spot was first thought to be caused by the fungus, *Cercospora zeae-maydis*. Later, it was found that there was more than one species; there was *C. sorghi*, and two groups within *C. zeae-maydis*: Group I is the dominant type in the USA, and occurs elsewhere, and Group II occurs mostly in USA and Africa. Molecular studies have now found that Group II is a separate species, *C. zeina*, and that *C. zeae-maydis* does not occur in South Africa. Whether *C. zeae-maydis* is in other places in the continent needs to be resolved. There is even speculation that *C. zeina*, the African species, came from sorghum because of molecular similarity between that species and *C. sorghi*.

Grey leaf spot is a serious fungal disease of maize. It first became widespread in the early 1990s. The severity of the disease depends on three factors: the amount of fungus surviving on remains from previous crops, the tolerance/resistance level of the particular maize variety and, most importantly, weather conditions.

The fungus can survive for more than a year between crops on maize debris left above and on the soil surface. Spores are then splashed by rain to the lower maize leaves. There is no evidence that the fungus is spread either in or on the seeds, i.e. it is not seed-borne.

Once the lower leaves are infected, rain-splash spreads the spores to higher leaves and to leaves on plants nearby. Spread further afield occurs via spores carried on the wind. The time from infection to spore production varies from 15-30 days depending on the disease tolerance of the variety. The disease is often seen following periods of warm (25-32°C) overcast days with heavy dews.

IMPACT

The disease has become one of the most serious maize diseases worldwide. It is of particular concern in the USA and Africa. In the USA, the disease has increased because of no-tillage practices and the susceptibility of hybrid maize. In some states of the USA corn belt, maize yields in the 1990s were said to have fallen 20-40% due to grey leaf spot. In Africa, economic losses have been reported from South Africa, but have yet to be detailed in East, West and Central Africa where it is also present. Impact depends on when the eight or nine leaves above the ear become infected, and the amount of disease that develops on them, as these determine 70-90% of the yield: the earlier the infection sets in, the greater the yield loss. If the disease does not affect these top leaves until 6 weeks after tasselling, then yield losses will be small. Yield loss also depends on the length of the crop. Hybrid maize in South Africa and other countries of Africa are particularly vulnerable to this disease as they are long-season types, giving the fungus more time to damage them.

DISTRIBUTION

The disease is present in North and South America, Central America and the Caribbean, with a limited distribution in Asia. In Africa, it is widespread in Ethiopia, Kenya, Malawi, Mozambique, South Africa, Swaziland and Zimbabwe. It is also present in Cameroon, Nigeria and Tanzania, and locally present in Uganda and Zambia. The distribution of the two species, *Cercospora zeae-maydis* and *C. zeina* differs. *Cercospora zeae-maydis* is present in North, South and Central America, the Caribbean and Africa (but not South Africa); *C. zeina* is present in China, Brazil, the USA eastern corn belt and throughout Africa.

FURTHER READING AND OTHER RESOURCES

Cercospora zeae-maydis. CABI Crop Protection Compendium. (http://bit.ly/1UOLzv2).

Crous PW, Groenewald JZ, Groenewald M. Caldwell P, Braun U, Harrington TC (2006) Species of Cercospora associated with grey leaf spot of maize. Studies in Mycology 55:189–197. (http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2104713/).

Stromberg EL (2009) Gray leaf spot disease of corn. Virginia Cooperative Extension. Virginia Tech. (http://bit.ly/1IRTgMd).

Wang J, Levy M, Dunkle LD (1998) Sibling Species of Cercospora Associated with Gray Leaf Spot of Maize. Phytopathology 88(12):1269-1275. (http://apsjournals.apsnet.org/doi/abs/10.1094/PHYTO.1998.88.12.1269).

Ward JMJ, Stromberg EL, Nowell DC, Nutter FWJr (1999) Gray leaf spot: A disease of global importance in maize production. Plant Disease 83:884-895.

Wise K (2010) Gray leaf spot. Purdue Extension. (https://www.extension.purdue.edu/extmedia/bp/BP-56-W.pdf).

Maize ear rots and mycotoxins

Fusarium and Aspergillus species

Photo: Eric Boa, CABI, CC BY 4.0

Unidentified fungal mould on ear rot, a potential source of mycotoxins.

Photo: Eric Boa, CABI, CC BY 4.0

Drying of cobs and rejection of damaged cobs both help to reduce mycotoxin contamination.

SUMMARY: Several fungi occur on maize and produce poisonous chemicals which contaminate food and feed. Known collectively as mycotoxins, these poisons have serious effects on human and animal health. *Aspergillus* is said to be the most important mycotoxin producer in Africa, though other fungi such as *Fusarium* are also involved. Both groups of fungi grow on dead and decaying plant material and cause rotting of maize ears in the field. They produce powdery masses of spores on cobs, pre- and post-harvest, but can also be present without any mould production. Ear rots caused by mycotoxin-producing fungi are more common when maize is stressed or growing poorly. The most effective control of these fungi combines timely harvesting of maize and drying of cobs before storing. Aflasafe™, a new biological control option against *Aspergillus*, is applied in the field before flowering of maize and shows considerable promise in reducing contamination of ears prior to harvest and later accumulation of mycotoxins in stored products.

KEY SIGNS

Aspergillus species typically produce a yellow-green mould on maize ears, although sometimes it is brown to black. The mould is similar in appearance to that found on decaying food. The powdery appearance is due to the production of millions of spores. *Aspergillus* causes little damage to living plants and often follows insect damage or other wounding. The fungus persists on dead and decaying material and in the soil and is an ever-present risk.

Fusarium species also persist on dead plant material and in the soil. They cause an ear rot with mouldy growth occurring between the ear and the husk (enclosing leaves). The symptoms usually start at the tip of the ear and advance downwards. Sometimes the whole ear becomes rotten. Infections can also follow physical damage, such as bird feeding. The mould is red to pink and more cottony or woolly when compared to *Aspergillus*.

There are other fungi which cause ear rots but do not produce mycotoxins. Both *Fusarium* and *Aspergillus* may be present on maize without producing moulds or other visible symptoms. The fungi are able to rapidly grow and produce mycotoxins when ears are harvested too early and become wet in storage.

MANAGEMENT

Prevention – what to do before signs are seen

Cultural approaches: Use clean seed for planting. If saving own seed, discard mouldy ears. Varieties of maize with a tighter husk will help to limit bird and insect feeding damage on ears, which allows *Aspergillus* and *Fusarium* to become established. Control of insects attacking ears will reduce fungal ear infections.

The general view is that, despite many years of trying to produce 'resistant' varieties, this approach has had little success and is unlikely to have a major impact on reducing mycotoxins. Maintaining good soil fertility and avoiding water stress will strengthen the ability of maize plants to limit infections of *Aspergillus* and *Fusarium*.

Attention is now focused on a new biocontrol option against *Aspergillus*, known as Aflasafe™, applied when maize is in the field. Aflasafe does not control *Fusarium*.

Aflasafe™ consists of sterile sorghum grains carrying a non-mycotoxin producing strain of *Aspergillus*. The grains are scattered throughout the maize field to encourage the displacement and therefore exclusion of mycotoxin-producing strains. Aflasafe™ is available in 5 kg and 10 kg plastic tubs from selected sources. Commercial production and distribution is being developed. Before applying Aflasafe™, weed and ridge the plot and apply fertilizer. It is important to avoid walking through a field after application since the sorghum grains need to rest on the soil. Broadcast Aflasafe™ two to three weeks before flowering to give the fungus enough time to grow and sporulate.

A tub of 10kg is sufficient for one hectare. Apply after rains, or when expected, or when the soil is wet. This will help the non-mycotoxin production, friendly fungus to grow quickly. Check the scattered grains about a week after applying to see if they are covered with a green powder.

Divide the Aflasafe™ into equal portions to enable even application to the maize field. A half kg portion is enough for an area 10 metres by 50 metres (500 m^2). Chickens, birds and wildlife may pick up the sorghum grains but will not be affected. Ants may carry the grains below ground but later the grains will be returned to the soil surface.

There is a high risk of contamination with *Aspergillus* and/or *Fusarium* on harvested cobs in all maize growing areas. Mouldy ears should be discarded after harvest. Remove plant debris and practice crop rotation in order to reduce fungal inoculum in the field.

When harvesting maize, avoid damaging the grains, to prevent invasion by fungi. Do not harvest and store rotten maize cobs. Reject those with a mouldy appearance. This may be difficult to accept for small-scale, poor farmers who need to maximize harvests. A partial solution is to remove at least the mouldy part of the ear before storage.

Maize grain should be dried on raised platforms or over a plastic sheet or tarpaulin. Do not dry directly on the ground. Store the grain in cool and dry conditions. Promoting these practices amongst farmers will help reduce contamination with mycotoxins and make food and feed safer to eat.

Although this fact sheet focuses on maize and ear rots, mycotoxins are associated with other important crops, such as cassava. Choice of planting material, reducing stress on crops, timely harvesting and reducing the wetness of stored products to control fungal growth apply equally to other crops affected by mycotoxins.

CAUSE

There are two important *Aspergillus* mycotoxin producing species: *Aspergillus flavus* (a yellow-green, powdery mould) and *Aspergillus parasiticus* (a dark green, powdery mould). *Aspergillus niger* has a black powdery mould and is a common ear fungus on maize in fields but it is not a mycotoxin producer. *Fusarium moniliforme* (pink, cottony) is said to be the most common pathogen of maize ears around the world and also an important mycotoxin producer. *Fusarium* is generally more of a problem in temperate maize-growing areas.

Mycotoxin is a generic term. Specific types are associated with different fungi, such as aflatoxins and *Aspergillus*, and fumonisins from *Fusarium*. Species of both fungi also produce other mycotoxins, such as ochratoxins.

IMPACT

The damage caused by *Aspergillus* and *Fusarium* to standing crops is small compared to the adverse health effects on humans and animals due to ingestion of mycotoxins. The effects on young children and during pregnancy are especially harmful. Chickens are highly susceptible to mycotoxins.

Mycotoxins cannot be seen or tasted. They have no smell, taste or colour and they can only be detected by chemical analysis. Their presence is often only revealed by wide-ranging symptoms shown by people and animals that have eaten contaminated maize products, particularly over a long period of time. Long term (chronic) exposure to mycotoxins leads to reduced immunity to disease, kidney and liver damage, and stunting of children.

Reliable estimates suggest that 25% of the world's food is contaminated with mycotoxins and that 2.5 billion people are regularly exposed to them. A high proportion of soils (40-80%) contain aflatoxin-producing strains of *Aspergillus*. Financial losses are substantial. A reduction in groundnut exports from Africa (to meet EU mycotoxin testing requirements) meant an annual loss of US$670 million to exporting countries.

DISTRIBUTION

Aspergillus species producing mycotoxins occur in all areas in Africa where maize is grown.

FURTHER READING AND OTHER RESOURCES

Crop Protection Compendium (www.cabi.org/cpc) and Plantwise (www.plantwise.org).

Mycotoxin fact sheets: http://bit.ly/1O3OVWh 'Tackling killer aflatoxins in African food crops' and other information about Aflasafe™: www.aflasafe.com

Maize lethal necrosis disease

Multiple virus infections

Photo: Eric Boa, CABI, CC BY 4.0

Once the disease enters a field there is little that can be done to prevent total loss of the crop.

Photo: Eric Boa, CABI, CC BY 4.0

Advanced symptoms include browning from the edge of the leaves, which does not occur in maize streak virus.

SUMMARY: Maize lethal necrosis disease (MLND) is a new viral disease for Africa. It has caused great concern because plants are killed and little or no grain is produced. The main thrust of the current management strategy is to prevent the introduction of the disease through sound surveillance and early destruction of diseased plants. Promising resistance exists to maize chlorotic mottle virus (MCMV), the main virus associated with the disease, but further work is needed before recommendations backed by scientific evidence can be given on what varieties to grow.

KEY SIGNS

The first symptoms are a scattering of small yellow areas (mottling) on leaves. These start to merge and the leaf becomes paler before the edges go brown and dry inwards. In some plants the young leaves die before they have expanded, producing a 'dead heart'. The first stages of mottling are similar to maize streak virus, but thereafter the two diseases are quite distinct: with maize streak virus the streaks do not merge and there is no marginal browning and drying up.

Maize lethal necrosis disease (MLND) kills plants. Plants become stunted and grain production, if it occurs at all, is poor: a consequence of distorted and shrivelled ears. All stages of plant growth are affected and the earlier the plant becomes infected, the greater the damage.

MANAGEMENT

Prevention – what to do before signs are seen

Cultural approaches: Take early action to avoid introducing the disease to new areas by using certified seed and varieties recommended by local extension services and national agricultural bodies. Although promising resistance has been identified in Kenyan trials, the maize types are 'pre-commercial hybrids' and therefore not yet available to the public.

Maize planted less than 500 metres from an infected area is more likely to get the disease. Other advice includes removing weeds and burning grasses around the field, both potential sources of the insect vectors.

Avoid walking through established fields since the virus can be physically transmitted on clothes.

Rotate maize crops with non-cereals to reduce the virus inoculum of both MCMV and sugarcane mosaic virus (SCMV), the other virus needed for development of MLND.

Chemical approaches: Reduction of vector populations through insecticide application is unlikely to have any impact on the disease unless applied consistently and over large areas. These conditions are difficult to achieve in dense patchworks of smallholder plantings.

Control – what to do after signs are seen

Cultural approaches: Early removal of infected plants showing first symptoms may reduce the local spread of MLND but is unlikely to constrain its longer term spread.

Collecting and burning of plant remains to reduce the spread of the disease must be done on a large scale to be effective.

CAUSE

First discovered in 2011 in Kenya, the disease has spread rapidly to neighbouring countries.

MLND is caused by a combination of two viruses, one of which is always maize chlorotic mottle virus (MCMV). This by itself will cause only minor damage on maize, but when another potyvirus of cereals, such as SCMV, wheat streak mosaic virus (WSMV) or maize dwarf mosaic virus (MDMV) is present, the result is MLND. In Kenya only SCMV has been found in co-infections, but other potyviruses are implicated elsewhere.

Much is already known about MLND from research done in the United States. MCMV, for example, is transmitted by thrips, rootworms (*Diabrotica* spp.), leaf beetles and in seed. Different insects may be responsible in Kenya, where research is only beginning. Little is known about the importance of virus movement in seeds. It is important not to make too many assumptions about MLND in Africa generally while maximizing the use of existing knowledge of the disease from other countries.

MCMV occurs widely in maize in Africa but is less damaging on its own, compared to co-infections with another 'enabling' virus, such as SCMV, which result in MLND. There is resistance to MCMV and/or SCMV in maize but of 119 commercial maize varieties for sale in Kenya, 117 were susceptible to MLND.

IMPACT

MLND destroys whole fields of maize. The stunting and outright death of plants, together with little or no grain production, makes MLND a devastating disease whose long-term impact on maize production in other countries has been significant and poses a major threat to countries in East Africa and beyond.

DISTRIBUTION

MLND has been confirmed in Kenya, Tanzania, Uganda, Rwanda, South Sudan, Malawi and Mozambique. Unconfirmed reports suggest that the disease is also in DR Congo and Ethiopia.

FURTHER READING

Check www.cimmyt.org and www.kari.org for regular updates on MLND. Helpful leaflets summarise current progress:

www.cimmyt.org/en/component/docman/doc_download/37-update-promising-cimmyt-maize-inbreds-and-pre-commercial-hybrids-identified-against-maize-lethal-necrosis-mln-in-eastern-africa (January 2013)

www.cimmyt.org/en/where-we-work/africa/item/maize-lethal-necrosis-mln-disease-in-kenya-and-tanzania-facts-and-actions (January 2013)

Check maize doctor for up to date information on management:

http://maizedoctor.cimmyt.org/index.php/home/22-welcome-to-maize-doctor

The Plantwise Knowledge Bank contains factsheets on MLND (www.plantwise.org).

The Crop Protection Compendium has information on maize viruses and pests and diseases (www.cabi.org/cpc).

Pearl millet downy mildew

Sclerospora graminicola

Photo: Rikhab Raj-Bhansali, CAZRI, CC BY-NC 3.0 US, www.bugwood.org

Deformed flower head of pearl millet caused by internal infection by downy mildew.

SUMMARY: Downy mildew of pearl millet is caused by an oomycete or water mould, *Sclerospora graminicola*. It is a serious disease in India and Africa with losses of at least 30% reported on susceptible varieties. Infection comes from the seed or from the soil. Leaves become yellow, flowers become leaf-like and plants are stunted. Two types of spores form: sporangia on the leaves, which spread the mildew to plants nearby, and oospores, thick walled spores, on all plant parts. These survive in the soil and are spread long distances in soil blown by the wind. Management is dependent on hybrids bred for resistance, and treatment of the seeds with fungicides, most commonly metalaxyl.

KEY SIGNS

Symptoms can occur on the first leaf that forms. Generally, however, they are first seen on the second leaf and then on all those that develop afterwards. The base of the leaves turn yellow and, as new leaves develop, the yellow area becomes larger. In some cases there is a 'half-leaf' symptom of yellow at the base and green above. Severe infection can cause death of the plants; most often the plants are stunted and either lack flowers, or parts of the flower head become leaf-like. This symptom is known as 'green ear', a distinctive feature of this disease.

If humidity is high (>95%), with moderate temperatures (20-25°C), spores (called sporangia) form on the yellow areas, particularly on the lower surface, giving the leaves a 'downy' appearance. The resistant spores (the oospores) develop when separate downy mildew infections grow together and form a sexual stage.

MANAGEMENT

Prevention – what to do before signs are seen

Cultural approaches: The use of resistant varieties developed by ICRISAT[1] and seed treatment are the two main ways of managing this disease.

The first releases were open-pollinated varieties and have been adopted by farmers in nine countries in West Africa. Examples are: SOSAT-C88, GB 8735 and ICMV-IS 89305. In eastern and southern Africa, ICMV 88908 (Okashana 1 and 2) has been released and adopted; for instance, it is used in over 50% of the production area in Namibia. Check whether tolerant varieties are available locally.

Chemical approaches: Seed from all sources, purchased or saved by farmers, should be treated with the fungicide metalaxyl (2g active ingredient/kg). Seed companies usually do this before seed is sold. Metalaxyl will eliminate seed-borne downy mildew infections for approximately 30 days after sowing. Farmers who save their own seed should dust with metalaxyl, or alternatively with captan, another fungicide – always following the manufacturers' instructions on the pack.

[1]International Crops Research Institute for the Semi-Arid Tropics

Commercial producers growing pearl millet for seed should spray with metalaxyl, or metalaxyl plus mancozeb, at 25-30 days. The cost of this treatment is likely to be too high for smallholders. Strobilurin fungicides are also effective against this disease.

Control – what to do after signs are seen

Cultural approaches: During crop growth, especially during the first month after sowing, crops should be monitored carefully. Any plants with symptoms should be removed promptly and burnt.

CAUSE

Pearl millet downy mildew is one of the most economically damaging diseases of this crop. The mildew is not a fungus, although there are many similarities in appearance, life cycle, and symptoms. It is a water mould, also known as an oomycete, closely related to brown algae. Downy mildew of pearl millet is caused by the oomycete, *Sclerospora graminicola.*

The presence of spores (sporangia), which produce smaller internal swimming spores with two whip-like threads, and the presence of cellulose not chitin in their cell walls, sets them apart from fungi. There are different races (strains) of the oomycete. In India, the range of pearl millet varieties infected by downy mildew differs, as does severity of infection. Also, differences exist between African and Indian populations of pearl millet downy mildew.

A prominent feature of the disease is that the primary sources of infection are seed and soil. Seed becomes infected internally through the flowers (the stigma) and resistant spores (called oospores) develop on the seed coat. The soil contains oospores produced from infected plants of previous crops. Seedlings are infected from these sources.

There are reports of up to 8% infection in pearl millet seed.

Survival of the downy mildew occurs as growth inside the seed and as oospores. Oospores can survive from a few months up to 10 years, depending on variety, environment and interaction with other microorganisms. The number of oospores present in soil is related to number of plants with disease at three months. By contrast, alternative hosts are not considered important in the spread or survival of this mildew, although it has been recorded on *Setaria italica* (foxtail millet) and wild grasses.

IMPACT

Yield losses in pearl millet attributed to downy mildew vary with location, variety and season. On susceptible varieties, a disease incidence of 60% equates to about a 30% loss in yield compared to a healthy crop. There are examples from India of severe effects on yield of certain varieties from epidemics of this downy mildew and there is a report of 60% loss from Mozambique. Incidence is much less in West Africa where it is between 0 and 50%, with the exception of Nigeria where grain yield losses range from 50-70%. Losses have also been reported from China. However, the use of resistant varieties and seed dressing with metalaxyl have significantly reduced the incidence of downy mildew. In Mali, for instance, these technologies have helped increase millet yield and, consequently, farmers' incomes.

DISTRIBUTION

The downy mildew is reported in more than 50 countries in the temperate and tropical areas of Africa, Asia, North and South America, Europe and Oceania. In Africa it is found in all pearl millet growing countries, including Benin, Burkina Faso, Cameroon, Chad, Egypt, Ghana, Ivory Coast, Kenya, Malawi, Mali, Mozambique, Niger, Nigeria, Senegal, Sierra Leone, South Africa, Sudan, Tanzania, Togo, Uganda, Zambia and Zimbabwe.

FURTHER READING

Downy mildew of pearl millet (Sclerospora graminicola). Plantwise Knowledge Bank. http://www.plantwise.org/KnowledgeBank/Datasheet.aspx?dsid=49159).

Kenganal M. Downy mildew. APS. (http://www.apsnet.org/publications/imageresources/Pages/FI0038.aspx).

Sclerospora graminicola. CABI Crop Protection Compendium. (http://www.cabi.org.ezproxy.library.uq.edu.au/cpc/datasheet/49159).

Sharma HC (2006) Integrated pest management research at ICRISAT: present status and future priorities. Patancheru 502 324, Andhra Pradesh, India: International Crops Research Institute for the Semi-Arid Tropics. 48 pp.

Singh SD (1995) Downy mildew of pearl millet. International Crops Research Institute for the Semi-Arid Tropics, Patancheru, Andhra Pradesh, India. (http://www.apsnet.org/publications/plantdisease/backissues/Documents/1995Articles/PlantDisease79n06_545.pdf).

Rust of pearl millet

Puccinia substriata

Photo: Vivek Gupta

Reddish-brown (rust coloured) spots, with yellow halos, producing masses of spores which spread between pearl millet plants.

SUMMARY: Rust of pearl millet is caused by the fungus *Puccinia substriata*. It causes losses in grain yield, especially if infection is early, and also reduces the quality of fodder for livestock. The disease is present in USA, Asia and is widespread in semi-arid tropical and sub-tropical Africa. Several asexual spore stages of the rust occur on pearl millet and wild grasses, with the sexual stage on eggplant (aubergine). Spores carried on the wind spread the rust, and survival is in the soil, on debris, volunteer pearl millet and alternative hosts. Management relies on crop rotation, weeding to remove volunteer plants and weeds, tolerant varieties and destruction of crop remains after harvest. Fungicides are not economically viable unless crops are grown for commercial purposes.

KEY SIGNS

On pearl millet, small green to yellow slightly raised spots develop over the leaf surface, with more on the topside than the underside. As the spots grow larger, up to 2 mm, and rise further from the surface of the leaf, they develop into reddish-orange pustules, round to elliptical, with yellow halos. Masses of spores (urediniospores) form in the pustules and spread the rust within and between crops. Later, as the pustules age they become darker as another spore type (teliospores) appears; the leaves start to die from the tips towards the base. If the disease is severe, spots occur on the stems and the plants fall over (lodge). Note: the teliospores have thick walls and this helps them survive in the soil.

MANAGEMENT

Prevention – what to do before signs are seen

Cultural approaches: Before planting, check if resistant varieties are available locally. ICRISAT[1] began breeding varieties for resistance against this rust in the 1970s. High yielding, early maturing hybrids for most rainfed areas (600-800 mm) are available for sub-Saharan Africa, building on Indian successes. Open-pollinated varieties are also being developed. Consult seed suppliers for varietal characteristics. Note, the resistance of many hybrids is based on single genes; there is always a possibility that the rust will overcome the resistance. Varieties with tolerance (durable resistance) do not appear to be available for this rust, although they have been made available in the USA.

Plant early, as soon as weather conditions allow; this will minimize yield and grain quality losses as the crop will have matured before maximum disease development. Do not plant pearl millet continuously on the same land, but rotate with other crops such as sorghum and legumes.

During crop growth, remove weeds, grasses in particular; do not plant near eggplant or its weed relatives, e.g. *Solanum torvum* (turkey berry). After harvest, collect and bury crop residues.

Chemical approaches: Fungicides are not recommended for smallholders, as they are not likely to be economical. If pearl millet crops are grown commercially use copper compounds, chlorothalonil, sulphur or mancozeb.

[1]International Crops Research Institute for the Semi-Arid Tropics

CAUSE

The rust is caused by *Puccinia substriata*, but in recent years it has been given a number of different names, for instance, *P. penniseti, P. substriata* var. *Penicillariae* and *P. substriatavar. substriata.* Other reports using DNA comparisons regard *P. substriata* var. *penicillariae* the same as *P.substriata* var. *indica*.

Infection of *Solanum* hosts has been reported from Brazil, India and the USA. When the rust was first found on eggplant (*Solanum melongena*) in 1915, it was given the name *Aecidium tubulosum*. It was only later that it was recognised as being the pearl millet rust, *P. substriata*. The rust also occurs on *S. aethiopicum* (Ethiopean eggplant) and *S. torvum* (turkey berry). Likewise, several grasses are hosts, e.g. species of *Digitaria, Echinochloa, Paspalum*, *Pennisetum* and *Setaria*. Several different races of the fungus have been reported from the USA.

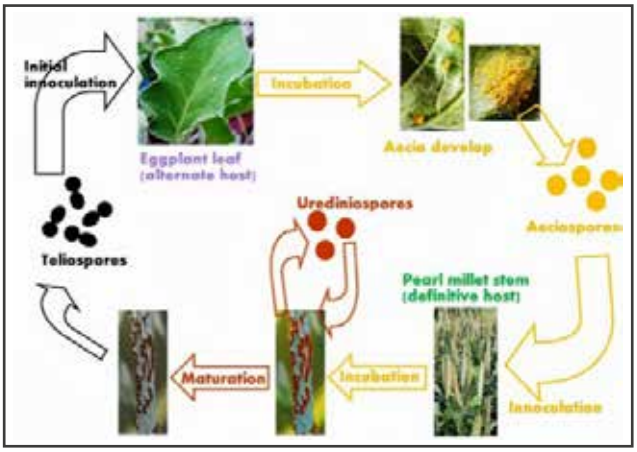


Figure 1: Image by kind permission of Roots 'n' shoots http://rsandss.blogspot.com/

The rust fungus that causes this disease has a complex life cycle with many different stages on two quite different hosts. Most stages occur on pearl millet and related grasses, and some on eggplant and its wild relatives. The disease reproduces sexually on eggplant. The life cycle is shown in Figure 1, above.

The life cycle of *P. substriata* has not been studied in detail, but it is likely that the teliospores germinate, producing basidiospores which infect eggplant, or wild *Solanum* species. Here, mating occurs between different strains, the result of which is another type of pustule which is yellow at first becoming reddish-brown, up to 15 mm across. The spores (aeciospores) that develop in these cup-shaped protruding structures on the underside of the leaf infect pearl millet. This completes the life cycle.

Long distance spread is on the wind. Because some stages have thick dark walls, they can travel long distances, high in the atmosphere.

Survival between crops of pearl millet occurs in a number of ways: as dormant spores (teliospores) in the soil; as spores on infected crop debris; or as infections on volunteer (self-seeded plants) pearl millet, on wild grasses, and on *Solanum* species, many of which are perennial weeds.

The disease is favoured by cool nights (15-20°C) and warm days (25-34°C) as this promotes abundant dew on the foliage, thus assisting the urediniospores to germinate and infect.

IMPACT

Severe losses occur if infection takes place before flowering. Epidemics of the disease were first recorded from the USA and since then they have occurred frequently around the world with losses of over 75% in grain production and fodder quality. In India, for instance, the disease has become widespread due to large-scale seed production schemes and overlapping crops. Trials in the southeast USA in 1993 and 1994 with a susceptible variety showed that grain losses began when rust severity scores reached 50% at harvest: scores above 50% resulted in major losses. Recently, there have been reports of serious outbreaks in Brazil after cultivation became more widespread for cattle feed using minimum tillage (no ploughing) systems. The disease was so serious that it prevented the increased expansion of the crop.

DISTRIBUTION

The rust occurs in North, South and Central America, the Caribbean, Asia and Africa. In Africa it occurs in Burkina Faso, Chad, Congo, Ethiopia, Ghana, Guinea, Ivory Coast, Kenya, Malawi, Mozambique, Niger, Nigeria, Senegal, Sierra Leone, South Africa, Sudan, Tanzania, Uganda, Zambia and Zimbabwe.

FURTHER READING

Fungal diseases of pearl millet. Rust: Puccinia substriata Ell. & Barth. indica Ramachar&Cumm. USDA Forage and turf grass research. USDA ARS. (http://www.tifton.uga.edu/fat/fungaldiseasesPM.htm - Rust).

Puccinia substriata var. substriata. CABI Crop Protection Compendium.http://www.cabi.org.ezproxy.library.uq.edu.au/cpc/datasheet/107870).

Roots 'n' shoots. (http://rsandss.blogspot.com.au/2013_10_01_archive.html).

Thakur RP, Sharma R, Rao VP (2011) Screening techniques for pearl millet diseases. Information Bulletin No. 89.Patancheru 502 324, Andhra Pradesh, India. International Crops Research Institute for the Semi-Arid Tropics. 56 pp. (http://oar.icrisat.org/5527/1/Information_Bulletin_no_89.pdf).

Yun HY. Systematic Mycology and Microbiology Laboratory, ARS, USDA. Invasive Fungi. Eggplant-pearl millet rust –Puccinia substriata.(http://nt.ars-grin.gov/taxadescriptions/factsheets/pdfPrintFile.cfm?thisApp=Pucciniasubstriatasubstriata).

Sorghum downy mildew

Peronosclerospora sorghi

SUMMARY: Sorghum downy mildew is caused by *Peronosclerospora sorghi*, a fungus-like pathogen. It is predominantly a soil-borne disease. Thick-walled oospores can survive for several years in the soil before infecting young plants. Oospores can also be carried over in seed. Systemic infections result in a distinctive striping of young leaves and stunted growth. Most plants fail to produce grain. Localized infections from wind-borne conidia are less damaging. The main control methods are clean seed and resistant varieties. Some strains of sorghum downy mildew also attack maize.

KEY SIGNS

New leaves produced in systemically infected plants first show a loss of colour (chlorosis) before developing white stripes, widening with subsequent leaf production – this differs from much narrower streaks associated with maize dwarf mosaic virus. Leaves are narrower and more erect in diseased plants, compared to healthy plants. The white areas eventually dry up, going reddish-brown as oospores are formed. The weakened leaves eventually shred, releasing oospores. Infected plants are stunted and usually do not produce any grain.

Localized infections caused by conidia, another type of spore produced by downy mildews, result in brown, rectangular lesions on leaves. Masse of conidia produced on the underside of leaves give them a typical downy or woolly appearance.

MANAGEMENT

Prevention – what to do before signs are seen

Cultural approaches: A combination of clean, healthy seed and resistant varieties is the best strategy for managing downy mildew.

Resistant varieties are effective in reducing disease losses but their use will depend on farmer acceptance, adaptation to local growing conditions and availability.

Seeds from areas with downy mildew have a higher risk of being contaminated. Given that the disease is well-established across Africa, it is important to buy seed from a reliable source.

Early planting of sorghum will limit the spread of the disease through reduced availability of wind-borne conidia. Experimental trials suggest that soils with a low sand content suppress oospore germination, which is also less likely at lower soil temperatures. Translating these observations into practical recommendations requires further investigation.

Another option is to alternate sorghum with crops other than maize, which may be infected by the same strains of downy mildew that infect sorghum. Crop rotation will limit build-up of oospores in the soil, the principal source of disease outbreaks in many areas.

Sorghum downy mildew also infects related weed hosts, such as Columbus grass (*Sorghum almum*). The importance of wild reservoirs is still not fully understood but may be small in comparison to persistence of oospores in the soil and aerial spread of conidia. Removing weeds and plant remains from previous crops is often helpful in reducing potential sources of disease although, as with crop rotation, this may not be practical for smallholders.

Chemical approaches: Seed for sowing next season should be dried to less than 20% moisture to reduce the risk of disease transmission. Seed dressing with a suitable fungicide such as metalaxyl, is also effective in reducing carry-over.

Foliar sprays with fungicides containing metalaxyl at 10 and 40 days after emergence of young sorghum plants will, in conjunction with seed treatment, eliminate the disease. Late season spraying will prevent systemic infections and limit the production of oospores. The cost and practicality of foliar sprays suggests this method is unsuited to small-scale farmers. There is little evidence that this is a common practice in Africa. There is an added danger of metalaxyl-resistance arising, as seen in the USA.

CAUSE

Downy mildew is caused by *Peronosclerospora sorghi*. This is a fungus-like organism, or water mould, belonging to the same group of organisms as *Phytophthora* (one of which causes late blight) and known scientifically as

oomycetes. Powdery mildews are caused by true fungi, which are controlled by different types of fungicide. There are no powdery mildews of sorghum.

P. sorghi was originally divided into strains that infected sorghum and maize, and those that only infected maize. Molecular techniques suggest that strains infecting maize may be different species.

When oospores penetrate the roots of young plants the infection spreads to the leaves, where wind-borne spores known as conidia are produced, as well as more oospores. The oospores can be blown in the wind, though soil is a more important source of disease carry-over. Oospores and fungal fragments are also seed transmitted.

The short-lived conidia are blown on to new plants where they infect leaves. Disease spread is limited, perhaps less than 100 metres. It is rare for such infections to become systemic and their overall impact on production is small. Aerial spread is, however, still important in spreading the disease to new areas and in maintaining disease pressure.

IMPACT

The persistence of oospores in soils and their spread in seed, together with aerial dispersal by conidia, has helped establish sorghum downy mildew across Africa and beyond. The disease was introduced to North America, where it also causes significant losses, in the early 1960s. Effective quarantine measures have prevented the disease spreading to Australia. Disease outbreaks are common and their potential impact is kept in check largely through the use of clean seed and resistant varieties. However, major losses occur regularly in Nigeria, for example, with similar impacts in other West African countries.

Sorghum downy mildew is most damaging when it infects roots and results in systemic infections. Localized infections are less severe and the effect on production is minimal. Systemic infections affect the overall growth of the plant, which is frequently barren and without any grain. Losses of up to 100 000 metric tonnes have been reported from India. In southern Nigeria losses of more than 10% have been observed, probably enhanced by the occurrence of a maize-infecting strain of the pathogen.

DISTRIBUTION

Sorghum downy mildew is probably present in all countries in Africa where sorghum is grown. It has been recorded from Egypt, south through Sudan and Ethiopia to Kenya, Tanzania and Malawi, and beyond to Zambia, Zimbabwe and South Africa. In West Africa the disease is reported from Burkina Faso, Ghana and Nigeria.

FURTHER READING

Crop Protection Compendium (www. cabi.org/cpc) and Plantwise Knowledge Bank (www.plantwise.org).

Fact sheets on sorghum downy mildew:http://amarillo.tamu.edu/files/2010/11/SorghumDownyMildewPart2.pdf

Frederiksen, RA (1980) Sorghum downy mildew in the United States: overview and outlook. Plant Disease 64, 903-908 (www.apsnet.org/publications/plantdisease/backissues/Documents/1980Articles/PlantDisease64n10_903.pdf)

The most recent comprehensive scientific review of sorghum downy mildew is:

Jeger MJ, Gilijamse E, Bock CH, Frinking HD, 1998. The epidemiology, variability and control of the downy mildews of pearl millet and sorghum, with particular reference to Africa. Plant Pathology 47, 544-569 (PDF can be downloaded free from http://onlinelibrary.wiley.com/doi/10.1046/j.1365-3059.1998.00285.x/abstract)

Frederiksen RA, Odvody GN (editors), 2000. Compendium of sorghum diseases. Second edition. American Phytopathological Society, Minnesota.

Bacterial leaf blight of rice

Xanthomonas oryzae pv. *oryzae*

Photo: IRRI Photos, Flickr, CC BY-NC-SA 2.0, http://bit.ly/1RTS8bt

Bacterial leaf blight symptoms in the field.

Photo: Donald Groth, Louisiana State University AgCenter, CC BY 3.0 US, www.bugwood.org

Pale yellow-whitish streaks caused by bacterial leaf blight on the leaves, showing wavy margins.

SUMMARY: Bacterial leaf blight of rice kills seedlings and destroys the leaves of older plants. The disease is extremely serious worldwide and has emerged as a major problem in irrigated crops in the Sahel. Recently, it has also been reported from East Africa. Wild hosts maintain the disease between crops and spread occurs in irrigation, floodwaters, in wind and rain, and in seed. Management requires planting resistant or tolerant varieties, good drainage of fields, removal of weeds, ploughing under of stubble and removal of volunteer seedlings.

KEY SIGNS

On seedlings, leaves show grey-green streaks starting from the tips and margins; later the streaks join together, turn yellowish-white with wavy edges, dry up and die. If the infection is systemic, i.e. throughout the entire plant, then leaves wilt, dry up and die – a symptom common on transplanted seedlings, known as 'kresek' (an Indonesian word). On older plants, the leaves show pale yellow steaks with wavy margins going from the tips towards the base, and then die.

Small milky drops of liquid (bacteria) ooze from the streaks, later drying as a crust on the leaves. Streaks also occur on the leaf sheaths and the stems of susceptible varieties. To check that bacteria are present in the leaves, cut a leaf across a young streak and place the cut end in a glass of water: if bacteria are present a cloudy liquid will stream out.

Some symptoms of bacterial leaf blight could be confused with bacterial leaf streak, caused by a different but related bacterium. The difference appears in the margins of the leaf streaks: those of bacterial leaf streak are straight, whereas those of bacterial leaf blight are wavy. Confusion could also occur between seedlings damaged by stem borers and bacterial leaf blight. To find out which one is present, squeeze the low part of the plant. If a yellowish liquid oozes from the ends of cut leaves, the cause is bacterial leaf blight. Also, infected plants cannot be pulled easily from the soil.

MANAGEMENT

Prevention – what to do before signs are seen

Cultural approaches: National biosecurity organisations need to ensure that rice seed entering the country originates from crops certified free of bacterial leaf blight and is subject to closed quarantine upon arrival, and further testing for the bacterium causing this disease.

In low-input farming systems, where resource-limited farmers can hardly ever afford external inputs, control of bacterial leaf blight is mainly through the use of resistant/tolerant varieties in combination with sound management practices, such as good weed control. Resistant varieties are available in Southeast Asia: PSB Rc82 is a standard variety with resistance; Macassane has been shown to have improved bacterial leaf blight resistance and is being used in Mozambique, and two IRRI varieties – IR22 and IR 54 – are resistant and moderately resistant respectively in Tanzania. Several NERICA hybrids from AfricaRice have been tested and found to have resistance. Check whether these are available locally.

Take care when selecting seed. For farmers who are using their own seed, it is important that they always select from plants uninfected by bacterial blight symptoms.

During planting and crop growth, do not damage seedlings when taking them from the nursery. They should not be pulled from the soil, but eased out with roots intact; the leaves should not be clipped.

Be careful with fertilizer applications: too much nitrogen increases shoot and leaf production and creates conditions that promote disease development because of higher humidity. 80-100 kg N/ha is suggested, depending on local conditions.

Ensure good drainage of fields and nurseries. Even in irrigated lowland rice, most varieties are not able to withstand flooding for more than a week, and floodwater spreads the bacterial leaf blight between plants. Keep fields clean of weeds, especially species of *Leersia* which are considered to be the main weed host of bacterial leaf blight. After harvest, plough land to bury rice stubble, straw, rattoon plants and volunteer seedlings. Note that the bacteria causing this disease do not live long in the soil.

Chemical approaches: Chemical treatments, such as applications of copper compounds or antibiotics, are costly and in any case have been shown to be ineffective.

CAUSE

Bacterial leaf blight is caused by *Xanthomonas oryzae pv. oryzae.* It is a vascular disease, meaning that the bacteria enters the plant and moves in the xylem, the water-conducting system of the plant. A number of strains are known from molecular studies; differences exist between those in Japan and the Philippines and, importantly, between strains in Asia and Africa.

There is another serious bacterial disease of rice, which is closely related; it is called leaf streak disease caused by *X. oryzaepv. oryzicola.* Both these diseases have re-emerged recently with the expansion of rice cultivation in West Africa. Current breeding programmes have not yet produced resistant or tolerant plants.

Bacterial leaf blight is one of the most serious diseases of rice. It is found worldwide in both temperate and tropical regions, causing epidemics in irrigated and rainfed lowlands, especially where fields have poor drainage and the potential for flooding.

Infection occurs through natural openings and wounds. Rice has natural openings at the leaf margins called hydathodes where excess water passes out of the leaf; leaves have stomata, the breathing pores. Wounds are made when seedlings are pulled from the seedbed and also when leaves are cut before transplanting. They are also made by insects; for example, leaf rollers, leaf folders and beetles. Once inside the plant, the bacteria invade the water conducting tubes, block them and cause a wilt.

Spread within the crop occurs in irrigation and floodwater, and in wind-driven rain. Over longer distances spread is probably via seed. In general, temperatures of 25-34°C and relative humidity of over 70% favour the disease.

Survival of bacterial leaf blight between crops is not well understood. It probably occurs in seed, but it seems that survival is not long in soil (about 5-6 weeks). In hot, dry conditions the bacterium dies rapidly. Similarly, survival is only for a few days in the stem (stubble) and root remains after harvest – about a month under dryland conditions and perhaps half as long when submerged. Longer survival between crops is on weeds, especially grasses, the most important of which are *Leersia* species, known as rice cutgrass.

IMPACT

Bacterial leaf blight can be devastating if it comes early. Yield losses range from 20% to more than 70% in Southeast Asia and India. In the Philippines, yield losses of susceptible varieties may reach 23% in the wet season, 7% in the dry. In Africa, it was first reported in the 1970s, but it is only more recently that it has become a serious disease, particularly of irrigated rice in the Sahel. There have been regular epidemics since 2002 in a number of countries. In Niger, for instance, a 2013 study showed losses in irrigated areas range from 19 to 63%, estimated to cost between 200,000 and more than 500,000 FCFA/ha (approximately, US$400-1000/ha). Mali, too, is recording epidemics, with yield losses of 75-80% in parts of the country from a combination of bacterial leaf blight and bacterial leaf streak. These two diseases are holding back the intensification of rice production in the country. Even if the disease comes late it can still cause a reduction in quality of the grain by making it black and brittle, even though the loss in yield is small.

DISTRIBUTION

Bacterial leaf blight occurs in Southeast Asia, India, Japan and Africa. It is also present with limited distribution in USA, Central and South America and Europe. In West Africa, it is a serious disease of irrigated rice in the savannah and Sahel. It was identified for the first time from Mali in the late 1970s; since then has been found in many other African countries (Burkina Faso, Benin, Cameroon, Mali, Niger, Nigeria, Senegal and Togo). It appeared in East Africa in 2013, and is now known in Mozambique, Rwanda, Tanzania and Uganda.

FURTHER READING

Program & Abstracts.3rd Africa Rice Congress 2013. Rice Sciences for Food Security through Smallholder and Agri-business Development in Africa. 21-24 October 2013, Yaoundé, Cameroon. (http://bit.ly/1dUmAok).

Rice leaf blight (Xanthomonas oryzae pv. oryzae). Plantwise Knowledge Bank. (http://bit.ly/1fAodt8).

Xanthomonas oryzae pv. oryzae. CABI Crop Protection Compendium. (http://bit.ly/1URtFrt).

Xanthomonas oryzae pv. oryzae. Wikipedia. (http://bit.ly/1GfYZVU).

Rice blast

Magnaporthe grisea

Photo: IRRI Photos, Flickr, CC BY-NC-SA 2.0, http://bit.ly/1O6koWt

Diamond-shaped lesions on the leaves of rice.

Photo: IRRI Photos, Flickr, CC BY-NC-SA 2.0, http://bit.ly/1FHJY1R

Rice blast may attack the stem at the nodes.

SUMMARY: Rice blast, caused by the fungus *Magnaporthe grisea*, attacks leaves, stems and flowers, killing plants up to tillering, or reducing grain yield and quality on plants that reach maturity. In Africa it is a problem of upland rice in particular. Diamond-shaped spots with white centres and dark borders occur on the leaves and rots develop on stems and flower heads. Control is by using tolerant or resistant varieties, dividing nitrogen fertilizer into several splits, avoiding water-stressed plants, eliminating crop residues, and application of seed treatments if fungicides are affordable and available.

KEY SIGNS

Oval or diamond-shaped spots (5-15 mm long and 3-5 mm wide) with dark borders occur on the leaves. Often the spots have yellow halos. Spots develop quickly under moist conditions and produce a large number of spores on both sides of the leaves. As they age, the spots become longer, the centres turn whitish-grey and the borders become wider and red-brown. The spots join together and the leaves die. Severely infected fields have a scorched appearance.

Spores from the leaves infect the leaf sheath, stem and panicle and cause rots. There are several different types of rot: collar rot appears at the junction of leaf base and leaf sheath; this can kill the leaf; neck rot (also called rotten neck) appears on the stem below the panicles (the flower heads) and can destroy the stem or result in pale-coloured grains that are partly filled, known as whiteheads; panicle rot occurs on the branches of the panicle so that it appears brown or black; node rot (slightly swollen parts of the stem where the leaves and tillers develop) occurs on the stem below the panicles, the rots become black-brown and dry and, if the stem breaks, the plant dies.

MANAGEMENT

Prevention – what to do before signs are seen

Cultural approaches: Although the main method of control of blast is the use of resistant varieties, nevertheless, cultural practices help to lessen the disease and should always be considered.

Plant blast-resistant or tolerant varieties, especially NERICA varieties – hybrids between *Oryza sativa* and *O. glaberrima* bred by AfricaRice, that have high yield potential and short growth cycle. These include NERICA 9, 12, 15, 16 and 18, which have done well in Burkina Faso, Guinea, Mali and Nigeria. Additionally, lowland WITA varieties have been released for West and Central Africa, bred for improved grain yield, resistance to diseases (blast and rice yellow mottle virus), and tolerance to drought and iron toxicity. Check if these varieties are available locally.

Where it is possible to alter the planting date, select a time to avoid flowering coinciding with periods of high humidity, which favours blast disease. Ideally, neighbouring farmers should plant at the same time to avoid spread of blast from older infected crops to those that are younger.

During crop growth, be careful to avoid any cultural practices that weaken the plants and make them more susceptible to blast. For instance, if applying nitrogen, divide the application into two or three splits, rather than applying it all at once. If possible, avoid water stress, or extended drain periods. Flood the field as often as possible.

Chemical approaches: Treat seed of upland rice with fungicide 1-2 days before sowing to reduce seed-borne infections of blast. A number of products are effective, for instance captan and mancozeb, as well as systemic products such as pyroquilon, azoles and strobilurins. Check the registration of these products and their availability.

Control – what to do after signs are seen

Cultural approaches: Although fungicides are available for controlling blast, expense and availability are major issues for smallholders, and they are rarely used. If required, use the products mentioned above for seed treatments. Treatments should be applied when symptoms of blast are first seen in the crop. Eliminate crop residues as soon as possible after harvest, to reduce the possibility of the fungus surviving to infect crops of the next season's plantings.

CAUSE

The fungus *Magnaporthe grisea* is responsible for rice blast. This is the sexual state of the fungus (created when two fungal strains combine), but it is rarely found on rice plants in the field. It is the asexual state which causes blast on rice and also grey leaf spots on wild grasses. This is known as *Pyricularia grisea*. There are many strains of blast, and analysis of them in West Africa and Asia has shown that populations vary in their ability to infect rice and cause severe damage. This variability is important in the management of the disease because it allows the fungus to overcome the resistance in breeders' varieties.

Blast is the most serious disease of rice in West Africa, particularly in upland rice, which represents about 40% of the rice cultivated in West and Central Africa. The disease affects all above ground parts of the rice plant, at all growth stages – leaf development, flowering and seed formation. Seedlings are killed and so are older plants up to the time of tillering. After tillering the plants are more resistant to the disease, but leaf and panicle infections still lower yields.

The disease is particularly serious in areas of frequent, prolonged showers and temperatures in the range of 24-28°C. This is because the leaves need to be wet for 6-8 hours for spore germination, and high humidity – close to 100% – is needed for infection and spore formation. In upland areas, conditions are favourable to the disease because differences between day and night temperatures cause dew to form on the leaves and the overall temperatures are cooler. By contrast, in lowland tropical areas, leaf infection is less, but blast is still serious in seedling nurseries and on panicles.

Spread occurs mainly from spores released on the wind. There is also spread in irrigation waters. Spores are spread long distances on air currents and wind. Survival between crops is in straw and stubble, in or on seed, through volunteer rice plants, and alternative hosts, mostly grass species.

IMPACT

Blast is one of the most destructive diseases of rice. It is estimated that the amount of rice that is lost to blast annually could feed 60 million people. However, impact varies greatly with cropping system, the varieties grown and management practices. In Africa, the impact of the disease is said to be less than that in Asia where, in many places, 2-3 overlapping crops guarantee that spores are present to cause major epidemics. There are three main reasons for this difference in Africa: (i) rice crops are often rotated with root crops and legumes, (ii) they are intercropped with non-hosts, such as sorghum, maize, cassava and vegetables and (iii) there is lower fertilizer use. However, losses are reported in upland rice in Sierra Leone (3-14%), Liberia (77%), Ivory Coast (0.5-59%) and Uganda (up to 50%), and it can also cause major damage to rain-fed lowland and irrigated rice cultivation.

DISTRIBUTION

Blast occurs in all rice-growing countries. Over 85 countries have reported the disease.

FURTHER READING

Blast (leaf and collar rot). Rice Knowledge Bank. (http://www.knowledgebank.irri.org/training/fact-sheets/pest-management/diseases/item/blast-leaf-collar).

Magnaporthe grisea. CABI Crop Protection Compendium. (http://www.cabi.org.ezproxy.library.uq.edu.au/cpc/datasheet/46103).

Magnaporthe grisea. Wikipedia. (http://en.wikipedia.org/wiki/Magnaporthe_grisea).

National Diagnostic Protocol for Rice Blast on rice only: caused by Magnaporthe oryzae. (http://plantbiosecuritydiagnostics.net.au/wordpress/wp-content/uploads/2012/12/NDP-14-Rice-blast-Magnaporthe-V1.0.pdf).

Somado EA, Guei RG, Keya SO (eds) (2008) NERICA: the New Rice for Africa – a Compendium. (http://www.africarice.org/publications/nerica-comp/Nerica%20Compedium.pdf)

Rice yellow mottle disease

Rice yellow mottle sobemovirus

Photo: Noah Phiri.

Rice growing in northern Zambia showing symptoms of yellow mottle disease.

SUMMARY: Rice yellow mottle disease causes major epidemics and yield loss in lowland irrigated rice throughout sub-Saharan Africa. Leaves turn yellow or orange with green streaks, plants are stunted, tiller number is reduced and panicles produce unfilled or sterile grain. There are many ways it is spread: beetles and grasshoppers and perhaps also other insects and mites; leaf-to-leaf and root-to-root contact; and on harvest implements. Management depends on use of tolerant varieties – crosses between African and Asian rice – supported by cultural techniques, e.g. removal of grasses and sedges that are alternative hosts of both virus and insects before planting, and destruction of crop residues after harvest.

KEY SIGNS

Symptoms vary due to differences in variety, environmental factors (temperature, light, humidity) and the growth stage of the crop. The newly emerged leaves show yellow (or orange) and green stripes of different width, sometimes continuous for up to 10 cm. If seedlings are infected early, plants become stunted, the number of tillers is less than on healthy plants, panicles fail to emerge completely and the grain is unfilled or sterile. The virus kills susceptible varieties.

MANAGEMENT

Prevention – what to do before signs are seen

Cultural approaches: The use of tolerant varieties is the best way to manage rice yellow mottle disease. Traditional upland varieties of African rice, *Oryza glaberrima*, are moderately resistant or tolerant. One variety from Mozambique is highly resistant: the gene that provides this resistance has been transferred to several of the lowland NERICA varieties by backcrossing. These lines have been tested in Burkina Faso, Cote d'Ivoire, Gambia, Ghana, Guinea, Liberia, Mali, Niger, Nigeria and Sierra Leone. However, there are isolates of the virus in 'hot spots', where disease pressure is high, that can overcome the resistance provided by the *O. glaberrima* gene, so a second gene is being added. Check to see if these resistant and tolerant varieties are available from suppliers in the local area.

An integrated pest management approach incorporating a number of cultural techniques is also recommended. These include: cleaning weeds from irrigation canals and around rice fields to remove reservoirs of virus and insects, especially in the off-season; early planting; direct sowing to reduce transfer of disease and/or insects from contaminated nurseries; and burning straw and stubble after harvest.

In addition, synchronous planting, i.e. planting fields at the same time in an area so that insects do not spread the virus from older to younger crops, and a rice-free period between crops are also recommended.

Control – what to do after signs are seen

Cultural approaches: Remove infected plants from the field as soon as signs of the disease are seen.

CAUSE

Rice yellow mottle virus causes this disease, which is of great economic importance. The host range is narrow; apart from Asian rice (*Oryza sativa*) and African rice (*Oryza glaberrima*) the virus is found in some wild *Oryza* species and a few grasses, for instance, members of the genera *Eleusine, Eragrostis, Echinochloa* and *Cyperus*.

Chewing insects – beetles and grasshoppers – and possibly also some sucking bugs spread the disease, and there is also evidence of involvement by mites. Evidence suggests that the virus is non-persistent in chewing insects, meaning it is picked up when the insects feed on diseased plants and transferred straightaway when they feed again on healthy plants; there is no multiplication of the virus in insects.

Other methods of spread are leaf and root contact between infected and healthy plants, and on sickles used to harvest crops, in which case there is potential for ratooned crops (new crops sprouting after harvest) to become infected. Transplanted rice is more vulnerable to infection than that direct-seeded.

The disease does not spread through the soil or via seed.

IMPACT

Rice yellow mottle disease is a major problem on lowland irrigated rice, causing losses of between 25 and 100% depending on the variety. By 1990, only 25 years after it was first reported, it had spread throughout West and Central Africa, Tanzania and Madagascar, and had become a major problem in Burkina Faso, Côte d'Ivoire, Mali, Niger, Senegal and Sierra Leone. The epidemics of the disease that occurred at this time were the result of intensive rice cultivation – irrigation, monoculture and use of high-yielding, but highly susceptible, Asian varieties. Presently, this disease is a threat to some 3 million hectares of rice in sub-Saharan Africa.

Traditional African upland varieties are relatively more tolerant to the disease than introduced lowland varieties.

DISTRIBUTION

The virus is widely distributed throughout East and West Africa since it was first reported in Kenya in the mid-1960s. It is not found in other parts of the world.

FURTHER READING

Rice yellow mottle virus. CABI Crop Protection Compendium. (http://www.cabi.org.ezproxy.library.uq.edu.au/cpc/datasheet/47658)

The genes that could beat the AIDs of rice. Global Rice Science Partnership. Annual Report 2011. CGIAR. (http://bit.ly/1PoM6jG)

Red-billed quelea

Quelea quelea

Photo: Lip Kee Yap, Flickr, CC BY-SA 2.0, http://bit.ly/1Gs86sq

Juvenile, female and male red-billed quelea.

Photo: Eric Boa, CABI, CC BY 4.0

Local measures taken by farmers to scare birds are useful but not enough to prevent losses caused by major irruptions.

SUMMARY: The red-billed quelea is a small brown weaver bird that can occur in huge flocks. The world's most abundant wild bird, quelea are found only in Africa, especially in semi-arid zones. They are seed-eaters, both of wild grasses and cereals such as sorghum, rice and wheat, but also eat insects, including pests of crops. Estimated annual damage to crops of up to US$80 million has been recorded across Africa, and it is said to be the most important crop pest in Africa. Quelea attack crops when other natural food sources are exhausted. Rainfall linked to germination of wild grass seeds is used to predict likely growth in flocks and to plan early control interventions. Control methods include spraying fenthion, an organophosphate, but this also kills non-target birds and the pesticide needs careful handling. Other methods with fewer side-effects include fire bombs and dynamite. Large-scale control efforts need area-wide coordination, farmer cooperation and adequate funds. Control methods used by smallholders range from scaring tactics and physical barriers, to home-made weapons (e.g. catapults) and can be locally successful if the threat of bird damage is relatively low.

KEY SIGNS

Quelea are abundant and widespread in their semi-arid habitat. Drab in appearance, they have a distinctive red bill. They are small, between 11 and 13 cm long when adult, and weigh about 15-20 g. Individuals can eat up to 10 g of seed in a day. Huge populations of birds occur when conditions for breeding are favourable.

The presence of quelea does not always mean that they are a threat or indeed responsible for major crop losses. The threat depends on availability of natural food supply while poor growth of crops could have many causes. Even when quelea do attack small grain crops, some farmers suffer more losses than near neighbours. Large scale production of small grain crops presents a more vulnerable target.

MANAGEMENT

Prevention – what to do before signs are seen

Cultural approaches: The nature of the risk from quelea attack needs to be carefully assessed before committing money and time to control programmes that may not be as necessary as first thought. Money and effort may be best spent in population reduction rather than direct control in fields, a decision made in the 1980s in Kenya. Current strategies here and in other countries are difficult to assess given an apparent lack of published information.

Timing of planting and selection of different crops can reduce the risk of quelea damage. Smallholders may have limited

flexibility to delay planting or to grow alternative, non-cereal crops.

Small plots of cereals can be protected from quelea by scaring tactics, including banging and loud noises. Scarecrows should be placed at several places in a field to be most effective. Physical barriers, such as string and streamers, may also work though entail too much work or expense to be practical. In areas where quelea are a threat to smallholders it makes most sense to ask farmers what methods they have already tried, and with what level of success, before making final recommendations.

Predictive models were used in South Africa from 2002 to 2008 for the migration and breeding of quelea. The model was based on satellite-derived rainfall estimates and how this would affect the germination of natural grasses, the favoured food source of the bird. The aim was to give early warning of potentially damaging large populations of quelea. The forecasts were highly accurate though the model is no longer available.

Control – what to do after signs are seen

Cultural approaches: Trapping birds will reduce damage if populations of birds are low. Birds can be eaten, though this does not happen in all countries where quelea are abundant.

Chemical approaches: Chemical control targets breeding colonies of quelea, a strategy known as 'search and destroy'. Fenthion, an organophosphate pesticide, is commonly used in aerial spraying and from vehicle-mounted machines to kill the birds. The chemical needs careful handling and will also kill non-target birds. Chemical control is said to be less efficient than fire-bombs and dynamite, two other methods that usually require government or third party funding.

CAUSE

There are four species of quelea, of which *Quelea quelea* is the only major pest. Quelea are passerine (perching) birds and belong to the Ploceidae family. Known collectively as weaver finches, birds in this family produce typical hanging nests in trees, woven from straw and small twigs. Quelea pairs breed up to three times a year. Their ability to migrate long distances in search of food and new breeding grounds makes quelea a particularly difficult pest to control.

There are an estimated 170 million birds in southern Africa alone. A total population of 1.5 billion breeding pairs make quelea reportedly the most abundant wild bird in the world.

Quelea feed preferentially on wild grass seeds. The abundance of wild grass seeds is often patchy and, even though the birds work cooperatively to locate natural food sources, easier alternatives may be preferred. Quelea will eat grains of sorghum, millet, rice, teff, wheat and other small-sized cereals and cause enormous amounts of damage. They do not eat large seeds, such as maize grains or chickpeas. The birds migrate long distances to new areas with potential food (and water). Quelea also eat insect pests and therefore have 'good' features in addition to their notorious role as perhaps the most damaging crop pest in Africa.

The reasons why quelea shift from eating natural grass seeds to cereal crops appear to vary and so it can be difficult to predict when the bird is likely to become a major pest, even when large colonies can be readily observed. Damage can also be spatially uneven, with some farmers worse affected by feeding damage than close neighbours. Caution is needed in deciding when and to what extent large-scale control programmes should be implemented. There is also the danger of ascribing all crop losses to quelea when other things, such as low soil fertility and other pests and diseases, may be responsible for reduced production.

IMPACT

Reported losses include US$80 million per annum across Africa: 13% of rice production loss in the River Senegal valley each year; 50% loss of crop production in fields attacked by quelea. Note that overall losses on a national scale may be relatively small, no more than 5% as one source suggests, but that this will mask many cases of small-scale farmers losing all their major crop production.

DISTRIBUTION

The quelea is a sub-Saharan species. It is found from East Africa to South Africa. It occurs from Senegal in West Africa across the semi-arid zones towards Nigeria and then east towards Ethiopia and Somalia, but no further north, and down to South Africa.

FURTHER READING

Crop Protection Compendium (www.cabi.org/cpc) and Plantwise (www.plantwise.org) for technical fact sheet.

There are many published sources of information on quelea, available via a basic Google search. The University of Nebraska holds proceedings of bird control seminars held from 1964 – 1983 at http://digitalcommons.unl.edu/icwdmbirdcontrol from which various papers on Quelea can be accessed. For example: Erickson WA (1979) Diets of the red-billed quelea (Quelea quelea) in the Awash river basin of Ethiopia. Bird Control Seminars Proceedings, paper 25. Other quelea papers are available at http://digitalcommons.unl.edu/vpcthirteen

Ornithology sites may contain useful observations about quelea which help in formulating local control strategies. For example: www.natureuganda.org

Striga or witchweed (multiple crops)

Striga species

Photo: USDA APHIS PPQ Archive, CC BY 3.0 US, www.bugwood.org

Striga hermonthica *flowering on maize.*

Photo: Rob Williams, CABI

Photo: International Institute of Tropical Agriculture, Flickr, CC BY-NC 2.0, http://bit.ly/1JvujUT

Striga gesnerioides *parasitizing roots of cowpea (left); flowers of Striga hermonthica (right).*

SUMMARY: *Striga*, or witchweed, are parasitic weeds which infest millions of hectares of land planted to maize, sorghum, millets, upland rice, cowpea and sugarcane, reducing yields by 30-100%. There are many species in Africa, growing mostly in arid regions of low soil fertility, but four dominate: *S. hermonthica, S. asiatica, S. aspera* and *S. gesnerioides*. Witchweeds tap into the xylem of host plants causing yellowing, stunting and wilting. Seeds are tiny and can spread over long distances, probably in wind-blown soil and over shorter distances in rain run-off, on shoes and hooves of livestock. Management depends on use of resistant varieties and cultural control measures, including crop rotations, weeding, raising the fertility of soils and the use of trap crops.

KEY SIGNS

The common species of witchweed in Africa are *S. hermonthica, S. asiatica, S. aspera* and *S. gesnerioides.* They parasitise cowpea, maize, sorghum, millet, sugarcane, upland rice and wild grasses. Affected plants are stunted, yellow, scorched and wilted, symptoms which are similar to nutritional deficiencies or drought. Although it is sometimes difficult to distinguish between symptoms caused by witchweed and those caused by other environmental factors, if plants wilt when the soil is still moist then this is a sign that witchweed is a likely cause.

MANAGEMENT

Prevention – what to do before signs are seen

Cultural approaches: Host resistance together with cultural control measures offer the best methods. There are now several crops bred for resistance/tolerance. For instance, there are sorghum varieties resistant to *S. asiatica* (released in Tanzania in late 2003) and varieties of maize, upland rice and sugarcane resistant to the same species. Resistance in maize has been more difficult to attain, but varieties are now available. There are also cowpeas resistant to the strains of *S. gesnerioides* in West Africa. There are as yet no witchweed-resistant millet varieties.

Control measures for *S. aspera* are generally comparable to those for *S. hermonthica*, but there are differences in varietal susceptibility and resistance: maize hybrids tolerant of *S. hermonthica* are susceptible to *S. aspera*, while rice varieties resistant/tolerant to *S. hermonthica* are also resistant/tolerant to S. aspera.

Recommended cultural measures to prevent witchweed include: improving soil fertility by, for example, rotations or intercropping with leguminous crops and use of cattle or green manure; delaying planting until seasonal rains have set in to reduce host plant water loss; and use of trap crops. Trap crops stimulate witchweed seed germination, but do not become infected so the witchweed dies, e.g. cotton, cowpea, pearl millet and soyabean are trap crops for *S. asiatica*.

Chemical approaches: Coating maize seed resistant to the herbicide imazapyr (IR maize) with the same chemical has shown promise against S. asiatica and S. hermonthica in Kenya, especially as the maize can be interplanted with legumes, as long as they are at least 15 cm away from the treated seed.

Control – what to do after signs are seen

Cultural approaches: Hand pulling of the larger species, such as *S. hermonthica*, if populations are low, but not *S. asiatica*, which is much smaller and has seeds that mature and are shed faster.

Chemical approaches: There are a number of herbicides that can be used, but in terms of cost and effectiveness, 2,4-D is possibly the best for cereal crops. The disadvantage is that more than one application may be needed and it can damage broad-leaf (e.g. leguminous) crops, if these are intercropped.

CAUSE

Most species of witchweed have bright green leaves and stems 30-100 cm high, with bright flowers and poorly developed roots. *S. gesnerioides* is different – it is totally parasitic, with small, unexpanded leaves and a cluster of fleshy, pale-green or yellow shoots, 10-20 cm high.

Witchweed seeds are tiny: each plant produces hundreds of thousands. They remain viable in the soil for many years, germinating in response to chemicals from the roots of potential host plants. The radicle fastens to a root, penetrates it, growing into the xylem, taking water, minerals, sugars and amino acids needed by the parasite until it emerges and starts to photosynthesise; however, it remains dependent on the host for food, as its ability to photosynthesise is poor. In addition, chemicals that regulate plant growth pass from the parasite to its host. These are chemicals that stunt the growth of shoots, but stimulate growth of roots, all to the advantage of the parasite.

Witchweed seeds are probably spread long distances on the wind, along with soil. Spread over short distances occurs in run-off during heavy rains, on the feet, tools and machinery of farmers, and via the hooves and gut of livestock. There is concern that some species are continuing to spread in Africa.

Witchweeds get their name because symptoms occur before the appearance of the parasite above ground. Because they need a host for germination and early development scientists call them hemiparasites.

IMPACT

The effect of witchweed can be devastating, especially to food crops – legumes, maize, millet, sorghum, sugarcane and upland rice – grown in some of the poorest farming systems of Africa. It is estimated that witchweed affects 40% of the arable savannah region, resulting in losses of up to US$13 billion per annum.

S. hermonthica is responsible for greater crop loss in Africa than any other weed. It is widespread throughout most of the continent, attacking maize, millets, sorghum, sugarcane, upland rice and wild grasses. Estimates of crop losses on sorghum are: Ghana (21%), Nigeria (10%), Gambia (8%) and Benin (6%), but there are also undefined losses in Burkina Faso, Cameroon, Cote d'Ivoire, Ethiopia, Kenya, Mali, Niger, Senegal, Sudan, Togo, Tanzania and Uganda. For maize, it is estimated that 4 million hectares of land is infested, with losses of 30-80%, valued at US$380 million to US$1 billion.

S. asiatica is a serious weed of sorghum, maize, millets, rice, sugarcane, upland rice and many wild grasses. Over one million hectares in Malawi and 250,000 hectares in Angola are infested. In Malawi, average crop losses in maize were put at 28% in infested fields and 4.5% for the country as a whole. Like *S. hermonthica*, it is severe where crops are grown under marginal rainfall and low soil fertility, over a wide range of soil types. In Tanzania the weed is associated with mainly sandy soils. Climate change could assist its further spread in arid regions of North Africa.

S. aspera attacks the same range of crops as *S. hermonthica*, except that it attacks maize more than sorghum, and can parasitise irrigated rice. Where the two species occur together, S. aspera emerges and matures more rapidly.

S. gesnerioides is a serious weed of cowpea across West Africa. Surveys in northern Nigeria and Burkina Faso have shown that farmers rate this weed as highly damaging, and trials have recorded yield losses of 30 to more than 50%.

DISTRIBUTION

Witchweeds occur naturally in Africa, Asia and Australia. S. hermonthica is widespread throughout Africa, but is more common in west, east and central parts of the continent, commonly associated with both sandy and clay soils of low fertility, especially those low in nitrogen. It predominates in savannah regions associated with cereals.

S. asiatica is widespread in East Africa and occurs in numerous countries (as well as in Asia): Togo, southern Ethiopia, Tanzania, Botswana, Malawi, Mozambique, Namibia, South Africa, Swaziland, Uganda, Zimbabwe and Zambia. It occurs mostly in sandy soils. *S. aspera* has a similar distribution to S. *hermonthica*, but is much less common in eastern Africa. *S. gesnerioides* has wide distribution in Africa from north to south of the continent, but only in western Africa (Senegal, Mali, Togo, Burkina Faso, Ghana, Nigeria, Niger, Cameroon and Chad) is it a serious problem on cowpea.

FURTHER READING

Striga asiatica. CABI Crop Protection Compendium: http://www.cabi.org.ezproxy.library.uq.edu.au/cpc/datasheet/51786

Striga aspera. CABI Crop Protection Compendium: http://www.cabi.org.ezproxy.library.uq.edu.au/cpc/datasheet/51848

Striga gesnerioides. CABI Crop Protection Compendium: http://www.cabi.org.ezproxy.library.uq.edu.au/cpc/datasheet/51785

Striga hermonthica. CABI Crop Protection Compendium: http://www.cabi.org.ezproxy.library.uq.edu.au/cpc/datasheet/51849

5. The major pests and diseases of legumes

This section covers the most important pests and diseases that affect common bean, groundnut and cowpea in Africa.

Table 5.1 summarises the main signs associated with the most important pests and diseases of beans in Africa.

Table 5.2 summarises the main signs associated with the most important pests and diseases of groundnut in Africa.

Table 5.3 summarises the main signs associated with the most important pests and diseases of cowpea in Africa.

In the following pages, more details on the signs, management options, cause and impact are provided.

Table 5.1: Key signs of the most important pests and diseases of common beans in Africa.

Key signs	Possible pest or disease	See page
Holes in the bean. Small grey and reddish-brown beetle-like insects, 3-4.5 mm in length, on stored beans.	Bean bruchid	52
Round holes on the flowers and folded leaves that are stuck together. Caterpillars may be seen when flowers are opened. Pods have hole where the caterpillar entered: look for yellowish-brown balls (faecal matter) on the outside of the pods.	Legume pod borer	54
Small caterpillars: yellowish-white to reddish-brown, dark brown to black head, black rows of short hairs running along backs. Fully grown caterpillars 35-40 mm, dark brown, yellow-green, to red-brown, with grey-black stripes along the side.	Cotton bollworm	60
Dark brown to black sunken spots on the stems and first leaves of seedlings; spots produce pink spores. Red-purple streaks turn brown to black on lower surfaces of leaves, especially the veins, Lower leaves infected first. Small oval or circular spots with red-brown margins on stems and petioles. Dark brown irregular specks or small dark brown circular sunken spots, bordered by distinct brown to black margins on pods.	Bean anthracnose	62
Small angular spots with bright yellow haloes on leaves that look water-soaked, expanding as large brown dead areas. Dark streaks occur on stems. On pods, spots are water-soaked at first, becoming red-brown and sunken, mostly circular.	Bean blight	64
Plants stunted. Leaves are distorted with dark green areas along the main veins, light green-yellow between. Often green areas have a bubble-like (or blister-like) appearance; leaves may also curl downwards with rolling of the leaf blade.	Common mosaic of bean	66
Water-soaked spots on leaves; 'pin-pricks', scattered on the leaf blade. Yellow area (the 'halo') spreads out from spots, which then go red and dry up. Water-soaked areas also on pods, stems and leaf stalks; sometimes produce a whitish ooze.	Halo blight of beans	68
Small, lighter coloured areas on leaves, almost yellow; later become bronze to dark grey, roughly circular to more elongated and up to 10 mm across. Grey to dark powdery appearance on spots on lower side of leaves. When held up to the light the older leaf spots are darker, more reddish and often with a distinct ring.	Cercospora leaf spots	80

Table 5.2: Key signs of the most important pests and diseases of groundnut in Africa.

Key signs	Possible pest or disease	See page
Small caterpillars: yellowish-white to reddish-brown, dark brown to black head, black rows of short hairs running along backs. Fully grown caterpillars 35-40 mm, dark brown, yellow-green, to red-brown, with grey-black stripes along the side.	Cotton bollworm	60
Dark spots, roughly circular and up to 10 mm diameter on leaves. Oval spots on stems, leaf stalks and pegs – causes leaves to fall off. Early leaf spot: spots reddish-brown on the upper surface surrounded by yellow halo and brown on the lower leaf surface. Late leaf spot: spots dark brown to black arranged in concentric circles visible on lower leaf surface; not usually surrounded by yellow halo.	Early and late leaf spot of groundnut	70
Dark-brown spots on the stems just below soil level. Yellowing and wilting, first single branch then entire plant. Cottony growth on the lower stem; sometimes also soil surface.	Stem and pod rot of groundnut	72
Chlorotic rosette: bright yellow leaves, except for small parts that remain green. Early infections result in severely stunted plants with small, deformed leaves. Mosaic rosette: yellow and dark green areas on the leaves. Plants stunted, although less than those with chlorotic rosette. Green mosaic: very dark green small leaves, or light and dark green mosaic, with leaf margins rolled downward. All: stunted plants and severe pod loss.	3 similar groundnut rosette diseases: chlorotic rosette, mosaic rosette and green rosette	74
Small flecks on leaves, which develop into larger spots on leaves, petioles, pegs. On leaves, spots are round, 0.5 to 1.5 mm diameter, at first yellow, rapidly turning orange and then red-brown as masses of spores develop and break through the leaf surface. Spots mostly on the underside of leaves. On stems and petioles, spots elongate.	Groundnut rust	76

Table 5.3: Key signs of the most important pests and diseases of cowpea in Africa.

Key signs	Possible pest or disease	See page
Holes in the bean. Small grey and reddish-brown beetle-like insects, 3-4.5 mm in length, on stored beans.	Bean bruchid	52
Round holes on the flowers and folded leaves that are stuck together. Caterpillars may be seen when flowers are opened. Pods have hole where the caterpillar entered: look for yellowish-brown balls (faecal matter) on the outside of the pods.	Cowpea pod borer	54
Nymphs and adults of pod-sucking bugs suck on pods and under leaves. Feeding puncture wounds visible. Young pods shrivel and dry prematurely, become deformed and have reduced grain yields.	Pod-sucking bugs of cowpea	56
Silver streaks on the leaves, fruit and pods. Distortion, drying and shedding of flowers. Thrips are tiny, slim, elongated, shiny black with pale bands across the top of the forewing. Shake flowers on to white paper to check for thrips.	Bean flower thrips	58
Spots tan to brown and sunken, circling stems, branches, leaf and flower stalks.	Anthracnose of cowpea	78
Small, lighter coloured areas on leaves, almost yellow; later become bronze to dark grey, roughly circular to more elongated and up to 10 mm across. Grey to dark powdery appearance on spots on lower side of leaves. When held up to the light the older leaf spots are darker, more reddish and often with a distinct ring.	Cercospora leaf spots	80
Patchwork of irregular light-coloured areas scattered across the surface of the normal green leaf, most easily seen with the leaf held against the light. Discoloured areas have a clearly defined boundary corresponding to leaf veins. Leaves smaller than usual and may have wrinkled appearance.	Mosaic diseases of cowpea	82
Affected plants are stunted, yellow, scorched and wilted when the soil is still moist. Witchweed species affecting cowpea has unexpanded leaves and a cluster of fleshy, pale-green or yellow shoots, 10-20 cm high, with pink flowers.	Striga or witchweed	84

Bean bruchid

Acanthoscelides obtectus

Photo: Clemson University – USDA Cooperative Extension Slide Series, CC BY 3.0 US, www.bugwood.org

Adult bean bruchid on bean with exit hole.

Photo: Pest and Diseases Image Library, CC BY-NC 3.0 US, www.bugwood.org

Adult, 3-4.5 mm long. Body grey, brown and reddish-brown, without any distinctive patterns.

SUMMARY: The bean bruchid is a major post-harvest pest of most bean species. The infestation begins in the field, but becomes a major problem after harvest, when the holes left in the beans reduce the value of the crop. To prevent a major infestation it is important to harvest the beans as soon as they reach maturity. Storing the beans in a clean facility is the most important measure. Remove old beans from the store and use a disinfectant to clean the storage room if necessary. Use an air-tight storage container if possible.

KEY SIGNS

While the bean bruchid is primarily known as a storage pest of grain legumes, it starts attacking the pod while the crop is still in the field. However, it is during storage that it causes the most damage and can multiply. Signs include the eating away of the internal parts of the bean, holes in the bean and adults on the stored crop.

The eggs are milky white, dome shaped and oval. The larvae of bean bruchids are white and can be found in tunnels in the bean. The adults are 3-4.5 mm in length and grey and reddish-brown. The wings are short and have patches of yellowish and black hairs. Adults lay eggs on the outside of ripening pods and the larvae bore into the seeds and feed. Before pupating, the larvae cut a hole to exit through but remain inside the bean. When they reach the adult stage, the adults push their way out leaving a hole about 2 mm in diameter. When threatened, adults will pretend to be dead and fall from the plant.

MANAGEMENT

Prevention – what to do before signs are seen

Cultural approaches: Use clean certified seed.

Consider intercropping maize with beans.

Harvest beans as soon as they are mature to reduce the risk of heavy infestation. Remove and destroy all infested crop residues immediately after harvest.

Air-dry the beans to a moisture level of 12% or lower before storage.

A clean storage facility is one of the most important practices. Clean the storage facility prior to storage, using a disinfectant if necessary. Do not store old beans with newly harvested beans. Store beans in air-tight containers if possible, such as in plastic sealable bags, drums, or clay pots.

Chemical approaches: Mixing beans with vegetable oils, neem seed powder, wood ash or *Beauveria bassiana* (a fungus) can protect the stored beans and reduce losses. Add 1g of *Beauveria bassiana* or wood ash to 1 kg of the stored beans.

Control – what to do after signs are seen

Chemical approaches: The insecticide phosphine is also an effective fumigant for storage facilities, but is toxic, expensive and not widely available. For smallholders, the use of insecticides is not recommended since the beans are usually stored for short periods of time and intended for consumption.

CAUSE

The bean bruchid (*Acanthoscelides obtectus*), also known as the dry bean weevil, is a major pest of common bean and lima bean, and also cowpea. While the bean bruchid is primarily known as a storage pest of grain legumes, it starts attacking the pod while the crop is still in the field.

IMPACT

The bean bruchid is a major pest of stored beans. The damage caused has a negative impact on the value and marketability of the crop and can even change some quality characteristics of the crop, including the taste. The holes made by the larvae often make the crop unmarketable. Losses of up to 40% of the harvested crop have been reported in Tanzania.

DISTRIBUTION

The bean bruchid is native to South America, but has spread to most other warm regions of the world. In Africa, the pest has caused widespread damage in Kenya, Lesotho, Malawi and Nigeria, and is also present in Angola, Burundi, DRC, Rwanda, Tanzania, Uganda, Zambia, and Zimbabwe.

FURTHER READING

Kasambala, S. and H.Mziray. Avoiding bruchid infestation in stored beans. LEISA Magazine, 20.3, September 2004

Josephine M. Songa & Wilson Rono (1998). Indigenous methods for bruchid beetle (Coleoptera: Bruchidae) control in stored beans (Phaseolus vulgaris L.) International Journal of Pest Management, Volume 44, Issue 1, page 1-4.

Negasi, P. and T. Abate, Progress in Bean Bruchid Management. CIAT African Workshop Series, No. 27

Plantwise Knowledge Bank www.plantwiseorg/knowledgebank

AgriCultures Network http://www.agriculturesnetwork.org

Legume pod borer

Maruca vitrata

Photo: gailhampshire, Flickr, CC BY 2.0, http://bit.ly/1FRL4tM

The adult moth has brown forewings with white markings.

Photo: Merle Shepard, Gerald R.Carner, and P.A.C Ooi, Insects and their Natural Enemies Associated with Vegetables and Soybean in Southeast Asia, CC BY 3.0 US, www.bugwood.org

Caterpillars are whitish to pale green in colour with irregular brown-black spots. The head is dark brown.

SUMMARY: The legume pod borer, a moth, is a prominent pest of cowpeas and other beans throughout East and West Africa. The caterpillars feed on buds and flowers, and bore into the bean pods, eating the seeds and leaving a hole in the pod. Preventive approaches include early planting, use of resistant/tolerant and early maturing varieties, removal of alternative legume hosts, intercropping and crop rotation. Control options include handpicking and destroying eggs and larvae.

KEY SIGNS

The adult moths lay eggs one-by-one or in small groups on the flowers or flower buds and also on the terminal shoots of young plants. The eggs are oval, clear and light yellowish-white in colour; a single egg looks like a small drop of water.

The larvae, or caterpillars, can grow up to 17-20 mm long. They are whitish to pale green in colour with irregular brown-black spots and the head is dark brown. The young larvae can often be found in groups at this stage, but tend to be found individually later. Young larva feed on the flowers and foliage, but older larva are more mobile and more likely to be found feeding on and boring into the pods and eating the developing seeds. Once mature the larva drops from the plant to the soil where it pupates beneath plant litter.

The moth wings are blackish-brown with white markings on the forewings and a wingspan of 20-25 mm. The moths are active at night. During the day they can be found resting with their wings spread out on the undersides of leaves.

Cowpeas can be attacked by the legume pod borer from early budding through to harvest. Look for round holes on the flowers and also folded leaves that are stuck together. Open the flowers to look for larvae. The pods will have a distinct hole where the larva entered. Look for yellowish-brown balls on the outside of the pods; these are the droppings (faecal matter) left behind by the larvae as they bore into the pod.

MANAGEMENT

Prevention – what to do before signs are seen

Cultural approaches: Plant early to avoid the period of heavy infestation.

Use resistant/tolerant varieties and/or early maturing varieties if they are available in the area.

Remove alternate hosts such as common beans, kudzu, lima beans, green gram and other leguminous plants from in and around the field.

Intercropping cowpeas with sorghum or maize reduces the populations of the pod boring pests and decreases yield losses.

Rotate cowpea with maize.

Control – what to do after signs are seen

Cultural approaches: Hand-pick eggs and larvae from the plants and crush them. Prune leaves with white silk threads that stick together and also remove older leaves to allow more sunlight to reach the leaves and stems of the plants.

Chemical approaches: Pod borers are difficult to control with insecticides because they remain hidden in the pod/bean. However, neem products have proven to be effective against the larvae and are more cost-effective than synthetic insecticides. *Bacillus thuringiensis (Bt)* is also effective against the larvae.

CAUSE

Maruca vitrata was formerly classified as *Maruca testulalis* Geyer and *Crochiphora testulalis* Geyer. Common names include legume pod borer, bean pod borer, lima bean pod borer, mung moth or maruca.

Host plants include beans, cowpeas and other legumes like lablab and kudzu.

There are four stages in the life cycle: egg, pupae, larva and the adult moth. Egg development lasts an average of 3 days, the larva stage lasts an average of 13-14 days, the pupa stage 6-7 days and adults can live an average of 6-10 days.

IMPACT

Legume pod borers can cause yield losses of up to 80%. The legume pod borer causes the most damage to beans (*Phaseolus vulgaris*), cowpea (*Vigna unguiculata*), pigeonpea (*Cajanus cajan*) and green gram (*Vigna radiata*).

DISTRIBUTION

The legume pod borer is found throughout the tropics and subtropics, especially in East and West Africa, but cannot survive in temperate climates. It can be spread from country to country through exported legumes.

FURTHER READING

Plantwise Knowledge Bank www.plantwise.org/knowledgebank

Pod-sucking bugs of cowpea

Hemiptera spp.

Photo: IITA, Flickr, CC BY-NC 2.0, http://bit.ly/1Ktfx0V

Pod sucking bugs.

Photo: IITA, Flickr, CC BY-NC 2.0, http://bit.ly/1LNR2MX

Pod sucking bugs feeding on cowpea.

SUMMARY: The pod-sucking bugs are a group of major pests of cowpea in sub-Saharan Africa. They are difficult to control due to their mobility: a single control strategy is unlikely to succeed. An integrated approach that combines cultural practices, such as early planting and fertiliser use, combined with carefully timed insecticide applications can manage the pest.

KEY SIGNS

The nymphs and adults of several different species of pod-sucking bugs suck on the sap of the young pods causing them to shrivel and dry prematurely, become deformed and have reduced grain yields.

The shrivelling and drying symptoms can be confused with other stresses such as drought and disease, however, the feeding puncture wounds are a sign of pod-sucking bugs.

The insects can often be found on the pods or under the leaves of cowpeas and other host plants. Pod-sucking bugs feed on a wide range of legumes and are very mobile, which makes them challenging to control. Monitor crops regularly to look for pod-sucking bugs and symptoms such as shrivelling and prematurely dried out pods.

MANAGEMENT

Prevention – what to do before signs are seen

Cultural approaches: Plant resistant cultivars if available.

Plant early to avoid periods of heavy infestation.

Intercropping with sorghum or greengram is reported to help reduce populations and the need for insecticides. To be effective against pod-sucking bugs, intercropping should be used with other management strategies. Intercropping with maize, which is commonly practiced by smallholder farmers, is said to cause an increase in pod damage caused by pod-sucking bugs and therefore is not recommended.

Clean up haulms to prevent the insects from over seasoning in the crop residues.

Chemical approaches: Studies have shown that applying phosphorus at 30 kg P/ha significantly decreases the pod-sucking bug population and significantly increases yields.

Control – what to do after signs are seen

Cultural approaches: During flowering and pod formation, bugs can be collected and killed by hand in small plots.

Chemical approaches: IITA reports that cultural controls alone cannot control pod-sucking bugs; 2-3 sprays of insecticides are necessary to obtain a good crop. Pyrethrin-based insecticides can be used to control pod-sucking bugs. Commercial neem products (active ingredient azadirectin) are also reported to be effective against the pod-sucking bugs. Spray with

an insecticide once at budding (30 to 35 days after planting), once at full flowering (10 days after first spray) and, if a heavy infestation, spray once during the podding stage (10 days after second spray). Spray early in the morning or late in the evening when insects are active.

CAUSE

Pod-sucking bugs belong to the Hemiptera order of insects, which all have piercing, sucking mouthparts. There are several species of pod-sucking bugs in Africa, including the spiny brown bug (*Clavigralla tomentosicollis*), Riptortus bugs (*Riptortus dentipes*), the green stink bug (*Nezara viridula*), the tip wilter (*Anoplocnemis curvipes*) and *Mirperus jaculus*. Legumes are host plants of the pod-sucking bugs.

IMPACT

Cowpea is an important source of protein in the diet in sub-Saharan Africa. The pod-sucking bugs can cause yield losses between 30-70%. The impact in each country depends on the species.

DISTRIBUTION

Pod-sucking insects are distributed throughout Africa.

FURTHER READING

Plantwise Knowledge Bank www.plantwise.org/knowledgebank

Pitan, O.O.R. and J. A. Odebiyi. Crop Losses in Cowpea Due to the Pod-Sucking Bugs Riptortus Dentipes, Mirperus Jaculus, Noplocnemis Curvipes and Nezara Viridula. Insect Sci. Applic. Vol. 21, No. 3 pp. 237-241.

I.Y. Dugje, L.O. Omoigui, F. Ekeleme, A.Y. Kamara, and H. Ajeigbe. Farmers' Guide to Cowpea Production in West Africa. International Institute of Tropical Agriculture (IITA). 2009.

P. Nampala, M.W. Ogenga-Latigo, S. Kyamanywa, E. Adipala, N. Oyobo snd L.E.N. Jackai. Potential Impact of Intercropping on Major Cowpea Field Pests in Uganda. African Crop Science Journal, Vol. 10. No. 4, 2002, pp. 335-344. http://www.bioline.org.br/request?cs02032

Asiwe, J.A.N. The Impact of Phosphate Fertilizer as a Pest Management Tactic in Four Cowpea Varieties. African Journal of Biotechnology, Vol. 8 (24), pp. 7182-7186, 15 December 2009. http://www.ajol.info/index.php/ajb/article/viewFile/68820/56887

Olufemi O. R. Pitan; J. A. Odebiyi; G. O. Adeoye. Effects of phosphate fertilizer levels on cowpea pod-sucking bug populations and damage. International Journal of Pest Management, Volume 46, Issue 3, 2000.

Bean flower thrips

Megalurothrips sjostedti

Photo: Stan Diffie, University of Georgia, CC BY-NC 3.0 US, www.bugwood.org

Adult bean flower thrips, greatly enlarged.

Photo: Ko Ko Maung, CC BY-NC 3.0 US, www.bugwood.org

The tiny adults on a bean flower.

SUMMARY: Bean flower thrips are a major pest of cowpea and other leguminous crops throughout sub-Saharan Africa. The pest feeds on the buds and flowers of the crop, which can cause the flowers to be distorted and fall from the plant, resulting in crop losses. Control of the pest can be achieved through a combination of cultural practices, such as ploughing to destroy pupae, intercropping and crop rotation with maize, planting early, and use of chemical pesticides, including home-made remedies based on black pepper.

KEY SIGNS

Bean flower thrips affect many crops, particularly cowpeas. Thrips breed in the flowers of beans (legumes) and feed on the buds and flowers. The adults are tiny, slim, elongated, shiny black and are distinguished from other species of thrips by pale bands across the top of the forewings. They are also the largest of the flower thrips present in Africa.

Symptoms of bean flower thrips infestation include distortion, discolouration and shedding of flowers. Severe infestations – that is 20 or more thrips per flower – cause the flowers to fall from the plant, which prevents the pods from developing.

Thrips start attacking the plant before the flowers open, which causes the flowers to dry and become brown.

Monitoring for thrips should begin about 30 days after planting. Look for silver streaks on the leaves, fruit and pods. Open the flower buds to look for larvae or adult thrips, or shake the flowers onto a white piece of paper and count the thrips that fall out.

MANAGEMENT

Prevention – what to do before signs are seen

Cultural approaches: Prior to planting, plough and harrow the field to destroy pupae.

Plant early to ensure the crop is well established and better able to withstand an infestation.

Intercrop with maize or sorghum to reduce populations.

Rotate cowpea with non-leguminous crops, such as maize.

Control – what to do after signs are seen

Cultural approaches: Provide adequate water to plants, as water-stressed plants are more susceptible to attacks. In infested fields, destroy crop residues after harvest.

Chemical approaches: There are biological control options for the bean flower thrips.

Bio-pesticides can control and reduce populations. The fungi *Beauveria bassiana* and *Metarhizium anisopliae* have been found to be effective against bean flower thrips. Neem-based pesticides (azadirectin) are effective against nymphs and can reduce the number of eggs laid by adults. Spinosad is also an effective microbial insecticide.

Pepper extracts are also effective in controlling and reducing the populations of the bean flower thrips. Dry the pods of West African black pepper (*Piper guineense*) and grind into a powder. Mix 500-100 grams with 3 litres of water and let it sit for 12 hours. Filter the water to remove the solid parts and mix with an additional 1.5 litres tap water to make the concentrate. Add some soap to make it stickier. Spray 4-6 times a week.

Chemical pesticides, such as cypermethrin, dimethoate and lambda-cyhalothrin, are effective control options. All three chemicals are considered to be moderately hazardous and should be used carefully. These pesticides should not be applied more than twice per planting season. Farmers should follow label recommendations for dosage and safe use.

CAUSE

Megalurothrips sjostedti was formerly known as *Taeniothrips sjostedti*. Other common names include legume thrips, flower bud thrips and African bean thrips.

Adults lay eggs into the leafstalks (petioles). Development from egg to adult takes about 20 days.

IMPACT

Cowpea is an important protein source in sub-Saharan Africa. Bean flower thrips cause 20-70% yield losses in cowpeas in many parts of Africa. A heavy infestation can cause total crop loss.

DISTRIBUTION

Bean flower thrips can be found throughout sub-Saharan Africa, both in regions with heavy rainfall and semi-arid regions. They are, however, most commonly found in the dry savannah regions where cowpeas are produced. Infestation can occur when adults fly from other host plants.

FURTHER READING

Plantwise Knowledge Bank www.plantwise.org/knowledgebank

Infonet Biovision http://www.infonet-biovision.org/default/ct/78/pests

Ekesi, S., Maniania, N.K., Onu, I. and B. Lohr. 2009. Pathogenicity of entomopathogenic fungi (Hyphomycetes) to the legume flower thrips, Megalurothrips sjostedti (Trybom) (Thysan., Thripidae). Journal of Applied Entomology, 122 (1-5):629 - 634.

Oparaeke A.M., 2006. The Sensitivity of Flower Bud Thrips, Megalurothrips sjostedti Trybom (Thysanoptera: Thripidae), on Cowpea to Three Concentrations and Spraying Schedules of Piper guineense Schum. & Thonn. Extracts. Plant Protect. Sci., 42: 106–111

Cotton bollworm

Helicoverpa armigera

Photo: Donald Hobern, Wikimedia Commons, CC-BY-2.0, http://bit.ly/1a8PJuf

Adult moth of cotton bollworm.

Photos: Gyorgy Csoka, Hungary Forest Research Institute, CC BY 3.0 US, www.bugwood.org

Cotton bollworm caterpillars in two different colours.

SUMMARY: The cotton bollworm is a major pest of many important food, oil and cash crops worldwide, including cereals, legumes, fruits and vegetables. A severe infestation of caterpillars of this moth can cause a complete loss in yield. Chemical control needs to be carefully timed as the caterpillars bore into the grains or fruit of the plant and are then protected. Resistance to pesticides, such as pyrethroids, has been reported in many countries. *Bacillus thuringiensis* (Bt) and neem extracts provide effective control against the caterpillars while minimising damage to natural enemies. Important cultural controls include removal and destruction of post-harvest crop residues, ploughing the soil to expose the pupae and uniform planting times.

KEY SIGNS

The cotton bollworm is considered to be one of the most important pests worldwide, attacking over 200 species of plants. The caterpillars feed on leaves and developing grain heads, and bore into the fruiting parts of plants.

The eggs are 0.4-0.6 mm across, round and yellowish-white, but change to a dark-brown colour before hatching. The eggs are typically laid singly on plants that are flowering or about to flower. The young caterpillars are yellowish-white to reddish-brown, with a dark brown to black head and black bumpy rows of short hairs running lengthwise on their backs. The fully grown caterpillars are 35-40 mm long and vary in colour from dark brown, yellowish-green, to reddish-brown, with greyish-black stripes along the side of the body and a thin light stripe. The pupae are 14-16 mm long, brown, and smooth on the surface. The adults also vary in colour, but typically the forewings are yellowish-brown with black kidney shaped markings in the middle of the wings and 7-8 small spots in a line along the border of the wing. The hindwings are white with a darker coloured band along the edge. The wingspan is about 35-40 mm at its widest point, and the body is 14-18 mm long. The adult moths are active at night and are good at flying, so they can easily move from plant to plant and field to field.

The caterpillars typically feed on the buds, flowers, grains, fruits and pods, but will also feed on the leaves and stems. In sorghum and millet, they feed on the developing grains during the milky stage and hide in the head during the day. In beans and tomato, they bore into the fruit. In maize, they feed on the developing seeds and bore into the cobs. The bore holes left by the caterpillars provide an entry point for bacterial diseases.

Monitoring is extremely important to prevent a severe outbreak. Monitoring should be done one or two days per week once the sorghum flowers bloom. Caterpillars can be seen on the plants, or shake the panicle heads over a bucket or sweep net. Bore holes and caterpillar dropping (frass) can often be seen on the plant. Pheromone traps and light traps can also be used to monitor the adult moths and provide some control.

MANAGEMENT

Prevention – what to do before signs are seen

Cultural approaches: Sorghum varieties with tight compacted heads are more likely to be attacked; plant varieties with loose open heads to reduce infestations and damage.

Resistant varieties of cotton, pigeonpea and chickpea have been released. Talk to local agro-input suppliers to see which varieties are available.

Planting a whole field at the same time (uniform planting) prevents movement of the moths from the older plants to the younger plants and reduces the damage.

Two post-harvest practices are important for preventing the population of bollworms from building up and becoming a severe problem the following season. First, it is important to remove and destroy crop residues immediately after harvest. Second, plough the soil to expose the pupae to direct sunlight and natural enemies.

Rotating with non-host crops can also prevent the population from building up, but because the bollworm can attack and harbour on many species of plants, rotation must be used together with other prevention and control practices.

Control – what to do after signs are seen

Cultural approaches: For small plots, it is possible to hand pick and destroy the eggs and young caterpillars.

There are many natural enemies of the bollworm, including *Trichogramma* spp. (egg parasitoids), several species of wasps and flies (caterpillar parasitoids), as well as many predators such as ants, lacewings, ladybugs, assassin bugs, minute pirate bugs and birds. Providing habitat to attract and preserve these natural enemies is an important strategy for controlling the bollworm.

Chemical approaches: The older caterpillars feed inside the fruiting part of the plant, protecting them from pesticide sprays. Therefore, if pesticides are used, they should be applied before the caterpillars bore into the pods or fruit of the plant. It is important to monitor the field regularly to look for eggs and young caterpillars before they bore into the plant. The decision to use a pesticide needs to be based on the severity of the problem, the presence of natural enemies and the economic value of the crop.

The cotton bollworm has developed resistance to several pesticides, notably synthetic pyrethroids.

Two microbial pesticides, *Bacillus thuringiensis* subspecies *kurstaki* and *Bt aizawai*, and *Helicoverpa armigera* nuclear polyhedrosis virus (HaNPV) can be used to control the young caterpillars with minimal harm to natural enemies. There are commercial products of these microbial pesticides available in many countries. Neem extracts, made from the neem seeds and leaves, and neem oil are also reported to be effective against the larvae and eggs.

CAUSE

Helicoverpa armigera has also been classified as *Heliothis armigera*. It is commonly known as the cotton bollworm, but is also called the African bollworm, tomato fruitworm, corn earworm, gram pod borer, old world bollworm and tobacco budworm. In French it is called chenille des epis du mais, noctuelle des tomates, or ver de la capsule.

Adult females can lay hundreds of eggs in a lifetime. They lay eggs singly on the tender parts of plants including the leaves, flowers, or fruit, and the eggs hatch in 3 to 5 days. The caterpillar period lasts 17 to 35 days, and the pupa period 17 to 20 days. The caterpillars drop from the plant to pupate inside a silk cocoon, 3-15 cm deep in the soil. The adult moths live between 1 and 25 days. The total life cycle can last up to 60 days; if conditions are favourable, then the total life cycle can be completed in 30 days. The length of time for development is mostly dependent on availability of food and the temperature: development time is shorter at higher temperatures. In warmer areas there can be numerous cycles per season.

IMPACT

The cotton bollworm is a major pest of many crops, including sorghum, soy, cotton, maize, common beans, chickpeas, groundnut, sunflower, potatoes, winter grains, citrus, wheat, barley, oats and a wide range of vegetable crops, including okra. The larvae can develop on many wild plant species, including *Amaranthus*, *Cleome* and *Acalypha*. The bollworm has a preference for feeding on the flowering and fruiting parts of plants, which causes high economic losses. If the infestation is severe, then it can result in a complete crop loss.

DISTRIBUTION

Native to Africa, the cotton bollworm has spread to Asia, Europe, Australia and the Pacific Islands. There have also been recent reports of infestations in South America. The pest can be spread through infested plant propagation materials. The adults are strong flyers and can move easily from field to field in areas where host plants are available; they can also be spread by strong winds.

FURTHER READING

Plantwise Knowledge Bank www.plantwise.org/knowledgebank

Bean anthracnose

Colletotrichum lindemutheanum

Photo: Denis Persley, Department of Agriculture and Fisheries

Brown sunken spots with dark margins caused by bean anthracnose on the pods of French bean.

Photo: PABRA, Flickr, CC BY-NC-SA 2.0, http://bit.ly/1KQ3MBN

Red to black spots occur on the leaves on the underside.

SUMMARY: Bean anthracnose is a fungal disease primarily of the common bean, *Phaseolus vulgaris*. It occurs worldwide, including many countries of Africa. Signs of infection occur on leaves, mostly limited to veins on the underside. Spots, oval to circular, tan with dark borders, develop on stems and pods, and also on seedling stems and leaves. Spores formed in the spots are spread in rain-splash or by wind and rain. Infected seeds are sources of the fungus and responsible for long-distance spread. The disease is managed by using certified or approved seed. Seeds can also be treated with the fungicides captan or thiophanate-methyl. Intercropping with maize and use of 2-3 year rotations with non-legume crops are other preventive options reported to be effective.

KEY SIGNS

Bean anthracnose, caused by a fungus, is a major disease of common bean (snap or French bean), *Phaseolus vulgaris*, in both temperate and tropical countries worldwide. Anthracnose means 'coal disease'; it describes diseases caused by fungi that produce dark spots on leaves, petioles, stems and fruit.

Anthracnose fungi are invariably associated with wet-weather. Moderate rainfall and wind, and temperatures of 20-24°C, favour the diseases they cause. In the case of bean anthracnose it is seed-borne.

Seedlings from infected seeds show dark brown to black sunken spots on the stems and the first leaves, the cotyledons. Severely infected seedlings are stunted. If the spots surround the stems, the seedlings die.

The spots produce pink spore masses that spread to the other leaves. Dark streaks, red-purple at first, turning brown to black, appear on the lower surfaces, especially on the veins, but they are less obvious from above. Lower leaves are infected first, then those higher up the plant.

Infection of stems and petioles produces 1-2 cm diameter oval or circular spots with red-brown margins. If spots surround the stems, they collapse.

Spots are also evident on the pods. They are dark brown irregular specks or dark brown circular sunken spots, 1-10 mm diameter, bordered by distinct brown to black margins. During wet weather these spots, too, produce pink spore masses. In severe cases the pods shrivel and the seeds become infected.

MANAGEMENT

Prevention – what to do before signs are seen

Cultural approaches: The use of seed certified free from infection, and planting varieties with resistance to the disease should be used in combination with cultural techniques. However, there are different races of this fungus. If the local

races are not known it would be best for smallholders to test varieties said to be tolerant or resistant before planting all their land with any one selection.

Intercropping with maize is reported to reduce the incidence of disease.

Planting in widely spaced rows that run parallel to the prevailing wind enables the leaves and pods to dry as quickly as possible, reducing risk of infection.

During the growth of the crop, apply a mulch to prevent soil from splashing onto the plants during heavy rains. Do not carry out field operations when the plants are wet, to minimise the spread of the disease by water splash.

After harvest, collect and burn debris, or plough the remains into the soil.

Chemical approaches: If certified or approved seed is not available, and there is no information on resistant varieties, then treat the seed with fungicide; use captan or thiophanate-methyl. Note that benomyl and thiram, widely used in the past as seed dressings, are banned or restricted in some countries. Consult your agriculture authorities for the eligibility of any fungicide before giving recommendations to growers.

Control – what to do after signs are seen

Cultural approaches: Use a 2-3-year rotation between crops of beans on the same land if anthracnose is established in the field – for example, rotate with maize or solanaceous crops (tomato, potato and eggplant).

Chemical control: It is very unlikely that fungicides would be economical for the control of this disease, or that they would be effective; however, if a fungicide was required on, for instance, plants grown for seed, use mancozeb, copper compounds or chlorothalonil. The appropriate times to apply fungicides are: (i) at flower set; (ii) late flowering; and (iii) pod fill.

CAUSE

Bean anthracnose is caused by a fungus, *Colletotrichum lindemutheanum*. It exists as a number of races. *C. lindemunthianum* is the asexual state, producing small cylindrical spores on leaves, stems and pods, that are pale pink when massed together in cup-shaped structures in the spots.

Survival of the fungus between crops is in seed and in crop debris. Spread is in seed and by the movement of spores in wind-driven rain. There is some evidence that animals, insects and people walking through the crop when it is wet can spread the spores. Moisture is essential for spore development, spread and germination, as well as for infection.

IMPACT

The disease is important on the common bean, *P. vulgaris*, and other food legumes. It affects bean quality and hence its marketability. It was once widespread and serious in temperate and sub-tropical countries, leading to losses of economic significance. Problems have been reported in North, Central and South America, with up to 95% losses in Colombia, and also in Europe, Africa, Australia and Asia. However, thanks to the introduction of certified and approved bean seed schemes, its impact in many countries has declined. In Australia, for instance, the disease has been practically eradicated from commercial production using this method of control. Nevertheless, epidemics are still reported as new races of the fungus keep appearing. Bean anthracnose remains a problem in Kenya, Malawi (90% losses reported) Tanzania and Uganda, and often occurs in Burundi, Rwanda, and DR Congo.

DISTRIBUTION

The disease is found worldwide, and is reported wherever *P. vulgaris* is grown. It occurs throughout Africa.

FURTHER READING AND ADDITIONAL INFORMATION

Anthracnose of bean (Colletotrichum lindemuthianum). Plantwise Knowledge Bank. (http://www.plantwise.org/KnowledgeBank/Datasheet.aspx?dsid=14918).

Collectotichum lindemuthianum. CABI Crop Protection Compendium. (http://www.cabi.org/cpc/datasheet/14918)

Persley D, Cooke T, House S (2010) Diseases of vegetable crops in Australia. CSIRO Publishing, Collingwood, Victoria 3066, Australia.

Vegetable crops: Bean anthracnose. Cornell Vegetable MD online. (http://vegetablemdonline.ppath.cornell.edu/factsheets/Beans_Anthracnose.htm).

Bean blight

Xanthomonas axonopodis pv. phaseoli

Photo: V.R. Wallen, Agriculture and Agri-Food Canada, CC BY 3.0 US, www.bugwood.org

Brown spots with wide yellow halos caused by bean blight.

Photo: Howard F. Schwartz, Colorado State University, CC BY 3.0 US, www.bugwood.org

Spots, some water-soaked, others brown, on the pods. Roughly circular and joining together.

SUMMARY: Bean blight is caused by a bacteria, *Xanthomonas axonopodis pv. phaseoli*. Key signs of the disease are spots on leaves, stems and pods. Seeds become infected internally and on the outside. The bacteria spread over short-distances in wind-blown rain, surface water run-off, and by people, machinery and insects moving through the crop. Long-distance spread is via infected seeds. The bacteria survive in plant debris, volunteer bean plants, weeds and seeds. Priority measures for management involve use of disease-free (ideally certified or approved) seed, rotations (2-3 years) with maize, avoiding entry to fields when foliage is wet, and removing volunteer plants and weeds.

KEY SIGNS

Common bacterial blight is a serious disease which attacks the foliage and pods of various kinds of beans. The main host is the common bean (snap or French bean), *Phaseolus vulgaris*, but other *Phaseolus* and *Vigna* beans are susceptible.

Plants grown from infected seed develop spots on the cotyledons (the first leaves) which then produce spores to infect other leaves. The first symptom is small angular spots that look water-soaked, expanding as large brown dead areas. The spots often have bright yellow haloes. In very susceptible varieties the spots continue to expand and the leaves appear burnt and become torn. Dark streaks occur on the stems, becoming lighter as they age.

On pods, the spots are water-soaked at first, becoming red-brown and sunken, mostly circular. A yellow liquid containing bacteria seeps out of the spots when plants are wet and humidity is high. In severe cases, the pods shrivel and die.

MANAGEMENT

Prevention – what to do before signs are seen

Cultural approaches: Ideally, use seed that is certified free from bacterial infection, or purchase from an approved source.

For smallholders who save their own seed:

- Carefully select plants for seed that do not show symptoms of disease. Choose only those plants without marks on the leaves or pods.
- If most plants show symptoms, do not use them as a source of seed; obtain seeds from a reliable retailer.
- If a thermometer is available, treat seed with hot water: 20 minutes at 52°C. Afterwards, surface sterilise the seed using sodium hypochlorite to kill bacteria on the seed surface. DO NOT treat all the seed with this method; try it out on some of the seed first, and always use a thermometer to measure temperature.

Remove weeds, volunteer (those that have grown without being planted) beans and other legume crops from the field before planting. Do not plant new crops next to those that have the disease.

Use maize as an intercrop; it reduces spread of the bacterium between bean plants.

Test different varieties or mixtures of different varieties for tolerance or resistance, but note high resistance to this disease is not reported.

Remove weeds as they develop. Weeding is important, not only to eliminate sources of the bacterium, but also to improve aeration so that the leaves dry out as rapidly as possible after rains or heavy dews.

Control – what to do after signs are seen

Cultural approaches: Use a 2-3-year rotation between crops of beans on the same land if bean blight is established in the field, e.g. use rotations with maize.

Avoid workers and machinery passing through diseased crops and then those that are disease-free, particularly in wet weather.

After harvest, collect and burn or plough back the diseased crop. It is likely that the bacterium survives only a few months in soil in the absent of a host.

Chemical approaches: Copper fungicides can be used to slow disease development, however this may not be a practical recommendation for smallholders, unless they wish to establish disease-free seed lots. To obtain maximum benefit from the use of fungicide, cultural control methods should also be applied.

CAUSE

Common bacterial blight is caused by *Xanthomonas axonopdic pv. phaseoli*. There are different strains of the bacterium; one is sufficiently genetically distinct and more aggressive to be considered a separate subspecies.

An important feature of this disease is that the bacteria are in or on the seed – it is seed-borne. Once inside the plant – though natural openings and wounds – the bacteria move though the vascular system to the leaves, stems and pods, and into the seeds. Seeds can also be infected from the spots that develop on the pods. Seeds can also become contaminated with bacteria if they come into contact with plant debris at harvest or when handled by people.

Wet weather and temperatures between 28-32°C favour disease development.

Spread of the bacteria occurs by wind-blown rain, rain-splash, surface water run-off, insects (beetles, grasshoppers and whiteflies) and by people and machinery moving through the crop.

Seed is also important in the survival of the bacterium: it can remain alive for more than 30 years. Bacteria also survive on plant debris and both on and in soil, where they can last for 6-18 months, and on other hosts, such as mung bean (*Vigna radiata*), Phasey bean (*Macroptilium lathyroides*) and Lablab (*Lablab purpureus*). The bacterium can also survive on the surfaces of plants without causing signs of disease.

IMPACT

This is a very important disease of common bean in temperate, sub-tropical and tropical countries. It is widespread throughout the bean growing areas of Africa. Surveys have shown that seed lots are often contaminated with the bacterium: in Uganda, just 0.2% seed infection can start an epidemic. Exact losses are difficult to determine because bean blight often occurs together with halo blight (*Pseudomonas syringae pv. phaseolicola*), another bacterial disease. Nevertheless, in North and South America, the Caribbean and Africa there are documented instances of the disease affecting crops grown for seed with yield losses of between 20 and 40%. It is a constraint to production in the Caribbean, and East Africa, in Ethiopia, Kenya and Uganda in particular.

DISTRIBUTION

Common bacterial blight is found worldwide in temperate, sub-tropical and tropical countries. It is present throughout Asia, North and South America, Central America and the Caribbean, Europe and in more than 20 African countries.

FURTHER READING AND ADDITIONAL INFORMATION

Allen DJ, Ampofo KO, Wortmann CS (1996) Pests, Diseases, and nutritional disorders of the common bean in Africa: A field guide.(http://books.google.com.co/books?id=kHoxVlEFW7AC&printsec=frontcover&hl=en&source=gbs_v2_summary_r&cad=0 - v=onepage&q&f=false).

Hortanswers. (University of Illinois Extension) (http://urbanext.illinois.edu/hortanswers/detailproblem.cfm?PathogenID=129).

Persley D, Cooke T, House S (2010) Diseases of vegetable crops in Australia. CSIRO Publishing, Collingwood, Victoria 3066, Australia.

Xanthomona saxonopodispv. phaseoli. CABI Crop Production Compendium.(http://www.cabi.org/isc/datasheet/56962).

Xanthomona saxonopodispv. phaseoli. Data sheets on quarantine pests. EPPO quarantine pest. Prepared by CABI and EPPO for the EU. (https://www.eppo.int/QUARANTINE/bacteria/Xanthomonas_phaseoli/XANTPH_ds.pdf).

Common mosaic of bean

Bean common mosaic virus

Photo: Howard F. Schwartz, Colorado State University, CC BY 3.0 US, www.bugwood.org

Bubble-like appearance of leaf blades of Phaseolus*; also the plant is stunted.*

Photo: Grahame Jackson, CABI, CC BY 4.0

Mosaic patterns on the leaves of Vigna sp.*, yard long bean.*

SUMMARY: Common mosaic of bean is caused by a virus that mainly affects *Phaseolus* and *Vigna* beans. It is spread in seed and by a number of aphid species. Plants are stunted, with leaves that show dark and light green patterns (mosaics), dark green areas along the main veins, and a bubble-like appearance. Pod yield losses range from 35% to near 100%. In Africa, management options are limited by the lack of healthy seed programmes and access to commercial seed with resistance to the virus, and rely mainly on cultural controls. Insecticides do not offer solutions, even if affordable and available.

KEY SIGNS

Common mosaic of bean is caused by a virus that infects *Phaseolus* species, especially *P. vulgaris* (common, snap or French bean) but also *Lablab purpurea* (hyacinth bean) and *Vigna unguiculata* subsp. *sesquidpedalis* (yard long bean). Additionally, many pasture legumes are hosts, i.e. they are susceptible to infection. The disease is spread in infected seed and also by aphids.

Plants grown from infected seed are stunted; leaves are distorted and show dark green areas along the main veins and light green-yellow between. This light and dark green pattern is called a 'mosaic'. Often the green areas have a bubble-like (or blister-like) appearance. The leaves may also curl downwards with rolling of the leaf blade. Plants with these symptoms rarely produce pods.

MANAGEMENT

Prevention – what to do before signs are seen

Cultural approaches: Use seed that is certified free from virus infection, or from an approved source.

Varieties have been bred for resistance to common mosaic of bean. Check to see if these are available locally.

Smallholders who save their own seed should:

- Carefully select plants for seed that do not show symptoms of disease, i.e. they look healthy.
- If most plants show symptoms, do not use them as a source of seed, but instead obtain seed from reliable sources, such as a commercial company or from other growers whose plants have been monitored for the disease.

Interplant with maize to reduce aphid infestation and virus infection.

Plant mixtures of bean varieties – a strategy used in parts of Africa.

Control – what to do after signs are seen

Cultural approaches: Do not plant new crops next to those that have the disease.

During early crop growth the most practical recommendation is to learn to identify plants with symptoms of seed-borne infection (see KEY SIGNS, above) and remove them as soon as symptoms are seen.

After harvest, collect and burn or plough back the diseased crop to destroy the aphids.

Chemical approaches: The use of insecticides for the control of aphids that spread the virus is not recommended. The time is short between an aphid sucking up the virus when it feeds on a diseased plant and spreading the virus as it feeds again on a healthy one. By the time the insecticide has killed the aphid it has spread the virus.

CAUSE

Bean common mosaic virus is transmitted through seed and by a number of aphid species. The virus can also spread in pollen, although seed and aphid transmission are the more important.

The aphids *Acyrthosiphon pisum*, *Aphis fabae* and *Myzus persicae* do not normally colonise beans. These transmit the virus in a non-persistent way: this means that after feeding on an infected plant, aphids can straight away move to a healthy plant and infect it as they feed once more. However, the aphids quickly lose the ability to spread the virus in this way. Other species, including the common aphid, *Aphis gossypii,* infest crops of beans and transmit the virus.

Several strains of the virus are known. It was previously thought that some cause wilting and death, known as 'black root', but these symptoms are now recognised as belonging to a separate virus, Bean common mosaic necrotic virus.

IMPACT

Common mosaic of bean is an economically important disease throughout Africa, Europe and North and Central America. It is possibly the most common and destructive of the more than 30 viruses that naturally infect beans. Seed infection can be high, with over 30% being common and up to 70% having been reported. A combination of infected seed and secondary spread by aphids can result in total infection with yield losses ranging from 35 to 98%. In general, the earlier the symptoms present, the greater the impact on yield. Plants produced from infected seedlings rarely produce beans.

DISTRIBUTION

The virus occurs worldwide, wherever beans are grown. It is present in North and South America, Central America and the Caribbean, Europe, Asia, Oceania, and in more than 20 African countries.

FURTHER READING AND OTHER RESOURCES

Bean common mosaic virus. CABI Crop Protection Compendium. (http://www.cabi.org.ezproxy.library.uq.edu.au/cpc/datasheet/9424).

Common mosaic of beans (Bean common mosaic virus).Plantwise Knowledge Bank. (http://www.plantwise.org/knowledgebank/datasheet.aspx?dsid=9424).

UC IPM Online. Dry beans: Bean Common Mosaic. (http://www.ipm.ucdavis.edu/PMG/r52101611.html).

Halo blight of beans

Pseudomonas savastanoi pv. *phaseolicola*

Photo: Howard F. Schwartz, Colorado State University, CC BY 3.0 US, www.bugwood.org

Early leaf spots with yellow halo.

Photos: Howard F. Schwartz, Colorado State University, CC BY 3.0 US, www.bugwood.org

Water soaked lesions on pods (A) showing internal decay (B).

SUMMARY: There are two serious bacterial diseases of bean: common blight, caused by *Xanthomonas* and halo blight, caused by *Pseudomonas*. Both diseases are spread in similar ways: in seeds, rain splash and by physical contact. Bean halo blight has a distinct yellowing around the initial leaf spot, which spreads outwards, though the symptoms of common blight are similar. Bean cultivars vary widely in their resistance to the two bacterial blights, and laboratory testing may be necessary to distinguish them. The use of clean seed is critical for both bacterial blights.

KEY SIGNS

Symptoms of halo blight of beans are most clearly seen on leaves. The first symptoms are water-soaked spots, little bigger than a pin-prick, scattered on the leaf blade. The bacteria produce a toxic chemical which results in a yellow area (the 'halo') spreading outwards from the spots, which then go red and dry up. The yellow areas from adjacent spots often join up. Water-soaked areas or lesions also develop on pods, stems and leaf stalks, and sometimes produce a whitish ooze which contains bacteria.

Seedlings that develop from diseased seed are systemically infected and lesions develop around the stem. The nodes rot and plants are stunted and distorted, with an overall lime-green colour.

Common blight has similar symptoms to halo blight. If in doubt, send samples to a laboratory to confirm which pathogen is present. Other fungal diseases which attack the leaves produce different shaped spots which are not water-soaked and lack the characteristic halo associated with halo blight.

MANAGEMENT

Prevention – what to do before signs are seen

Cultural approaches: The most important measure is to use certified seed. An alternative is to sow seed saved from plants which are healthy and occur in areas free from halo blight.

Some varieties of common bean are resistant to halo blight and they should always be used in areas of high rainfall, where the risk of halo blight is greatest. Choosing the right variety to plant can be difficult because there are always trade-offs: in Kenya GLPX92 (*Mwitemania*) is resistant to halo blight but susceptible to bean common mosaic virus; GLP 1004 (*Mwezi moja*) is susceptible to halo blight but less so to other major problems. Check with seed suppliers on the characteristics of available varieties in order to recommend the most suitable ones for farmers.

Other steps to take include deep-ploughing or removal of the remains of bean plants after harvest. This may be impractical for smallholders because of labour and cost constraints.

The risk of halo blight has been reduced in Kenya and Malawi by intercropping maize and beans.

Chemical approaches: There are no preventive chemical options.

Control – what to do after signs are seen

Cultural approaches: Hand removal of infected plants, particularly at the early stage of disease development, will limit spread of the disease.

Chemical approaches: Chemical treatment with copper-containing pesticides, applied at the first signs of the disease, can reduce the rate of spread of halo blight. This is, however, costly for smallholders to apply and gives only marginal gains. It is not effective against common blight.

CAUSE

Halo blight is a bacterial disease of *Phaseolus vulgaris*, the common bean, caused by the bacterium *Pseudomonas savastanoi* pv. *phaseolicola*. Previous names include *Pseudomonas syringae* pv. *Phaseolicola* and *P. phaseolicola*, but these are no longer accepted.

Halo blight affects the leaves and pods and can severely reduce yields. The main means of transmission is in seeds. Some infected seeds are wrinkled and discoloured but the majority show no symptoms.

The bacterium is also found on other hosts but there is little evidence that these are a common source of new infections on common bean. Although the pathovar (pv.) name suggests that this only attacks the common bean, other hosts are affected: in Tanzania, for example, these include *Phaseolus coccineus* (runner bean), soybean and certain weeds.

The disease spreads rapidly during rainfall when cooler temperatures, water splash and wind help transfer the bacteria to other plants. Bacteria may also be transferred as people walk through fields.

Several races or types of the bacterium have been characterised. Some occur only in certain countries or are associated with different alternative hosts; others are more aggressive. Scientists are studying these different races to develop resistant bean varieties which are acceptable to farmers.

IMPACT

The risk of infection and disease losses are greatest in cool, humid regions. Halo blight can spread rapidly in a crop and cause big losses. Examples include up to 43% yield losses in the UK and the US, where mechanisation and large-scale production of common beans may favour rapid disease spread, for example in irrigated fields. However, major losses due to halo blight have also been observed in Lesotho, Rwanda and Zimbabwe.

DISTRIBUTION

In eastern and central Africa, the disease occurs in Burundi, DR Congo, Ethiopia, Kenya, Rwanda, Tanzania and Uganda. In southern Africa, halo blight is found in Malawi, Mozambique, South Africa, Zambia and Zimbabwe. The disease has not been recorded from West Africa and is unlikely to occur there because growing conditions are less favourable to bean halo blight.

FURTHER READING AND ADDITIONAL INFORMATION

Crop Protection Compendium (www.cabi.org/cpc)

Plantwise Knowledge Bank (www.plantwise.org)

There are two useful fact sheets which compare different bacterial diseases on bean, including halo blight and common blight. Note that the recommendations and other advice relate to growing beans on a commercial scale in the western United States. The first fact sheet is:

Bacterial diseases of Beans. Fact sheet 2913. Colorado State University (www.ext.colostate.edu/pubs/crops/02913.pdf)

Common Bacterial Blight and Halo Blight. Two Bacterial Diseases of Phytosanitary Significance for Bean Crops in Washington State. Washington State University extension fact sheet • FS038E. (http://mtvernon.wsu.edu/path_team/FS038E-CommonBacterialBlightAndHaloBlight.pdf)

Another useful article from the US looks at common blight, halo blight and other bacterial diseases of beans in the most important area for bean production. Waveson RM, Schwart HF (2007). Bacterial diseases of dry edible beans in the Central High Plains. (www.plantmanagementnetwork.org/pub/php/diagnosticguide/2007/beans/)

Early and late leaf spot of groundnut

Mycosphaerella arachidis & M. berkeleyi

Photo: Grahame Jackson, CABI, CC BY 4.0

Early leaf spot, showing spots on the top surface of the leaf and clear yellow halos.

Photo: Jill Lenne, ICRISAT

Late leaf spot: spots on the underside of the leaf, with smaller halos. Microscopic examination is needed to tell early from late leaf spot.

SUMMARY: Early and late leaf spots, caused by the fungi *Mycosphaerella arachidis* and *M. berkeleyi*, are severe diseases of groundnuts worldwide. In Africa, they are reported to be major problems in Burkina Faso, Malawi, Mali, Nigeria and Sudan. These diseases cause spots on leaves, stems and petioles, resulting in leaf fall and high pod yield losses. Early leaf spots are brown with halos; late leaf spots are dark brown to black with dense spores forming ring patterns on the undersurface. Management of both diseases involves use of resistant, early yielding, varieties and cultural controls, which include at least a 1-year rotation, removal of volunteer plants and weeds, isolating crops from those that are infected, and elimination of plant debris after harvest. Applications of fungicide, e.g. chlorothalonil, are beneficial for leaf spots and also against a rust disease (*Puccinia arachidis*) common on groundnuts.

KEY SIGNS

There are two types of fungi that cause leaf spots on groundnuts: early leaf spot and late leaf spot. They look very similar and need microscopic examination to differentiate them. They are often found together in the same crop and even on the same plant. There are slight differences in symptoms, explained below, but from a farmer's point of view they are the same disease and should be treated as such.

Both types of fungi produce dark spots, roughly circular and up to 10 mm diameter on the leaves. Those of early leaf spot are reddish-brown on the upper surface surrounded by a yellow halo and brown on the lower leaf surface. Spots of the late leaf spot are dark brown to black and they are not usually surrounded by a yellow halo; if a halo is present it is smaller. The other difference is that the spores of the late leaf spot are arranged in concentric circles and these are visible on spots on the lower leaf surface. Both fungi produce oval spots on stems, leaf stalks and pegs, and cause leaves to fall off.

The first sign of the diseases is usually at 45-60 days after sowing, as spots on the older leaves. Early leaf spot is more common at first. This is gradually overtaken by late leaf spot, which produces more spores and more loss of leaves as the crop matures. Masses of spores are produced on the spots, but a hand lens, or better still a microscope, is needed to see them.

MANAGEMENT

Management of early and late leaf spot of groundnut is the same for both diseases.

Prevention – what to do before signs are seen

Cultural approaches: The best way to manage the disease is by growing resistant varieties[1] and by selecting those that produce yields early. Check whether these varieties and others are available locally (ICGV-SM 86715, ICGV 91225, ICGV-SM 93535). Priority should be given to varieties that yield in the shortest time to avoid the diseases. Note that some of the releases are tolerant to early leaf spot, but still susceptible to late leaf spot.

Whilst the crop is growing, keep weeds under control, otherwise they are likely to increase humidity within groundnut crops, thus promoting infection. Temperatures of 25-30°C and 6-8 hours of high humidity are needed for infection and disease development.

After harvest, collect and burn or bury the remains of the crop, and leave at least one year between groundnut crops planted on the same land, so that the remains of the old crop decompose before another crop is planted.

Control – what to do after signs are seen

Cultural approaches: Before sowing, plan to plant the new crops as far away as possible from older ones, especially if they are infected by leaf spots. If it is not possible to avoid planting near old crops, do not plant downwind from them, otherwise spores will easily spread to the new crop in wind and rain.

Chemical approaches: Inspect the crop at least once a week, and if growing the crop for sale, and fungicides are affordable and available, spray with chlorothalonil as soon as leaf spots are seen, even if they appear only on one or a few plants. Spray every 10-14 days, continuing until 14 days before harvest. Chlorothalonil controls both leaf spots and rust disease. Mancozeb can also be used. Spray more often (7-10 days) if:

- The first treatment is late and there are many plants with spots. In most cases, spraying should begin no later than 30-35 days after planting.
- Rainfall is high and disease control is poor.

CAUSE

The fungus that causes early leaf spot is *Mycosphaerella arachidis* and late leaf spot, *M. berkeleyi*. These are the sexual stages of the fungi. However, these fungi exist mostly in their asexual states, *Cercospera arachidicola* and *Cercosporidium personatum*, respectively.

Wind, rain-splash and insects spread the spores. There is no evidence that they are seed-borne. The spores germinate in water on the leaf surface, where they infect and produce more spots and spores. The life cycle takes 10-14 days.

Survival of these fungi occurs on volunteer groundnut plants, and infected crop debris.

IMPACT

Infection leads to defoliation, and in many parts of the world where fungicides are rarely used, losses of over 50% are common. Rust, caused by the fungus *Puccinia arachidis,* is now widespread, and the combination of leaf spot diseases and rust means that 6-8 applications of fungicides are needed to produce healthy crops. For most smallholders this represents a considerable investment that is unlikely to be justified economically.

DISTRIBUTION

Both early and late leaf spots are present wherever groundnuts are grown, including all parts of Africa. In Burkina Faso, Malawi, Mali, Nigeria and Sudan the diseases are widespread.

FURTHER READING AND OTHER RESOURCES

Early leaf spot of groundnut (Mycosphaerella arachidis). Plantwise Knowledge Bank. (http://www.plantwise.org/KnowledgeBank/Datasheet.aspx?dsid=35255).

Managing leaf diseases in peanuts: Fact Sheet. (http://www.pca.com.au/pdfs/GRDCfactsheet0210.pdf).

Mycosphaerella arachidis. CABI Crop Protection Compendium. (http://www.cabi.org.ezproxy.library.uq.edu.au/cpc/datasheet/35255).

[1]Note ICRISAT has developed varieties that have been selected in farmer-participatory trials in Mali, Niger and Nigeria. Improved varieties have also been released in Tanzania, Uganda and Malawi for Groundnut rosette virus resistance; nevertheless, varieties have also been screened for tolerance to leaf spot and rust. Selections have also been made in Cameroon.

Stem and pod rot of groundnut

Athelia rolfsii

Photo: Denis Persley, Department of Agriculture and Fisheries

Cottony growth on the stems of groundnut causing a wilt.

Photo: Grahame Jackson, CABI, CC BY 4.0

Sclerotia of the fungus Athelia rolfsii *on groundnut stem – at first white, later light brown.*

SUMMARY: Stem and pod rot, also called southern blight, is a fungal disease that occurs wherever groundnuts are grown. It causes up to 10-25% reduction in pod yields worldwide. Losses in Africa are not well recorded but, as it is present in more than 45 countries, they are likely to be high. The fungus is soil-borne, attacking groundnut stems just below soil level and causing leaves to yellow and wilt. As the disease develops, the characteristic thick white cottony fungus grows above ground, and round to oval, 0.5-2 mm, tan to brown sclerotia develop in it. Sclerotia are bundles of fungus with thick protective outer cells, allowing the fungus to survive for months to years in the soil depending on the conditions. The disease can be managed by crop rotation, early removal of affected plants, careful weeding and use of mulch.

KEY SIGNS

Stem and pod rot of groundnut is caused by a fungus. It has a very wide host range, attacking over 500 different species, such as legumes, solanaceous crops (tomato, potato, capsicum), cucurbits (pumpkin, squash) and many weeds.

On groundnut, the first sign of the disease is dark-brown spots on the stems just below soil level. The infection causes a yellowing and wilt of a single branch and, after a few days, the wilt of the entire plant. Often at this time the cottony growth of the fungus is obvious on the lower stem and, sometimes, over the soil surface. Groundnut pegs are also infected and the pods rot. During warm wet weather the cottony growth of the fungus spreads from plant to plant.

After about 7 days, the fungal growth begins to form sclerotia. These are 0.5-2 mm diameter tightly packed bundles of the fungus surrounded by a protective rind that is several cells thick. They are white at first and become light brown as they mature. These are the survival structures of the fungus, keeping it alive when there are no more plants to infect.

MANAGEMENT

Prevention – what to do before signs are seen

Cultural approaches: Cultural practices are very important in the management of this disease because of its wide host range, its ability to survive a long time in soil as sclerotia, the limited use of fungicides against soil pathogens[1] and the lack of resistant varieties.

If possible, avoid land with a previous history of this disease. If that is impossible, use a 2-3-year rotation between crops of groundnut on the same land; for example, rotate with maize, sorghum, cassava or yam. Never plant groundnut straight after crops of tomato, capsicum or beans, as the fungus commonly attacks those crops and it may be present in the soil.

[1]Note trials using difenoconazole (a broad-spectrum fungicide) and the fungus Trichoderma harzianum, a natural antagonist of Athela irolfii, have shown promise in South Africa.

During growth of the crop, it is important to monitor groundnut at least once a week, looking for plants that are beginning to wilt. If found, remove the plants together with the soil around the roots, taking care not to spread the fungus by dropping soil and/or sclerotia on to other plants.

Add mulch to the soil, such as corn or sorghum straw. Amendments of this kind can be useful, possibly by increasing certain organisms in the soil that compete with the stem and pod rot fungus. Combined with crop rotation this is likely to be a practical method of control for smallholders.

Remove weeds, but take care not to wound the stems to allow entry of the fungus.

After harvest, collect and remove plant debris and burn or bury it deeply. Sclerotia do not survive for more than 45 days if buried 20-30 cm beneath the surface.

CAUSE

Athelia rolfsii is the sexual state of the fungus, but it is rarely seen, in contrast to the asexual state, known as *Sclerotium rolfsii*. In addition to stem and pod rot, the disease is known as southern blight.

The fungus has a wide host range. Apart from groundnuts, it is common on carrot, beans, cucurbits, capsicum, tomato, sweet potato, soybean, taro and other aroids. Differences between isolates (from cowpea) in growth and sclerotia production have been reported from Benin and South Africa.

The sclerotia survive best when they are near the soil surface in well-drained soil. They can remain alive for several years in soil or in plant debris. As well as sclerotia, the fungus can survive between crops by growing in the remains of plants.

Spread over short distances is by the cottony growth; spread over long distances is by movement of infected plant material and infested soil. The wind can carry soil containing sclerotia.

IMPACT

Stem and pod rot of groundnuts is a common disease in many countries. Yield losses of 10-25% are said to be common. Occasionally, losses have been reported at more than 80%.

DISTRIBUTION

The fungus *Athelia rolfsii* is reported worldwide in wet and warm countries in the subtropics and tropics. It is especially common in North and South America, Central America and the Caribbean, southern Europe and Asia. The fungus has been recorded in more than 45 countries in Africa.

FURTHER READING AND OTHER RESOURCES

Mullen J (2006) Southern blight, Southern stem blight, White mold. The Plant Health Instructor. (http://www.apsnet.org/edcenter/intropp/lessons/fungi/Basidiomycetes/Pages/SouthernBlight.aspx).

Athelia rolfsii. CABI Crop Protection Compendium. (http://www.cabi.org.ezproxy.library.uq.edu.au/cpc/datasheet/49155).

Sclerotium rot (Athelia rolfsii). Plantwise Knowledge Bank. (http://www.plantwise.org/KnowledgeBank/Datasheet.aspx?dsid=49155).

Groundnut rosette disease

Groundnut rosette virus

Photo: Philip Taylor, CABI, CC BY 4.0

Groundnut with small distorted leaves and severe mosaic symptoms.

SUMMARY: Groundnut rosette disease is the most important disease of groundnuts of sub-Saharan Africa. Epidemics occur without warning. It is caused by a virus complex – two viruses and a virus-like nucleic acid molecule called satellite RNA. The satellite RNA occurs in different forms and results in at least three types of field symptoms: chlorotic (yellowing), mosaic and green rosette. These diseases differ in the patterns of yellow and green on the leaves, but all cause stunting and severe loss of pod yield. The groundnut diseases are spread by the aphid, *Aphis craccivora*. Management is mainly dependent on use of tolerant varieties. Cultural measures can also be helpful, including removing volunteer and diseased groundnut and weeds, early planting, intercropping with other legumes and cereals, and crop rotation.

KEY SIGNS

Groundnut rosette disease is not one but three diseases: chlorotic rosette, mosaic rosette and green rosette.

Plants with chlorotic rosette have bright yellow leaves, except for small parts that remain green; these are known as 'green islands'. The yellowing may affect the whole plant or only some shoots, or parts of shoots. Early infections result in severely stunted plants with small, deformed leaves.

Plants with mosaic rosette have yellow and dark green areas on the leaves. Plants are stunted, although less than those with chlorotic rosette.

Plants with green mosaic have very dark green small leaves, or they have a light and dark green mosaic, and margins that are rolled downward. They, too, are stunted if infected early. In all forms of the disease early infection causes severe pod loss.

MANAGEMENT

Prevention – what to do before signs are seen

Cultural approaches: A number of measures help to delay infection: (i) removing volunteer plants (self-sown groundnuts) from the last crop; (ii) planting at high densities, to cover the soil as quickly as possible (the landing behaviour of aphids, which spread the disease, is disturbed when the soil is covered); (iii) sowing early in order to produce a crop before the arrival of winged aphids; (iv) intercropping with beans, maize or sorghum; and (v) rotating with maize or sorghum.

The most reliable method of control, however, is to plant resistant varieties. High-yielding, long duration varieties for medium and high rainfall areas were the first to be bred. More recently short duration Spanish types, suitable for eastern and southern Africa, have become available.

In recent years, the international agricultural research centres have released rosette resistant groundnut varieties in many African countries, e.g. Ghana, Malawi, Niger, Tanzania and Uganda. A survey conducted in Uganda in the last three years found that more than 50% of the groundnut area was occupied by improved varieties. The release in 2010 of the new red-seeded rosette resistant variety, ICGV-SM 93535, will likely have stimulated increased adoption of improved cultivars in Uganda. In Malawi, improved groundnut varieties currently occupy more than 60% of the total area under groundnuts. Check if these are available from local retailers or research institutes.

Control – what to do after signs are seen

Cultural approaches: Remove rosette-diseased groundnuts as soon as they are seen, and destroy them.

Remove weeds from within and around the plots.

After harvest, collect all the plant debris and destroy it, or use it as fodder if this is usual practice in the area.

CAUSE

Groundnut rosette diseases are caused by two viruses and a particle which is just nucleic acid – ribonucleic acid – and is called a satellite RNA. The two viruses belong to different virus families. One is called *Groundnut rosette virus*, and is an umbravirus; the other is *Groundnut rosette assistor virus*, a luteovirus. *Groundnut rosette virus* needs this so-called assistor or helper virus for transmission. The satellite RNA comes in different forms, and this is the reason why there are different rosette diseases.

All three components are spread by the aphid *Aphis craccivora*. The aphid picks up the viruses and satellite RNA as it feeds on infected plants; about 18 hours later it can infect other groundnuts and continue doing so for at least 15 days. The viruses and satellite RNA do not multiply in the aphid.

Neither of the viruses causes symptoms in groundnuts when alone, or only very slight symptoms. It is the satellite RNA with its different forms that causes the symptoms. However, the satellite RNA depends on *Groundnut rosette virus* for multiplication and *Groundnut rosette assistor virus* for spread by aphids.

Only groundnuts have been found naturally infected with the either of the viruses or the satellite RNA, although other crop plants can be infected experimentally. As the viruses are not seed-borne, it may mean that volunteer (self-seeded) groundnuts are the source of infection, which is carried to crops by winged aphids. Where volunteer groundnuts do not survive the dry season, aphids on the wind may bring the viruses from others parts of the continent.

IMPACT

Groundnut rosette disease is the most important of all diseases of the crop. Although not present every year, when epidemics occur they can result in devastating losses. For example, the disease affected 0.7 million ha of groundnut in Nigeria in 1975 and caused an estimated yield loss of 0.5 million tonne valued at US$250 million. Twenty years later, in Zambia, some 43,000 ha were affected and losses for 1995-1996 were estimated to be US$5 million. Importantly, the outbreaks are unpredictable, and the sudden loss of an important source of protein, cooking oil, income, and seed for the next year results in farmers abandoning the crop. This is what happened in Malawi in 1994/95 when, following an epidemic, the area under groundnut shrank by 23%. In the 1990s, the annual loss from the disease in Africa was put at about US$155 million.

DISTRIBUTION

Groundnut rosette disease occurs throughout sub-Saharan Africa. Green rosette disease occurs in West Africa and Angola, Malawi, Swaziland and Uganda. Mosaic rosette is found only in East Africa. Reports of the disease from South America, South and Southeast Asia and Oceania are now thought to be incorrect.

FURTHER READING

Groundnut rosette virus. CABI Crop Protection Compendium. (http://www.cabi.org.ezproxy.library.uq.edu.au/cpc/datasheet/45604)

Murant AF, Robinson DJ, Taliansky ME (1998). Groundnut rosette virus. Descriptions of plant viruses. (http://www.dpvweb.net/dpv/showdpv.php?dpvno=355)

Plantwise Knowledge Bank. Groundnut leaf rust (Puccinia arachidis).

(http://www.plantwise.org/KnowledgeBank/Datasheet.aspx?dsid=45604)

Subrahmanyam P, van der Merwe, PJA. Management of groundnut rosette, past, present, and future. ICRISAT-Malawi, Chitedze Agriculture Research Station, Lilongwe, Malawi.

(http://www.icrisat.org/text/research/grep/homepage/grephomepage/archives/rosette.html

Groundnut rust

Puccinia arachidus

Photo: Mike Hughes, DAFF

Groundnut rust as it appears in the field; note that spots are mostly on the older leaves.

Photo: Mike Hughes, DAFF

Yellowing on the upper surface of leaflets (left), and pustules of rust on the underside (right).

SUMMARY: Groundnut rust is a relatively new disease to Africa, becoming widespread only since the 1970s. It affects leaves, stems and pegs, producing many small red spots or pustules containing masses of spores. Foliage turns yellow, dries up and plants die early. Infection often occurs with early and late leaf spots. When epidemics occur, pod yield losses of 40% are common. Rust spores spread over long distances by wind and also on seed, and over short distances within crops by rain-splash, and possibly by insects. Spores need water to infect. Management is primarily by removing volunteer (self-seeded) groundnut plants before planting, and by growing tolerant varieties, although keeping fields free from weeds, avoiding growing crops of different ages in the same area, site selection, rotations with cereals and possibly fungicide use are all measures to consider.

KEY SIGNS

Rust infection causes leaves to turn yellow, dry and curl, but mostly they remain attached to the plant. Once infections occur, rust develops very rapidly, faster than the diseases caused by early and late leaf spot (*Cercospera arachidicola* and *Cercosporidium personatum*). The first sign of infection is small flecks on the leaves, which develop into larger spots (or pustules as they are called) on leaves, petioles, pegs (the shoots that grow into the ground) and stems. On the leaves, the pustules are round, 0.5 to 1.5 mm diameter, and yellow at first, rapidly turning orange and then red-brown as masses of spores develop and break through the leaf surface. The pustules occur mostly on the underside of leaves, but on susceptible varieties may appear opposite them on the top surface. On the stems and petioles, the pustules are more elongate.

The rust is spread over long distances as spores during dry windy weather, but leaf wetness from rain or dew is necessary for infection, so periods of cloudy wet weather and temperatures of 20-28°C favour outbreaks of the disease. Rust spores can also be spread long distances on seed. Over short distances spread is by rain-splash and insects.

MANAGEMENT

Prevention – what to do before signs are seen

A number of measures help delay infection: removing volunteer plants from the last crop; planting new crops as far away as possible from older crops (especially those with severe rust infections) and where this is not possible avoiding planting downwind from them to avoid spread of spores from older to younger crops.

Keep weeds to a minimum; not only will weeds reduce yield of groundnuts, but also they will create conditions of high humidity around groundnut plants that favours rust infection.

There are varieties with different tolerance to rust that have been bred at ICRISAT, the International Crop Research Centre for the Semi-Arid Tropics. Since the mid-1980s, over 60 ICRISAT improved varieties have been released in 22 African countries, with some combining tolerance to rust and leaf spots. Check if these are available from local seed suppliers.

If the measures outlined are not effective or tolerant varieties are not available, rotate groundnuts with other crops to create a break from recycling rust from one crop to another. A rotation of groundnuts followed by two cereal crops, one after the other, is recommended.

Control – what to do after signs are seen

Cultural approaches: After harvest, all plant debris should be collected and destroyed, or used as fodder. The rust fungus will not remain alive in old vines or in the soil, but will survive on volunteer plants (those that are self-seeded).

Chemical approaches: Carry out regular inspections, at least twice a week, and spray when plants are first seen with infections. Do not wait until the plants are heavily infected before applying fungicide.

Begin to spray as soon as rust spots are seen, even if they appear on only one or a few plants. Spray at regular intervals: 10-14 days is best, continuing until 14 days before harvest. Spray more often if the first treatment is late, and there are many plants with rust spots. In most cases, spraying should begin no later than 30-35 days after planting. Use chlorothalonil. It is effective against rust, and also leaf spot diseases.

In an effort to reduce pesticide use, ICRISAT has been trying out integrated disease management practices based on tolerant varieties, seed dressings (using thiram plus bavistin at 2.5g per kg seed) and a single spray of chlorothalonil at 65-70 days.

CAUSE

Groundnut rust is caused by *Puccinia arachidis*. In general, rust fungi have complex life histories, with several different types of spore, some of which occur on different host plants. Groundnut rust is unusual in that there is mostly only one type of spore – urediniospores – and it occurs only on groundnuts, although another type of spore has been seen very occasionally on wild *Arachis* species in South America.

IMPACT

In many regions of the world, including Africa, rust can cause pod yield losses up to 40% when an epidemic occurs. Infected plants mature 2-3 weeks earlier than if they were healthy. In general, losses are highest when attack occurs early. In most growing areas, the disease is severe when it occurs together with early and late leaf spot diseases. Apart from loss of pod yields, there are also reductions in the amount and quality of foliage used for fodder. In Nigeria, there are reports of reduction in protein content of fodder from 16 to 12%.

DISTRIBUTION

Groundnut rust was considered a disease of South America prior to 1970, but since then it has spread around the world wherever the crop is grown. It is now common throughout sub-Saharan Africa.

FURTHER READING AND OTHER RESOURCES

Goyal A, Manoharachary (Eds) (2014) Future challenges in the crop protection against fungal pathogens. Springer New York Heidelberg Dordrecht London.

(http://books.google.com.au/books?id=iDRxBAAAQBAJ&pg=PA4&lpg=PA4&dq=cereal-cereal-groundnut+crop+rotation.&source=bl&ots=zcAd_8CGNO&sig=nnZawCNj7wFCaDd3ZdJHHQxsgwQ&hl=en&sa=X&ei=OLt8VI_1D4Xy8gWY3YHoAQ&ved=0CCgQ6AEwAQ#v=onepage&q=cereal-cereal-groundnut%20crop%20rotation.&f=false)

Oiling the wheels of groundnut production. Integrated disease management technologies in groundnut for more pods, nutritious crop residue-fodder, income, and livelihood in the Deccan plateau. Research Into Use.

(http://www.researchintouse.com/nrk/RIUinfo/PF/CPP15.htm)

Plantwise Knowledge Bank. Groundnut leaf rust (Puccinia arachidis).

(http://www.plantwise.org/KnowledgeBank/Datasheet.aspx?dsid=45745)

Puccinia arachidis. CABI Crop Protection Compendium. (http://www.cabi.org.ezproxy.library.uq.edu.au/cpc/datasheet/45745)

Anthracnose of cowpea

Colletotrichum destructivum

SUMMARY: Anthracnose of cowpea is a fungal disease affecting stems, branches, and leaf and flower stalks. Previously, it was thought to be caused by the same fungus that attacks *Phaseolus* beans, but is now considered a separate species, *Colletotrichum destructivum*. Epidemics start when infected seed is planted. Brown spots appear on the foliage and produce large numbers of spores, which are then spread by rain-splash and in wind-driven rain. The disease occurs in high rainfall parts of Latin America, Asia and Africa (especially the rainforest zone of southwest Nigeria). Important management measures involve the use of tolerant or resistant varieties, seed treated with fungicide and intercropping with cereals.

KEY SIGNS

An anthracnose fungus, *Colletotrichum destructivum*, causes a major disease of cowpea (*Vigna unguiculata*) which is grown for pods, leaves and fodder. Anthracnose means 'coal disease'; it is a term used for diseases caused by fungi that produce dark spots on leaves, petioles, stems and fruit. Anthracnose fungi are invariably associated with wet weather.

The spots are tan to brown, sunken and, on susceptible varieties, they circle the stems, branches, leaf and flower stalks. Infections on the stems are particularly damaging.

MANAGEMENT

Prevention – what to do before signs are seen

Cultural approaches: The best way to manage the disease is by planting anthracnose-resistant varieties[1]. Check with retailers before purchasing. Many varieties have now been bred for different types of resistance to anthracnose.

One option is to intercrop cowpea with other food plants to reduce the crop density and, therefore, the impact of the disease. Intercrops of cassava, millet and sorghum are possibilities. A 2-row cereal:4-row cowpea cropping system has resulted in 1-2 cowpea crops in the same season, with acceptable cereal yields.

Chemical approaches: It is very unlikely that fungicides would be economical for the control of this disease, or that they would be effective. However, if a fungicide was required on, for instance, plants grown for seed, use mancozeb, copper compounds or chlorothalonil. The appropriate times to apply fungicides are: (i) at flower set; (ii) late flowering; and (iii) pod fill.

Control – what to do after signs are seen

Cultural approaches: Do not plant next to already infected crops, otherwise the new planting will be infected at an early growth stage and the impact of the disease will be greater.

After harvest, and after collection of vines for fodder, remove or plough in debris. Leave two years before planting cowpea on the same land.

CAUSE

Since the 1990s *C. destructivum* has been accepted as distinct from another anthracnose disease caused by the fungus *C. lindemuthianum*, anthracnose of *Phaseolus* bean. Mostly, the fungi associated with anthracnose of cowpea do not infect common bean, nor lima bean, groundnut, soybean, pigeon pea, pea, chickpea, lucerne and adzuki bean in laboratory tests, or they infect only some of these. This indicates that there are different races of the fungus, and at least two distinct races occur in Nigeria.

Spores are produced in large numbers in the spots. These are spread by rain-splash to nearby plants and further afield in wind-driven rain. Humans and machinery moving through the crop, especially when it is wet, also spread the fungus.

A key feature of this disease is that the fungus infects the seeds, i.e. the disease is seed-borne. Cases have occurred

[1]Note IITA has developed varieties with combined resistance to several viruses, bacteria, fungal diseases, nematodes and insects, including anthracnose. These varieties also have improved tolerance to heat, drought, low P, and have been bred for differing times to maturity.

where up to 40% of the seeds have found to be infected. Consequently, the seeds allow survival of the fungus through the dry season and ensure that it is well placed to continue its life cycle when the rains come. Being seed-borne also provides a way for long-distance spread of the fungus.

The disease can last for at least two years in diseased stems of plant debris. It can survive both on the soil surface and buried in soil.

IMPACT

The disease is severe in high rainfall areas of Africa. It also causes economic loss in tropical regions of Latin America and Asia. In the rainforest zone of southwest Nigeria, losses up to 50% have been reported, particularly where cowpea is grown as a monocrop; however, in recent years the impact has declined with the use of resistant varieties. Similarly, losses of grain yields have been reported from India but, here too, resistant varieties have been identified although, because there are different races of the fungus, the reaction of varieties is not the same in all environments.

DISTRIBUTION

The disease is reported in North America, South America (Brazil), south Asia (India and Pakistan) and Africa (Nigeria, Uganda and Zambia).

FURTHER READING AND OTHER RESOURCES

Adegbite AA, Amusa NA (2008) The major economic field diseases of cowpea in the humid agro-ecologies of South-western Nigeria. African Journal of Biotechnology 7(25): 4706-4712. (http://www.ajol.info/index.php/ajb/article/view/59659/47946).

Allen DJ, Thottappilly G, Emechebe AM, Singh BB (1998). In: Allen DJ, Lenne JM (eds) Diseases of cowpea. CAB International, Wallingford, UK, pp 267-324.

Emechebe AM, Florini DA (1997) Shoot and pod diseases of cowpea induced by fungi and bacteria. In: Singh BB, Mohan Raj DR, Dashiell KE, Jackai LEN (eds) Advances in cowpea research. International Institute of Tropical Agriculture (IITA), Ibadan, Nigeria and Japan International Research Center for Agricultural Sciences (JIRCAS), Tsukuba, Ibaraki, Japan. IITA, Ibadan, Nigeria, pp 176-192. (http://books.google.com.au/books?id=s_5Y5BFRU1EC&pg=PA176&lpg=PA176&dq=Shoot+and+pod+diseases+of+cowpea+induced+by+fungi+and+bacteria&source=bl&ots=YxSWqd4OsV&sig=icJngYaLyqzP3VP6D9ueA9wkH6w&hl=en&sa=X&ei=fVC3U9LdHsOekwWf2ICgBw&ved=0CB4Q6AEwAA#v=onepage&q=Shoot%20and%20pod%20diseases%20of%20cowpea%20induced%20by%20fungi%20and%20bacteria&f=false).

Singh BB (2010) The quiet revolution. IITA R4D Review. Edition 5. (http://issuu.com/iita/docs/r4d_review_sep_2010).

Cercospora leaf spot of cowpea

Mycosphaerella cruenta

Photo: Howard F. Schwartz, Colorado State University, Bugwood.org, CC BY 3.0 US, http://bit.ly/1DTSR4z

First symptoms with discrete spots, reddish, not restricted by veins

Photo: Howard F. Schwartz, Colorado State University, Bugwood.org, CC BY 3.0 US, http://bit.ly/1JRVbBb

Leaf spots merge as leaves go yellow and die. Photos are of cercospora leaf spot on Phaseolus vulgaris*, common bean, which has similar symptoms to cowpea.*

SUMMARY: Cercospora leaf spot is a fungal disease. It has a widespread distribution and occurs all over Africa. It causes leaves to fall off and serious yield losses of up to 40% in cowpea. There are many resistant varieties but also susceptible ones, so care is needed in identifying suitable varieties for farmers. The disease occurs on other legumes, including closely related plants such as mung bean, 'true' beans (*Phaseolus*) and soybean. The disease is not seed transmitted but carried over to the next growing season on alternative hosts, as well as crop remains. Fungicides can be used to dress (clean) seeds and manage outbreaks. The disease is important in countries where cowpeas are widely planted, such as Nigeria and Niger. Resistant varieties appear to have limited losses in many countries but vigilance is needed to prevent future outbreaks and limit the damaging effects of this disease.

KEY SIGNS

Cercospora leaf spots of cowpea begin as small, lighter coloured areas, almost yellow. Later they become bronze to dark grey, roughly circular to more elongated and up to 10 mm across. The fungus produces masses of wind-borne spores on the lower surface of the leaf giving the spots a distinctive grey to dark powdery appearance. When held up to the light the older leaf spots are darker, more reddish and often with a distinct ring. Dead areas fall out, giving a shot-hole appearance. The leaf withers as the spots join together. Leaves die and fall off.

The fungus also attacks the stems and pods but here lesions are less prominent and damage less significant.

The leaf spots vary in shape, size and colour and could be confused with other similar symptoms. Check with a hand lens: septoria leaf spots, caused by another fungus, do not have a powdery appearance; ascochyta blight, another damaging fungus disease of cowpea, has leaf spots with circles within circles (concentric) and no powdery surface. Other fungi may sporulate on diseased leaves and give it a powdery appearance but with a different colour to Cercospora. Note the angular leaf spots restricted by veins on common bacterial blight and yellow haloes.

MANAGEMENT

Prevention – what to do before signs are seen

Cultural approaches: Much work has been done in identifying resistant varieties, the main and most important method for controlling this disease. In Sierra Leone varieties such as Slipea 1, 2 and 3 are recommended. VRB-10 is completely resistant to Cercospora leaf spot while VRB7 is highly susceptible, so care should be taken in choosing varieties to plant. Consult the local extension office or research station for advice on available cowpea varieties.

Manual seed cleaning, to remove plant debris, will prevent carry-over of the fungus and should be encouraged, given the many farmers who save seeds for the next season.

Intercropping by planting alternate rows of cowpea and another suitable non-legume crop, such as maize or sorghum, will limit spread of the disease within a field but not eliminate it.

Burying or destroying the remains of a cowpea harvest will reduce the amount of fungus able to infect new crops, as will removing alternative hosts, but these are costly and time-consuming measures which may not appeal to, or be feasible for, all farmers.

Chemical approaches: Some experts recommend seed treatment with mancozeb as a preventative measure although, given lack of evidence to support seed transmission of the fungus, the justification for this is not clear-cut.

Control – what to do after signs are seen

Chemical approaches: Fungicides are used to control outbreaks if favourable conditions or choice of cowpea varieties enable the disease to become established and the risk of major losses is high. The disease flourishes on older leaves and early monitoring of a crop is unlikely to reveal much, particularly since the first symptoms could be difficult to distinguish from other types of damage. Mancozeb should be applied after the crop has flowered and pods are starting to develop, with a maximum of 2-3 applications per planting season. It is unclear, however, whether this would be cost-effective in all circumstances.

CAUSE

Cercospora leaf spot is caused by the fungus *Mycosphaerella cruenta*. Originally there were thought to be two closely related leaf spots associated with *Cercospora canescens* and *Pseudocercospora cruenta*, but these are now considered to be the same fungus. It produces air-borne spores on the underside of the leaf and is carried over from one season to the next in left-over planting material. Seed transmission has yet to be confirmed.

The fungus has a wide host range, attacking other legumes such as 'true' beans (*Phaseolus*), soybean and bambara groundnut. These alternative hosts extend the reservoir of plants which can carry over infections to the next growing season.

IMPACT

Crop losses of between 20 and 40% occur as a result of defoliation, even though this usually occurs late in the growing season. The disease spreads rapidly through air-borne spores. The importance of Cercospora leaf spot of cowpea appears to have diminished over the last 15-20 years, presumably because of the availability of resistant varieties. But the constant presence of the disease across Africa coupled with a wide host range, the potential heavy losses and the risk of new fungal strains means that extension services should be vigilant and responsive.

DISTRIBUTION

The disease occurs around the world and is most prevalent in warmer areas of the sub-tropics and tropics. It is recorded from Sierra Leone to Nigeria and also Niger. Also present from Sudan to South Africa, Cercospora leaf spot is likely to occur in all countries in Africa where cowpea is grown. The apparent absence of the disease from several countries (e.g. Kenya, Burundi) could indicate low incidence or lack of published records.

FURTHER READING

Plantwise (www.plantwise.org)

Crop Protection Compendium online (www.cabi.org/cpc).

Regular international conferences are held on cowpea. Earlier proceedings and papers have been published as books. The most recent conference in 2010 has abstracts available online (http://cowpea2010.iita.org). Check this website and that of IITA (www.iita.org) for current information about cowpea generally.

Much of the information about cowpea diseases comes originally from a chapter in Allen et al's excellent book on diseases of food and pasture legumes, though there have been important scientific advances since its publication in 1998, particularly in breeding.

Allen, DJ, Lenné JM (editors) 1998. The Pathology of Food and Pasture Legumes. CAB International, Wallingford.

Singh B, Mohan Rah DR, Dashiell KE, Jackai Len (editors) 1997 Advances in Cowpea Research. IITA, Ibadan, Nigeria.

Mosaic diseases of cowpea

Multiple viruses

Photo: Eric Boa, CABI, CC BY 4.0

Advanced, non-specific mosaic symptoms: this could be one of several different viruses.

Photo: Eric Boa, CABI, CC BY 4.0

Mosaic viruses can also reduce and distort growth – note the rough surface of smaller infected leaves.

SUMMARY: There are several viruses associated with mosaic symptoms on cowpea. The two most important ones in Africa are blackeye cowpea mosaic virus (BlCMV) and cowpea aphid-borne mosaic virus (CABMV). There are at least seven other viruses which infect cowpea in Africa, including some that produce a mottling of leaves similar to a mosaic. More than one virus can be present and additive effects increase the damage to cowpea and subsequent yield losses. The control of viruses in cowpea is fundamentally about prevention: use of resistant varieties and healthy seed. Both BlCMV and CABMV are seed-borne and transmitted by aphids. The opportunities for vector control are limited and only effective in the early stages of symptom development.

KEY SIGNS

Mosaic refers to a patchwork of irregular light-coloured areas scattered across the surface of the normal green leaf. Mosaic symptoms are easier to see with the leaf held against the light. The discoloured areas in a leaf mosaic disease have a clearly defined boundary corresponding to leaf veins and differ from mottling, caused by different viruses on cowpea, where similar areas are not defined by veins. Also look out for leaves that are smaller than expected. They may have a wrinkled appearance and loss of colour.

In the field it will be difficult to differentiate mosaics and mottling caused by different viruses (or combinations of them). All cowpea viruses will affect the growth and development of the plant though not all pose a serious threat. CABVM can cause severe losses by itself or together with other viruses. Both CABMV and BlCMV, and other viruses on cowpea are seed borne. CABMV and BlCMV are transmitted by aphids, as is cowpea mosaic virus (CPMV). Other viruses are transmitted by beetles or by whitefly.

Laboratory testing is essential if the precise identification of viruses is required, for example in phytosanitary regulation. Good general advice on control can be given, however, based on a reliable field diagnosis of general virus attack. Leaf mosaic symptoms are relatively easy to detect, though their importance as an indicator of reduced yields may not be fully appreciated by farmers, let alone extension agents.

MANAGEMENT

Prevention – what to do before signs are seen

Cultural approaches: The threat from cowpea viruses is constant in all countries where cowpea is grown. The best and most effective way to reduce damage is to plant cowpea varieties with known resistance to the main virus diseases, such as BlCMV and CABMV. Ask for advice from research stations and seed sellers on available varieties and their disease resistance.

Farmer-saved seed is a potential source of new infections. Seeds should be taken only from healthy plants, those which lack obvious virus symptoms, particularly leaf mosaics and mottling.

Removal of weeds which harbour aphids or other insect vectors is recommended but has to be thorough and done every season to influence disease spread.

Avoid planting in the dry season when plants may be more stressed and aphid vectors, for example, more likely to feed on plants.

Chemical approaches: Vector control will reduce the spread of the disease but if seeds are already infected it is unlikely that reducing insect populations will have much effect. Insecticides can be used to reduce if not eliminate potential vectors but this must be done before populations increase and at the early stages of the insect life cycle.

Apply cypermethrin or dimethoate, by themselves or in combination, early in the morning or late in the evening, no more than twice in the growing season. Botanical preparations containing, for example, leaf extracts of papaya, neem or other plants with known activity against aphids can also be used.

It is important to stress that the cost of buying, preparing and applying natural and synthetic insecticides at optimal times during the early growth of cowpea plants should be carefully weighed against the expected gains in yields. Vigorously growing cowpea plants of varieties resistant to viruses (and their vectors) are the best method for limiting yield losses.

Control – what to do after signs are seen

There are no treatments for plants that already have mosaic disease.

CAUSE

The two most important mosaic-inducing viruses of cowpea in Africa are blackeye cowpea mosaic virus (BlCMV) and cowpea aphid-borne mosaic virus (CABMV). Scientific advances in characterising plant viruses has led to many changes in original names and BICMV is now officially bean common mosaic virus (blackeye strain), but this new name is still uncommon in publications. BlCMV may also appear as cowpea (blackeye) mosaic virus.

Other viruses of importance are cowpea mosaic virus (CPMV) also described incorrectly as cowpea yellow mosaic virus. Both names are used in publications.

Cowpea mottle virus occurs in West Africa and cowpea mild mottle virus in East Africa also, but little information about their importance has been found.

Nine different viruses have been recorded on cowpea in Africa and multiple viruses sometimes co-infect plants.

IMPACT

Although there is limited data on yield losses there can be little doubt that virus infections, particularly those associated with BlCMV and CABMV, will seriously affect production. The reduction in area of healthy leaves together with smaller sized plants will inevitably affect pod and bean production, particularly when at least one of these two major viruses has spread to whole fields.

DISTRIBUTION

CABMV has been studied more than BlCMV and has been recorded in 16 sub-Saharan countries in Africa, including Sierra Leone, Ghana, Nigeria, Kenya, Tanzania, Uganda, Zimbabwe and South Africa.

BlCMV has an apparently narrow distribution, with confirmed records from Burkina Faso, Ghana, Nigeria, Kenya, Botswana and Zambia. However, it is likely that BlCMV is present in other countries and that the threat of virus infection on cowpea is both widespread and constant.

FURTHER READING

Crop Protection Compendium online (www.cabi.org/cpc).

Plantwise (www.plantwise.org).

PMDG on aphids that transmit cowpea mosaic virus.

http://www.plantwise.org/FullTextPDF/2013/20137804290.pdf

Striga or witchweed (multiple crops)

Striga species

Photo: USDA APHIS PPQ Archive, CC BY 3.0 US, www.bugwood.org

Striga hermonthica *flowering on maize.*

Photo: Rob Williams, CABI

Photo: International Institute of Tropical Agriculture, Flickr, CC BY-NC 2.0, http://bit.ly/1JvujUT

Striga gesnerioides *parasitizing roots of cowpea (left); flowers of Striga hermonthica (right).*

SUMMARY: *Striga*, or witchweed, are parasitic weeds which infest millions of hectares of land planted to maize, sorghum, millets, upland rice, cowpea and sugarcane, reducing yields by 30-100%. There are many species in Africa, growing mostly in arid regions of low soil fertility, but four dominate: *S. hermonthica, S. asiatica, S. aspera* and *S. gesnerioides*. Witchweeds tap into the xylem of host plants causing yellowing, stunting and wilting. Seeds are tiny and can spread over long distances, probably in wind-blown soil and over shorter distances in rain run-off, on shoes and hooves of livestock. Management depends on use of resistant varieties and cultural control measures, including crop rotations, weeding, raising the fertility of soils and the use of trap crops.

KEY SIGNS

The common species of witchweed in Africa are *S. hermonthica, S. asiatica, S. aspera* and *S. gesnerioides.* They parasitise cowpea, maize, sorghum, millet, sugarcane, upland rice and wild grasses. Affected plants are stunted, yellow, scorched and wilted, symptoms which are similar to nutritional deficiencies or drought. Although it is sometimes difficult to distinguish between symptoms caused by witchweed and those caused by other environmental factors, if plants wilt when the soil is still moist then this is a sign that witchweed is a likely cause.

MANAGEMENT

Prevention – what to do before signs are seen

Cultural approaches: Host resistance together with cultural control measures offer the best methods. There are now several crops bred for resistance/tolerance. For instance, there are sorghum varieties resistant to *S. asiatica* (released in Tanzania in late 2003) and varieties of maize, upland rice and sugarcane resistant to the same species. Resistance in maize has been more difficult to attain, but varieties are now available. There are also cowpeas resistant to the strains of *S. gesnerioides* in West Africa. There are as yet no witchweed-resistant millet varieties.

Control measures for *S. aspera* are generally comparable to those for *S. hermonthica*, but there are differences in varietal susceptibility and resistance: maize hybrids tolerant of *S. hermonthica* are susceptible to *S. aspera*, while rice varieties resistant/tolerant to *S. hermonthica* are also resistant/tolerant to S. aspera.

Recommended cultural measures to prevent witchweed include: improving soil fertility by, for example, rotations or intercropping with leguminous crops and use of cattle or green manure; delaying planting until seasonal rains have set in to reduce host plant water loss; and use of trap crops. Trap crops stimulate witchweed seed germination, but do not become infected so the witchweed dies, e.g. cotton, cowpea, pearl millet and soyabean are trap crops for *S. asiatica*.

Chemical approaches: Coating maize seed resistant to the herbicide imazapyr (IR maize) with the same chemical has shown promise against S. asiatica and S. hermonthica in Kenya, especially as the maize can be interplanted with legumes, as long as they are at least 15 cm away from the treated seed.

Control – what to do after signs are seen

Cultural approaches: Hand pulling of the larger species, such as *S. hermonthica*, if populations are low, but not *S. asiatica*, which is much smaller and has seeds that mature and are shed faster.

Chemical approaches: There are a number of herbicides that can be used, but in terms of cost and effectiveness, 2,4-D is possibly the best for cereal crops. The disadvantage is that more than one application may be needed and it can damage broad-leaf (e.g. leguminous) crops, if these are intercropped.

CAUSE

Most species of witchweed have bright green leaves and stems 30-100 cm high, with bright flowers and poorly developed roots. *S. gesnerioides* is different – it is totally parasitic, with small, unexpanded leaves and a cluster of fleshy, pale-green or yellow shoots, 10-20 cm high.

Witchweed seeds are tiny: each plant produces hundreds of thousands. They remain viable in the soil for many years, germinating in response to chemicals from the roots of potential host plants. The radicle fastens to a root, penetrates it, growing into the xylem, taking water, minerals, sugars and amino acids needed by the parasite until it emerges and starts to photosynthesise; however, it remains dependent on the host for food, as its ability to photosynthesise is poor. In addition, chemicals that regulate plant growth pass from the parasite to its host. These are chemicals that stunt the growth of shoots, but stimulate growth of roots, all to the advantage of the parasite.

Witchweed seeds are probably spread long distances on the wind, along with soil. Spread over short distances occurs in run-off during heavy rains, on the feet, tools and machinery of farmers, and via the hooves and gut of livestock. There is concern that some species are continuing to spread in Africa.

Witchweeds get their name because symptoms occur before the appearance of the parasite above ground. Because they need a host for germination and early development scientists call them hemiparasites.

IMPACT

The effect of witchweed can be devastating, especially to food crops – legumes, maize, millet, sorghum, sugarcane and upland rice – grown in some of the poorest farming systems of Africa. It is estimated that witchweed affects 40% of the arable savannah region, resulting in losses of up to US$13 billion per annum.

S. hermonthica is responsible for greater crop loss in Africa than any other weed. It is widespread throughout most of the continent, attacking maize, millets, sorghum, sugarcane, upland rice and wild grasses. Estimates of crop losses on sorghum are: Ghana (21%), Nigeria (10%), Gambia (8%) and Benin (6%), but there are also undefined losses in Burkina Faso, Cameroon, Cote d'Ivoire, Ethiopia, Kenya, Mali, Niger, Senegal, Sudan, Togo, Tanzania and Uganda. For maize, it is estimated that 4 million hectares of land is infested, with losses of 30-80%, valued at US$380 million to US$1 billion.

S. asiatica is a serious weed of sorghum, maize, millets, rice, sugarcane, upland rice and many wild grasses. Over one million hectares in Malawi and 250,000 hectares in Angola are infested. In Malawi, average crop losses in maize were put at 28% in infested fields and 4.5% for the country as a whole. Like *S. hermonthica*, it is severe where crops are grown under marginal rainfall and low soil fertility, over a wide range of soil types. In Tanzania the weed is associated with mainly sandy soils. Climate change could assist its further spread in arid regions of North Africa.

S. aspera attacks the same range of crops as *S. hermonthica*, except that it attacks maize more than sorghum, and can parasitise irrigated rice. Where the two species occur together, S. aspera emerges and matures more rapidly.

S. gesnerioides is a serious weed of cowpea across West Africa. Surveys in northern Nigeria and Burkina Faso have shown that farmers rate this weed as highly damaging, and trials have recorded yield losses of 30 to more than 50%.

DISTRIBUTION

Witchweeds occur naturally in Africa, Asia and Australia. S. hermonthica is widespread throughout Africa, but is more common in west, east and central parts of the continent, commonly associated with both sandy and clay soils of low fertility, especially those low in nitrogen. It predominates in savannah regions associated with cereals.

S. asiatica is widespread in East Africa and occurs in numerous countries (as well as in Asia): Togo, southern Ethiopia, Tanzania, Botswana, Malawi, Mozambique, Namibia, South Africa, Swaziland, Uganda, Zimbabwe and Zambia. It occurs mostly in sandy soils. *S. aspera* has a similar distribution to S. *hermonthica*, but is much less common in eastern Africa. *S. gesnerioides* has wide distribution in Africa from north to south of the continent, but only in western Africa (Senegal, Mali, Togo, Burkina Faso, Ghana, Nigeria, Niger, Cameroon and Chad) is it a serious problem on cowpea.

FURTHER READING

Striga asiatica. CABI Crop Protection Compendium: http://www.cabi.org.ezproxy.library.uq.edu.au/cpc/datasheet/51786

Striga aspera. CABI Crop Protection Compendium: http://www.cabi.org.ezproxy.library.uq.edu.au/cpc/datasheet/51848

Striga gesnerioides. CABI Crop Protection Compendium: http://www.cabi.org.ezproxy.library.uq.edu.au/cpc/datasheet/51785

Striga hermonthica. CABI Crop Protection Compendium: http://www.cabi.org.ezproxy.library.uq.edu.au/cpc/datasheet/51849

6. The major pests and diseases of root and tuber crops

This section covers the most important pests and diseases that affect cassava, sweet potato, yam and potato in Africa.

Table 6.1 summarises the main signs associated with the most important pests and diseases of cassava in Africa.

Table 6.2 summarises the main signs associated with the most important pests and diseases of sweet potato in Africa.

Table 6.3 summarises the main signs associated with the most important pests and diseases of yam in Africa.

Table 6.4 summarises the main signs associated with the most important pests and diseases of potato in Africa.

In the following pages, more details on the signs, management options, cause and impact are provided.

Table 6.1: Key signs of the most important pests and diseases of cassava in Africa.

Key signs	Possible pest or disease	See page
Yellow spotting on leaves; leaves can become mottled and die. No leaf distortion. Mites feeding on underside of leaves and green stems appear as tiny greenish-yellowish spots. In severe attacks, newest leaves die and fall off and shoot tip looks like a 'candle stick'.	Cassava green mite	89
Only seen in one location in Uganda so far, but a disease to look out for. Yellowish leaves, shortening of internode and slight stunting. The weed sticky mallow (*Malvaviscus arborus*) growing nearby may have little leaf, leaf yellowing and deformation.	Phytoplasma diseases of cassava	99
On older or mature leaves, patchy yellowing of leaves along the thin secondary veins that branch off from the main central vein. These narrow areas can spread and form bigger blotchy patches. No leaf distortion. Dry rot of the tubers: yellow-brown, corky death in localized areas. Sometimes tubers appear like a series of rings stuck together.	Cassava brown streak disease	101
New shoots wilt and the stem dies back. Small dark-green to blue leaf spots: minor veins initially restrict development of spot, which is angular and water-soaked; later, spots enlarge and join up, often along the leaf edge. Yellow patches spread outwards from leaf spots as leaf withers and droops. Sticky ooze containing bacteria on stems and from infected leaves. Dark areas develop along the leaf stalks and woody stems. Top of the growing shoot dies, giving 'candle' appearance.	Cassava bacterial blight	103
Patchy yellowing of distorted leaves. Characteristic white threads (hyphae) on the outside of the tuber may be present.	Cassava mosaic disease	105

Table 6.2: Key signs of the most important pests and diseases of sweet potato in Africa.

Key signs	Possible pest or disease	See page
Larvae are chunky white legless grubs, 5-10 mm long and slightly curved; the head is brown or light yellow. Larvae feed on the lower base of the stem leaving small tunnels: the stems can turn yellow, crack and wilt. Later, larvae bore tunnels in storage roots that can rot becoming brown, soft and spongy: damaged roots have bitter taste and bad smell. Adult weevils are black and shiny blue with long slender bodies (6-8 mm long), a long snout with antennas on both sides. Before storage roots are formed, adults are found on leaves and vines, leaving small round feeding holes.	Sweet potato weevil	91
Small dark brown to black oval spots occur on the fully-grown leaves in a pattern of rings: grow up to 5 mm in diameter, frequently join together and are often surrounded by yellow haloes. Underside of leaves veins turn black. Leaves turn yellow and fall off: in severe cases a carpet of dead blackened leaves covers the soil. On petioles and stems spots grey at first, later becoming black and sunken.	Leaf petiole and stem blight disease of sweet potato	107
Yellow, narrow leaves, often with deformed edges. Vines are severely stunted. Yields of storage roots are generally low.	Sweet potato virus disease	109
Rats dig through the mounds or ridges to eat tubers, or attack them when they are exposed above ground . Mole rats burrow into the soil, eating storage roots from below.	Rats and mole rats	119

Table 6.3: Key signs of the most important pests and diseases of yam in Africa.

Key signs	Possible pest or disease	See page
Feeding signs on setts and tubers leaving small round holes, 1-2 cm deep. Tubers rot, leading to significant post-harvest losses. Plants can wilt and die. Adult beetles are blackish-brown in colour and shiny; the greater yam beetles are 23-33 mm long and have two prominent lumps on their heads, and lesser yam beetles are smaller at 21-23 mm.	Greater and lesser yam beetles	93
Caterpillars bore into the tubers during harvest and storage leaving tunnels. Signs of caterpillars include black frass (faecal matter) woven together by silk threads and pupa cases that are left behind after the adult emerges.	Yam moths	95
On young leaves, small dark brown spots with yellow margins that expand rapidly as leaves approach full size. Sometimes large irregular blotches form; 'shot-holes' occasionally develop. On more resistant varieties, only the young leaf veins are infected; in this case the leaves become cup-shaped as they grow. On older leaves, pinpoint spots are present that do not expand. During epidemics, mature leaves and stems can rapidly turn black, which can be mistaken for lightning strike.	Yam dieback	111
Feeding nematodes produce cavities in tubers which become darker as the rot progresses, normally to a depth of less than 2 cm. Cracks appear in the skin, which becomes crinkly and parts flake off revealing the dark rot beneath. Rots continue in storage and can lead to complete decay of the tube. No above ground symptoms. External symptoms not always obvious on the tubers; rot is only seen when the skin is scraped away.	Dry rot of yam	113
Commonly, plants show yellow and green patterns, called mosaics: mostly between the veins or in narrow green strips bordering them. In more severe cases, the foliage shows shoe-string symptoms: leaves are long, thin and strap-shaped, and the plants may also be stunted. Often there is apparent recovery of some plants soon after infection when symptoms disappear but those infected may be slower to sprout and show poor vigour.	Yam mosaic virus	115

Table 6.4: Key signs of the most important pests and diseases of potato in Africa.

Key signs	Possible pest or disease	See page
Small caterpillars: yellowish-white to reddish-brown, dark brown to black head, black rows of short hairs running along backs. Fully grown caterpillars 35-40 mm, dark brown, yellow-green, to red-brown, with grey-black stripes along the side.	Cotton bollworm	97
Irregularly shaped small leaf spots, pale to dark green, spread rapidly, unrestricted by the leaf veins. Browning of veins on the lower leaf surface. Leaves dry up and go brown. Pale green to yellow halos develop around the dying areas Plants killed rapidly. Black or brown lesions on stems and leaf stalks. Infected tubers go brown outside and rot inside.	Late blight of Irish potato	117

Cassava green mite

Mononychellus tanajoa

Photo: Eric Boa, CABI, CC BY 4.0

Cassava green mite adult and egg.

SUMMARY: The cassava green mite is an important pest of cassava in Africa that can cause significant losses in yield. The mite can easily be spread from one place to another on leaves and cuttings of the plant, and by wind. An integrated approach is required to control the pest and reduce the damage. Cultural practices, such as using clean planting material, planting early in the rain season and intercropping with pigeon pea, can reduce populations of the pest.

KEY SIGNS

The cassava green mite is green to yellowish in colour and can barely be seen by the naked eye (they look like tiny greenish-yellowish spots). The mites feed on the underside of young leaves and green stems of the cassava plant. Use a hand lens to look for cassava green mites on the underside of leaves, along the veins and close to the base of the leaf. Mite populations increase on the young leaves in the early part of the dry season.

Mites attack the plant by sucking fluids out of the cells of the plant tissue, which causes chlorotic spots (yellow spotting) to appear on leaves due to the loss of chlorophyll (green pigment). The leaves may become mottled and die. These symptoms can be confused with cassava mosaic virus, but cassava mosaic virus causes larger greenish-yellowish patches and leaf distortion. A severe attack by cassava green mite causes the terminal (or newest) leaves to die and fall off, and the shoot tip to look like a 'candle stick'.

MANAGEMENT

Prevention – what to do before signs are seen

Cultural approaches: Use resistant varieties if they are available in the region.

If resistant varieties are not available, ensure clean planting material is used. Inspect the fresh sprouts on the cassava cuttings closely for mites and destroy any infested planting material.

Plant early, at the start of the rainy season, to encourage leaf growth and plants that are able to survive an attack. Cassava plants between 2 and 9 months old are the most vulnerable.

Intercrop with pigeon pea, in double or triple rows, to reduce the damage and also increase yields.

Control – what to do after signs are seen

Cultural approaches: The cassava green mite has a number of natural enemies that can be used for biological control, including predatory mites *Typhlodromalus manihoti* Moraes and *Typhlodromalus aripo* DeLeon, as well as many predatory insects from the *Stethorus* and *Holobus (=Oligota)* genera. There have been successful country-wide programmes to release these predatory mites and insects in several countries in Africa.

During the season, leaves that show signs of mites should be removed and destroyed away from the field. After harvest, destroy all infested crop residues.

Chemical approaches: Use of chemicals by smallholders is often not possible due to the cost. Furthermore, pesticides reduce the population of natural enemies which can cause the mite populations to increase rapidly.

CAUSE

The cassava green mite, *Mononychellus tanajoa*, belongs to the Acari family of mites. It has no wings or antenna. Adult females can grow up to 0.8 mm and are bigger than the adult males.

In development, there are four active instars including a six-legged larva, two nymphal stages (proto- and deuto-nymphs) and the adult stage. Development time from egg to adult takes approximately 11 days. The females can live up to 30 days, laying an average of 60 eggs over a 9 day period.

Temperature is the factor that has the greatest impact on development and growth rate of the mite. The higher the temperature the faster the growth rate: a female mite can produce on average 4.0 to 4.5 eggs per day at temperatures between 27°C and 34°C.

The mite is easily spread by the wind and through infested plant materials such as leaves (eaten as a vegetable), cuttings (used for propagation) and root tubers. Mites can survive on leaves, stems and cuttings removed from the field for a period of up to 60 days.

IMPACT

The cassava green mite is a major pest of cassava throughout Africa. It can severely reduce yields by reducing the leaves and lowering the photosynthetic activity of the plant. Due to the reduced plant growth, fewer leaves are available for harvest as green vegetables and yield losses of the tubers can range from 10-80%. Prolonged periods of drought can lead to an increase in the population of mites and further reduced yields.

DISTRIBUTION

Native to the neo-tropical areas of Brazil and South America, the cassava green mite was introduced into Uganda in the 1970s and has since spread throughout Africa.

FURTHER READING AND OTHER RESOURCES

Plantwise Knowledge Bankwww.plantwise.knowledgebank.org

Integrated Cassava Project http://www.cassavabiz.org/production/mites.htm

Onzo, A., Hanna, R. & M.W. Sabelis. Biological control of cassava green mites in Africa: impact of the predatory mite Typhlodromalusaripo. EntomologischeBerichten, 65(1), 2005.

Sweet potato weevil

Cylas spp.

Photo: Edwin M Escobar, Flickr, CC BY-NC 2.0, http://bit.ly/1yY5kTM

Adult sweet potato weevil (body length 6-8 mm).

Photo: Scot Nelson, Flickr, CC BY-NC-SA 2.0, http://bit.ly/1HUqI1j

A cut sweet potato showing larvae in tunnels.

SUMMARY: Sweet potato weevils are insect pests that can cause severe damage to sweet potatoes. The larvae cause the most damage by feeding on the stems and storage roots. When weevil populations are high, more than one preventive method should be used such as: use of clean planting material, planting early in season and/or using early maturing varieties, selecting deep rooting varieties or earthing up the soil around the base of the plant, removal of crop residues and volunteer plants, and crop rotation.

KEY SIGNS

The larvae cause the most damage by feeding on and tunnelling into the stems and storage roots. Adult weevils also cause damage by feeding on the leaves, vines and underground storage roots. When the plants are young, before the storage roots form, the larvae mainly feed on the lower base of the stem, leaving small tunnels. In heavy infestation, the stems turn yellow, crack and wilt. Damage to the stems can result in a significant loss in yield and cause young seedlings to die. Larvae bore into the storage roots, leaving tunnels that can rot becoming brown, soft and spongy. An infestation of the storage roots can make them inedible because the damaged tissue has a bitter taste and bad smell, lowering the quality and value of the sweet potato.

The weevils' eggs are yellowish-white, shiny and oval shaped. The larvae are chunky white legless grubs, about 5-10 mm long and slightly curved; the head is brown or light yellow. The pupae are about 5-6 mm long and initially creamy white but become grey as they get older. The adult weevils are black and shiny blue with long slender bodies (6-8 mm long), and a long snout with antennas on both sides.

Monitoring for the weevil early in the crop season is important. The adults can be difficult to detect because they are mostly active at night; during the day they can be found hiding under leaves or in cracks in the soil. Early in the season, before the storage roots are formed, the adults are often found on the plant leaves and vines, leaving small round feeding holes; if disturbed they will drop to the ground and hide. Inspect the base of the stems for feeding holes and larvae tunnels, and look for signs of wilting or swelling. Cut open the stems of plants showing symptoms and look for tunnels filled with larvae, droppings (frass) and adult weevils. If damage to the stems is detected, dig up the roots of a few plants and inspect the surface of the roots for deep feeding punctures and cavities plugged with frass, and inspect the tunnels for larvae. Pheromones for all three *Cylas* species can be used, but they are expensive and not widely available. Trials are being conducted to study the use of pheromones for mass trapping in East Africa. Homemade traps can be made using local materials to reduce costs.

MANAGEMENT

Prevention – what to do before signs are seen

Cultural approaches: To reduce the possibility of bringing weevils into a newly planted area, it is important to select clean,

uninfested cuttings for planting the new crop. Clean vine-tips are preferred, especially the tender tips (the top 25-50 cm), because weevil eggs and larvae are more likely to be found in the old woody parts of the vines. Using a pre-plant dip to treat the cuttings can provide control for the first few months of the growing season. Dip the cuttings into a solution of the fungus *Beauveria bassiana* for 10-15 minutes prior to planting.

Select deep rooting varieties with long necks as these are less vulnerable: adult weevils cannot burrow below 1 cm. Varieties that grow and develop roots near the soil surface are more likely to create cracks in the soils where the weevils can access the roots.

Plant early in the season, or early-maturing varieties, so that harvest can be completed before the dry season when the soil will dry and crack.

Build up the soil around the base of the plant with a hoe to create a small hill or mound and re-ridge about 30 days after planting. This buries the roots deeper and minimises cracks in the soil where the weevils can enter. Straw or plastic mulches can also provide cover and should be applied soon after planting.

If possible, irrigate regularly to prevent the soil from drying and cracking.

Remove alternative host plants, such as morning glory, water spinach and wild Ipomoea, from in and around the field.

Remove and destroy crop debris and volunteer plants immediately after harvest.

If possible, do not plant sweet potato in the same field year after year, and plant at least 1 km away from infested fields (weevils cannot easily fly from field to field). Rotating crops with non-host plants can significantly reduce the populations; if the weevils have nothing to feed on they will die. Rotate with a non-host crop, such as sorghum or rice, for 2 or 3 seasons. If rotation is not possible, an alternative is to flood the field for 24 hours after harvest to drown and kill the weevils.

Sweet potato weevils can continue to cause damage during post-harvest storage. Do not store roots that show symptoms with non-infested roots.

Control – what to do after signs are seen

Chemical approaches: Chemical control is difficult with sweet potato weevils because the larvae feed on the storage roots in the ground or inside the woody base of the stems. There are many natural enemies of the sweet potato weevil, including ants (predator of the eggs), earwigs, ground beetles and spiders. Minimising the use of chemicals will help preserve these natural enemies.

CAUSE

There are three main species of *Cylas* that are present in Africa, *Cylas puncticollis, Cylas brunneus* and *Cylas formicarius*, all of which are commonly known as the sweet potato weevil. Other names include the sweet potato root borer or the African sweet potato weevil.

In addition to sweet potato, host plants include coffee, morning glory, sesame, cowpea and maize.

All three species have a life cycle that includes four stages: egg, larva, pupa and adult. After mating, the adult female weevils will lay eggs one-by-one in small cavities at the base of the stems or storage roots and seal the cavity with grey frass. Female weevils do not have the ability to dig; they enter the soil through cracks in the soil surface to find the storage roots. The larvae (or grubs) hatch after 3 to 7 days and start feeding on and tunnelling into the storage roots and stems. The larval stage lasts 2-3 weeks depending on the temperature. The larvae begin pupation in the tunnel where they have been feeding. The pupa stage lasts about 3 to 7 days, depending on the conditions. The adult emerges from the storage root or stem to search for a mate. The total life cycle, from egg to adult, takes about 33 days under favourable weather conditions (dry and hot temperatures between 27° and 30°C).

IMPACT

The sweet potato weevil is one of the most important pests of sweet potatoes. The larvae can kill young seedlings by boring into the stems and cause severe damage by boring into the storage roots of older plants. Damaged storage roots develop a bitter taste, lowering their quality and economic value.

DISTRIBUTION

Cylas species are present in tropical and subtropical regions worldwide. *Cylas puncticollis* and *Cylas brunneus* are the two species most commonly found in sub-Saharan Africa. *C. puncticollis* is one of the major pests of sweet potato in tropical Africa, particularly Uganda, Rwanda, Kenya and Cameroon. *C. brunneus* is native to West and Central Africa and present in Burundi, Ivory Coast, Ghana, Kenya, Nigeria, Rwanda, Sierra Leone, Togo and Uganda. *Cylas formicarius* is also present in several countries, notably South Africa and the coastal regions of Kenya.

FURTHER READING

Plantwise Knowledge Bank www.plantwise.org/knowledgebank

Biovision Infonet http://www.infonet-biovision.org/default/ct/97/pests?search=sweet%20potato

T. Ames, N.E.J.M. Smit, A.R. Braun, J.N. O'Sullivan, and L.G. Skoglund. 1996. Sweetpotato: Major Pests, Diseases, and Nutritional Disorders. International Potato Center (CIP). Lima, Peru. 152 p.

Greater and lesser yam beetle

Heteroligus meles and *Heteroligus appius*

Photo: G. Georgen, IITA

An example of Heteroligus meles.

Photo: Dr Stephen Kwaku Asante, CSIR-Savanna Agricultural Research Institute.

Tuber damage caused by the yam beetle.

SUMMARY: Greater and lesser yam beetles are both important pests of yams in West Africa. The adult beetles burrow into the soil and feed on the tubers. Preventive methods, such as planting as late in the season as possible and treating planting material with an insecticide before planting, are the best ways to control yam beetles and reduce the damage they cause.

KEY SIGNS

Greater and lesser yam beetles are major pests of yams in West Africa. The adult beetles feed on the yam planting material (setts) as well as the tubers, starting just after planting and continuing until harvest. The beetles leave small round holes about 1-2 cm deep which allow secondary disease infections and cause the tubers to rot, leading to significant post-harvest losses. If the attack is severe it can cause the plant to wilt and die.

The adult beetles of both species are blackish-brown in colour and shiny; the greater yam beetles are 23-33 mm long and have two prominent lumps on their heads, and lesser yam beetles are smaller at 21-23 mm. The larvae are creamy-white to grey grubs with a curled body and a light brown head.

Light traps can be used for monitoring the adult beetles.

MANAGEMENT

Prevention – what to do before signs are seen

Cultural approaches: Timing of planting is the most important preventive measure available. Planting as late in the season as possible can significantly reduce the damage caused by the beetles. A study in the Nigerian delta showed that planting as late as June or July significantly reduced the damage caused by the yam beetles.

If possible, yams should not be planted near wet areas, along rivers, creeks, or tributaries where the beetles breed.

The white Guinea yam variety Tamenyo is reported to be significantly less prone to beetle damage compared to the Amula, Pepa and Ogoja cultivars.

Mulching with the leaves of lemon grass (*Cymbopogon citrates*) or mosquito plant (*Ocimum viride*) can increase yields and reduce the damage caused by the yam beetle. These plants are also believed to have properties that repel the yam beetles.

Chemical approaches: Treating setts with insecticides before planting, and applying a post-sprouting treatment, can significantly reduce the damage caused by yam beetles. Insecticides such as dieldrin, endosulfan, carbofuran or aldrin dust have been used in the past to treat planting material; however, these chemicals are extremely toxic to people, livestock and the environment. They have been banned in many countries and should not be recommended to farmers for use. Furthermore, one study reported resistance to aldrin.

Safer sett treatments are:

- Pirimiphos-methyl and deltamethrin. These can be either sprinkled on the setts as a powder or used for dipping (after mixing with water). As with all pesticides, label instructions for dosages and safety precautions must be followed. Only pesticides registered for the intended use in the relevant country should be used.
- Wood ash is a safer and more cost effective alternative. Studies have shown that wood ash can be just as effective as chemical pesticides against yam beetles. Wood ash can be sprinkled as a powder on yam setts just prior to planting. Alternatively, mix two parts wood ash with five parts water and immerse the setts in this mixture for 20 minutes, then remove the setts and dry them under shade before planting.

Post-sprouting treatments:

- Neem extract, 5% concentration, applied once per week for three weeks, starting 12 weeks after planting.
- Insecticides, such as pirimiphos-methyl or deltamethrin, applied once per week for three weeks, starting 12 weeks after planting.

CAUSE

There are two main beetle species that attack the yam in Africa: the greater yam beetle, *Heteroligus meles*, and the lesser yam beetle, *Heteroligus appius*. The greater yam beetle is bigger and more widespread, but the signs are similar for both. Yams are the primary crop the beetles attack; alternate hosts include the roots of grasses, bananas and coffee grown in marshy areas.

During the dry season, the greater yam beetle breeds near wet areas such as river basins, where they lay their eggs in the moist soil. When the eggs hatch the larvae start feeding on the roots of grass and other debris in the wet areas. After pupation, at the start of the rainy season when the yams are planted, the adults start to emerge and migrate by flying to the yam fields, where they cause the greatest damage. Here they burrow into the soil down to the tubers. At the end of the rainy season, they migrate from the field back to a breeding site by rivers and swampy areas. The total life cycle, from egg to adult, takes about 22-24 weeks.

Lesser yam beetles live in wetter areas during the dry season and migrate into yam gardens to breed.

IMPACT

About 95% of global yam production occurs in West Africa, primarily Nigeria, Ghana, Ivory Coast and Benin. Yams are one of the most important crops in West Africa, providing a source of carbohydrates and income. The adult beetles attack the tubers and leave holes, which reduces the value of the crop and provides an entry point for bacteria and fungi that cause the tuber to rot. It is reported that the beetle is the pest that causes the most damage to yam production in West Africa. They cause yield losses up to 77% and also significant post-harvest losses due to tubers that rot.

DISTRIBUTION

The greater yam beetle is widely distributed throughout tropical Africa, particularly in humid areas and near rivers in West Africa. It also occurs in India. The pest is often spread from country to country by tubers: movement of tubers or setts between countries should be restricted. The greater yam beetles are also found on islands in the Caribbean and Pacific Islands. Lesser yam beetles are found in southern Nigeria from Sierra Leone to Cameroon.

FURTHER READING

PIP Guide to Good Crop Protection Practices: for Yam (Dioscorea spp.). http://pip.coleacp.org/files/documents/GBPP-Ignames%2010-2011-09-1-UK.pdf

Infonet-Biovision http://www.infonet-biovision.org/default/ct/146/crops

FAO/IBPGR Technical Guidelines for the Safe Movement of Yam Germplasm. A. A. Brunt, G. V. H. Jackson, E. A. Frison Bioversity International, 1989, pp. 16-17.

Onwueme, I. C. and Charles W.B. 1995.Tropical Root and Tuber Crops: Production, Perspectives and Future Prospects. Food and Agriculture Organization of the United Nations, p. 90.

Okoroafor, E.; Amatobi, C. I.; Misari, S. M.; Onu, I. Field assessment of yam beetle (*Heteroligus meles* Billb) damage on yam cultivars (Dioscorea spp.). Research on Crops, 2009, 10, 2, pp 398-401.¨

Tobih, F.O. Evaluation of some plant materials as organic mulch for the control of yam tuber beetles (*Heteroligus* spp) In Delta State, Nigeria. Agriculture and Biology Journal of North America. 2011, 2(5): 818-825.

Yam moths

Dasyses rugosella, Euzopherodes vapidella and Erechthias minuscula

SUMMARY: Caterpillars of several different species of moth are major post-harvest pests of yams in Africa. Cleaning the storage facility and avoiding storing damaged tubers helps reduce damage. There are also botanical and synthetic pesticides that can be applied to stored yams to control the eggs, caterpillars and adult moths.

KEY SIGNS

Yam moths are post-harvest pests of yam tubers. Several species of yam moths have been observed in West Africa. The larvae of the moths bore into the tubers during harvest and storage, leaving tunnels. This causes the tubers to deteriorate, decreasing the value of the crop. The yams are most susceptible to an attack during the first four months of storage. The yam moths develop very quickly and can cause complete loss after only one month of storage. Favourable conditions for the moth occur during the dry season.

There are two main types of moths: pyralid moths, which cause the most damage, and tineid moths.

The adult pyralid moths (*Euzopherodes vapidella*) typically lay eggs in wounds on the surfaces of the tubers soon after harvest, but can also pierce the skin of the tuber to lay eggs. The yam variety *Dioscorea alata* (also known as water yam, greater yam or cuscus) is more commonly attacked by pyralid moths compared to other varieties; the tubers have a higher water content, which the moths prefer. Signs of yam moth caterpillars include black frass (faecal matter) woven together by silk threads and pupa cases that are left behind after the adult emerges. The adult moths do not eat the tubers; they only lay eggs which hatch into caterpillars that tunnel into the tubers, causing the damage.

The tineid moths (*Dasyses rugosella* and *Erechthias minuscula*) are a secondary pest that attack the tubers once the pyralid moths have already caused damage and the yams have lost moisture. Their larvae eat the inside of the tuber leaving only the skin. The tineid moths prefer the *Dioscorea cayenensis* variety (the yellow yam) but attack other varieties as well.

MANAGEMENT

Prevention – what to do before signs are seen

Cultural approaches: Use clean planting stock that does not have yam moth eggs or caterpillars on it.

Try not to pierce or damage the yams during harvest, as this can provide an easy access point for the yam moths.

Clean and disinfect the storage facility prior to storage.

If using a temperature-controlled storage facility, storing the tubers between 12°C and 20°C will delay the development of the moths and help control damage. Be careful not to store below 12°C, however, as this will cause chilling damage to the tubers.

Control – what to do after signs are seen

Cultural approaches: Do not store tubers that show signs of damage with undamaged tubers. Sort the tubers prior to storage and keep the damaged tubers separate.

Chemical approaches: There are several synthetic and botanical pesticides that can be effective against yam moths. The botanicals are less toxic to humans and the environment, and do not leave potentially dangerous chemical residues on the tubers.

- Powders of sweet pepper (*Capsicum annum*), also known as bell pepper or capsicum, and chili pepper (*Capsicum frutescens*) are effective against adult moths and can provide 100% control within 24 hours. The powders also prevent hatching and emergence of the adult moth. These powders can persist for 14 days, so they continue to provide control during that time. Apply by lightly dusting the pepper powder on cuts or other visible damage on the yam tuber surface.
- Pirimiphos-methyl (an organophosphate) and deltamethrin (a pyrethroid) can also be sprayed on stored tubers. Spray once after harvest before the tubers are placed in storage, and a second time after one month on damaged tubers only. Read the label for dosage rates and always follow safety precautions for use.

CAUSE

The pyralid moth, *Euzopherodes vapidella* Mann, and the tineid moths, *Dasyses rugosella* Stainton and *Erechthias minuscula* (formerly known as *Decadarchis minuscula* Walsingham) are species of yam moth known to be present in West Africa. There may be similar unidentified species as well. Yam moth is the common name, but *E. vapidella* is also known as the citrus stub moth or pyrale des greffons in French.

Euzopherodes vapidella life cycle: Eggs are 0.5 mm long, laid singly in rows and groups, and take 3.5 days to hatch. The larval stage lasts 14 days and the pupal stage about 7.5 days. Adult female moths (wingspan 13.8-16.8 mm, body length 7-9 mm) are bigger than the male moths (wingspan 11-13.8 mm, body length 6.2-7.5 mm) and live longer. The forewings of the adult moths are brown with a large dark brown-black thick stripe and dark brown-black along the edges near the tip of the wings. The total development time from egg to adult is an average of 27 days.

Dasyses rugosella life cycle: Eggs are elliptical and about 0.8 mm long. They are laid singly in rows and groups, hatching after about 5 days. The adult larvae have a dark head. The adult female moths (wingspan 17-18.5 mm, body length 6.5-8.5 mm) are bigger than the male moths (wingspan 13-15 mm, body length 5.3-6.8 mm). The total development time from egg to adult is an average of 61 days.

Erechthias minuscula: Adults are cream or light yellow in colour. The forewings are turned upwards at the tips, have light to dark brown scales and the wingspan is 3.5-4 mm.

IMPACT

Approximately 95% of the yams produced worldwide are produced in West Africa. Yams are one of the most important food crops and a main source of carbohydrates for people in this region. Yam moths are one of the most important post-harvest pests of yams. Storage losses of 10-15% after three months and up to 50% after six months have been reported. Yam moths are reported to cause 64% of the damage reported by yam chip producers in Benin.

DISTRIBUTION

Present primarily in West Africa, notably Nigeria, Ivory Coast and Benin, the three primary yam producing countries, but found throughout sub-Saharan Africa. E. vapidella is also a common pest of citrus crops in the Mediterranean regions of Europe and northern Africa (Morocco and Egypt), and has been found in Sri Lanka, Java, Australia, the Caroline Islands, Fiji, Samoa, the Marquesas, the West Indies, Hawaii and Florida.

FURTHER READING

PIP Guide to Good Crop Protection Practices: for Yam (Dioscorea spp.). http://pip.coleacp.org/files/documents/GBPP-Ignames%2010-2011-09-1-UK.pdf

Ashamo, M.O. 2010. Management of the yam moth, Dasyses rugosella Stainton, a pest of stored yam tubers (Dioscorea spp.) using plant products. 10th International Working Conference on Stored Product Protection. Julius-Kuhn-Archiv, 425.

Cotton bollworm

Helicoverpa armigera

Photo: Donald Hobern, Wikimedia Commons, CC-BY-2.0, http://bit.ly/1a8PJuf

Adult moth of cotton bollworm.

Photos: Gyorgy Csoka, Hungary Forest Research Institute, CC BY 3.0 US, www.bugwood.org

Cotton bollworm caterpillars in two different colours.

SUMMARY: The cotton bollworm is a major pest of many important food, oil and cash crops worldwide, including cereals, legumes, fruits and vegetables. A severe infestation of caterpillars of this moth can cause a complete loss in yield. Chemical control needs to be carefully timed as the caterpillars bore into the grains or fruit of the plant and are then protected. Resistance to pesticides, such as pyrethroids, has been reported in many countries. *Bacillus thuringiensis* (Bt) and neem extracts provide effective control against the caterpillars while minimising damage to natural enemies. Important cultural controls include removal and destruction of post-harvest crop residues, ploughing the soil to expose the pupae and uniform planting times.

KEY SIGNS

The cotton bollworm is considered to be one of the most important pests worldwide, attacking over 200 species of plants. The caterpillars feed on leaves and developing grain heads, and bore into the fruiting parts of plants.

The eggs are 0.4-0.6 mm across, round and yellowish-white, but change to a dark-brown colour before hatching. The eggs are typically laid singly on plants that are flowering or about to flower. The young caterpillars are yellowish-white to reddish-brown, with a dark brown to black head and black bumpy rows of short hairs running lengthwise on their backs. The fully grown caterpillars are 35-40 mm long and vary in colour from dark brown, yellowish-green, to reddish-brown, with greyish-black stripes along the side of the body and a thin light stripe. The pupae are 14-16 mm long, brown, and smooth on the surface. The adults also vary in colour, but typically the forewings are yellowish-brown with black kidney shaped markings in the middle of the wings and 7-8 small spots in a line along the border of the wing. The hindwings are white with a darker coloured band along the edge. The wingspan is about 35-40 mm at its widest point, and the body is 14-18 mm long. The adult moths are active at night and are good at flying, so they can easily move from plant to plant and field to field.

The caterpillars typically feed on the buds, flowers, grains, fruits and pods, but will also feed on the leaves and stems. In sorghum and millet, they feed on the developing grains during the milky stage and hide in the head during the day. In beans and tomato, they bore into the fruit. In maize, they feed on the developing seeds and bore into the cobs. The bore holes left by the caterpillars provide an entry point for bacterial diseases.

Monitoring is extremely important to prevent a severe outbreak. Monitoring should be done one or two days per week once the sorghum flowers bloom. Caterpillars can be seen on the plants, or shake the panicle heads over a bucket or sweep net. Bore holes and caterpillar dropping (frass) can often be seen on the plant. Pheromone traps and light traps can also be used to monitor the adult moths and provide some control.

MANAGEMENT

Prevention – what to do before signs are seen

Cultural approaches: Sorghum varieties with tight compacted heads are more likely to be attacked; plant varieties with loose open heads to reduce infestations and damage.

Resistant varieties of cotton, pigeonpea and chickpea have been released. Talk to local agro-input suppliers to see which varieties are available.

Planting a whole field at the same time (uniform planting) prevents movement of the moths from the older plants to the younger plants and reduces the damage.

Two post-harvest practices are important for preventing the population of bollworms from building up and becoming a severe problem the following season. First, it is important to remove and destroy crop residues immediately after harvest. Second, plough the soil to expose the pupae to direct sunlight and natural enemies.

Rotating with non-host crops can also prevent the population from building up, but because the bollworm can attack and harbour on many species of plants, rotation must be used together with other prevention and control practices.

Control – what to do after signs are seen

Cultural approaches: For small plots, it is possible to hand pick and destroy the eggs and young caterpillars.

There are many natural enemies of the bollworm, including *Trichogramma* spp. (egg parasitoids), several species of wasps and flies (caterpillar parasitoids), as well as many predators such as ants, lacewings, ladybugs, assassin bugs, minute pirate bugs and birds. Providing habitat to attract and preserve these natural enemies is an important strategy for controlling the bollworm.

Chemical approaches: The older caterpillars feed inside the fruiting part of the plant, protecting them from pesticide sprays. Therefore, if pesticides are used, they should be applied before the caterpillars bore into the pods or fruit of the plant. It is important to monitor the field regularly to look for eggs and young caterpillars before they bore into the plant. The decision to use a pesticide needs to be based on the severity of the problem, the presence of natural enemies and the economic value of the crop.

The cotton bollworm has developed resistance to several pesticides, notably synthetic pyrethroids.

Two microbial pesticides, *Bacillus thuringiensis* subspecies *kurstaki* and *Bt aizawai*, and *Helicoverpa armigera* nuclear polyhedrosis virus (HaNPV) can be used to control the young caterpillars with minimal harm to natural enemies. There are commercial products of these microbial pesticides available in many countries. Neem extracts, made from the neem seeds and leaves, and neem oil are also reported to be effective against the larvae and eggs.

CAUSE

Helicoverpa armigera has also been classified as *Heliothis armigera*. It is commonly known as the cotton bollworm, but is also called the African bollworm, tomato fruitworm, corn earworm, gram pod borer, old world bollworm and tobacco budworm. In French it is called chenille des epis du mais, noctuelle des tomates, or ver de la capsule.

Adult females can lay hundreds of eggs in a lifetime. They lay eggs singly on the tender parts of plants including the leaves, flowers, or fruit, and the eggs hatch in 3 to 5 days. The caterpillar period lasts 17 to 35 days, and the pupa period 17 to 20 days. The caterpillars drop from the plant to pupate inside a silk cocoon, 3-15 cm deep in the soil. The adult moths live between 1 and 25 days. The total life cycle can last up to 60 days; if conditions are favourable, then the total life cycle can be completed in 30 days. The length of time for development is mostly dependent on availability of food and the temperature: development time is shorter at higher temperatures. In warmer areas there can be numerous cycles per season.

IMPACT

The cotton bollworm is a major pest of many crops, including sorghum, soy, cotton, maize, common beans, chickpeas, groundnut, sunflower, potatoes, winter grains, citrus, wheat, barley, oats and a wide range of vegetable crops, including okra. The larvae can develop on many wild plant species, including *Amaranthus*, *Cleome* and *Acalypha*. The bollworm has a preference for feeding on the flowering and fruiting parts of plants, which causes high economic losses. If the infestation is severe, then it can result in a complete crop loss.

DISTRIBUTION

Native to Africa, the cotton bollworm has spread to Asia, Europe, Australia and the Pacific Islands. There have also been recent reports of infestations in South America. The pest can be spread through infested plant propagation materials. The adults are strong flyers and can move easily from field to field in areas where host plants are available; they can also be spread by strong winds.

FURTHER READING

Plantwise Knowledge Bank www.plantwise.org/knowledgebank

Phytoplasma diseases of cassava

Various phytoplasma

Photo: Paul van Mele, Agro-Insight

Cassava showing bunches of leaves, typical of witches' broom symptoms, in Thailand.

Photo: Paul van Mele, Agro-Insight

Brown vascular tissues are seen on witches' broom infected plants in Thailand when the stems are cut.

SUMMARY: Several severe diseases of cassava are associated with a number of different bacteria-like phytoplasmas in South America, the Caribbean, Asia, Africa, and the South Pacific. In some, shoots are bunched with small yellow leaves; in others, symptoms are seen only on roots at harvest. Management of these diseases is difficult: methods of spread are unknown and commercial growers and smallholders lack access to clean planting material. Careful selection of cuttings is recommended and also early removal of plants if disease symptoms occur.

KEY SIGNS

In recent years, a number of diseases have emerged that are caused by phytoplasmas. Often phytoplasma infections cause normally dormant shoots to grow, producing small, yellow leaves. These symptoms are called a witches' broom. However, not all the phytoplasma diseases of cassava produce symptoms like that. The phyoplasma diseases of cassava found so far are listed below.

In South America, frogskin of cassava has been reported. It is unusual in that there are few above ground symptoms of disease, apart from slight swellings of lower stems, and curling and yellow/green patterns on the leaves of some varieties. However, the roots are thin, woody, with thick outer layers, and deep cracks. Sometimes the cracks will form a ring around the root.

In Thailand, cassava witches' broom disease was first reported in 2008. It spread rapidly and is now widely distributed across the region. Affected plants show bunches of shoots with short internodes, small yellowish leaves at the top of the plants, brown vascular tissues (the tubes that carry nutrients and water) and poor storage root development.

In Brazil, a cassava disease associated with a phytoplasma has been known since the 1940s. Losses of 70% are reported on stunted plants with yellow distorted leaves and abnormal multiple shoots.

In Uganda, plants have recently been found with phytoplasma that have yellowish leaves and are slightly stunted. The weed sticky mallow (*Malvaviscus arborus*) was also found to be infected with the same phytoplasma.

MANAGEMENT

Prevention – what to do before signs are seen

Cultural approaches: The recent appearance of these diseases in different parts of the world suggests that they are new and, as such, must be monitored carefully. They reinforce the need for care when moving plants internationally. Unrestricted movement could spread phytoplasmas and other diseases. Transfers should only be made as pathogen-tested plants growing as sterile tissue cultures following the FAO/IBPGR (1991) *Technical Guidelines for the Safe*

Movement of Cassava Germplasm.

A priority in the management of these diseases is the use of clean planting material; however, there are few if any such schemes in African countries. Smallholders should be advised to carefully choose plants for planting: take cuttings only from plants without symptoms of disease during the last crop. This is the most important and probably the only effective method of managing these diseases, if sources of certified or approved cuttings are not available.

Additionally, do not plant new plots of cassava next to those that have the disease.

Control – what to do after signs are seen

Cultural approaches: During the crop growth, remove any plants with symptoms of phytoplasma as soon as they are seen: look out for witches' broom symptoms, as well as stunted plants with abnormal yellow leaves.

Collect and burn plants that show symptoms of disease.

CAUSE

Phytoplasmas are bacteria-like organisms that can only exist inside the phloem (part of the vascular system) of plants. They are mainly spread by insects. Unlike bacteria, they do not have rigid cell walls and so vary in shape. Phytoplasmas often cause diseases of economic importance.

Molecular analyses of the four phytoplasmas that cause disease in cassava have shown that they belong to different taxonomic groups and, therefore, are the cause of different diseases. Apart from these four, there are also reports of phytoplasma diseases of cassava in Cuba and the Wallis and Futuna Islands in the South Pacific. Methods of spread are unknown for any of them, except for movement in stem cuttings used for planting.

IMPACT

Cassava is the third most important crop in the tropics after rice and maize in terms of calories produced. Phytoplasma diseases of cassava are a threat to its production as an export crop (mainly Southeast Asia), and as food and income for smallholders. As such, phytoplasma diseases are a potential threat to food security for millions of people. In Vietnam, for instance, the impact occurs in three ways: (i) there is direct yield loss (in some parts of the country, 60,000 ha were affected in 2010, with overall yield and starch losses of 30%); (ii) processors need to buy more cassava roots to make up for the shortfall in starch content, and (iii) farmers, who are paid on starch content, are paid less. In the other regions affected by phytoplasma diseases the impact is just as serious. In parts of Colombia and Brazil, for instance, epidemics of phytoplasma disease have resulted in yield losses of 70-90%.

DISTRIBUTION

Cassava witches' broom spreads in Southeast Asia (Cambodia, China, Indonesia, Laos, the Philippines, Thailand and Vietnam). Frogskin is known from Colombia, Brazil, Venezuela, Peru, Costa Rica and Panama. Other diseases, as yet not well defined, occur in Brazil, Costa Rica, Cuba, Panama, Peru, the South Pacific and Venezuela.

FURTHER READING AND OTHER RESOURCES

Alvarez E, Pardo JM, Fernando MJ, Assunta B, Thanh ND, Hoat TX (2014) Detection and identification of 'Candidatus Phytoplasma asteris'–related phytoplasmas associated with a witches' broom disease of cassava in Vietnam. Phytoplathogenic Mollicutes 3(2): 77-81. (http://www.indianjournals.com/ijor.aspx?target=ijor:mollicutes&volume=3&issue=2&article=003)

Arocha Y, Echodu R, Talengera D, Muhangi J, Rockefeller E, Asher O, Nakacwa R, Serugga R, Gumisiriza G, Tripathi J, Kabuye D, Otipa M, Vutseme K, Lukanda M, Boa E (2009) Occurrence of 'Candidatus Phytoplasma aurantifolia' (16SrII group) in cassava and four other species in Uganda. Plant Pathology 58(2): 390.

Cassava diseases in Africa: A major threat to food security. (http://www.fao.org/docrep/012/i1460e/i1460e.pdf).

Flores D, Haas IC, Canale MC, Bedendo IP (2013) Molecular identification of a 16SrIII-B phytoplasma associated with cassava witches' broom disease. European Journal of Plant Pathology. Published online. (http://www.scribd.com/doc/228796504/1-Molecular-Identification-of-a-16SrIII-B-Phytoplasma).

Frison EA,Feliu E (eds) (1991) FAO/IBPGR Technical guidelines for the safe movement of cassava germplasm. Food and Agriculture Organization of the United Nations, Rome/International Board for Plant Genetic Resources, Rome. (http://ecoport.org/Resources/Refs/IPGRI/cassava.pdf).

Oliveira SAS, Abreu EFM, Araújo TS, Oliveira EJ, Andrade EC, Garcia JMP, Álvarez E (2014) First report of a 16SrIII-L phytoplasma associated with frogskin disease in cassava (Manihot esculenta Crantz) in Brazil. Plant Disease 98(1): 153-154.

Phytoplasma aurantifolia. CABI Crop Protection Compendium. (http://www.cabi.org.ezproxy.library.uq.edu.au/cpc/datasheetreport?dsid=54518).

Witches' broom – a curse on cassava. (http://www.new-ag.info/en/focus/focusItem.php?a=3184).

Cassava brown streak disease

Cassava brown streak viruses

Photo: Eric Boa, CABI, CC BY 4.0

Typical dry brown rot is only evident when tubers are cut open.

Photo: Eric Boa, CABI, CC BY 4.0

Leaf symptoms are seen clearly here against the light, but are easy to miss when walking through fields.

SUMMARY: Cassava brown streak disease (CBSD) has been known since the 1930s but has only recently become a major problem. Originally restricted mostly to the coastal regions of Tanzania and neighbours, an outbreak of this virus disease in the Great Lakes region in 2003 has since spread, particularly at higher altitudes (>1000 masl), to other countries in East and Central Africa. CBSD is not present in West Africa. CBSD is a serious threat to a key food crop. The leaf symptoms appear mild by comparison with the huge losses in tuber production: a dry brownish rot makes tubers unfit for eating and sale. CBSD occurs alongside cassava mosaic disease (CMD), another major threat to production caused by a different type of virus. Both diseases are spread by whiteflies and in infected planting material. Effective management depends on developing resistant varieties and making clean planting material available to farmers.

KEY SIGNS

CBSD and CMD may occur in the same field and co-infect plants. Although often discussed together there are key differences between these two virus diseases. In CBSD leaf symptoms occur only on older or mature leaves, not on young expanding leaves, as in CMD. There is no leaf distortion with CBSD. Both virus diseases cause a patchy yellowing of leaves but in CBSD this is less intense. In CBSD yellowing occurs along the thin secondary veins that branch off from the main central vein. This can spread and form bigger patches, with a 'feathery' or 'blotchy' appearance.

The most distinctive symptom of CBSD is a dry rot of the tubers. Some plants may have leaf symptoms while tubers are unaffected. CMD affects tuber production but not the tubers internal appearance. Weakly growing cassava may have other fungal rots of tubers: look out for characteristic white threads (hyphae) on the outside of the tuber, though these may not always be present.

In CBSD-affected plants tuber rots begin with localised dead areas that are yellow-brown, with a cork-like appearance. The most susceptible cultivars have extensive rotting and tubers cannot be eaten. Some tubers have restricted growth, appearing like a series of rings stuck together. In some cultivars the tubers are affected but the leaves remain healthy.

Although the disease is named after brown streaks that occur on stems, these are of minor significance in terms of damage caused. The brown streaks are difficult to see and occur infrequently. CBSD infections may go undetected for long periods of time.

MANAGEMENT

Prevention – what to do before signs are seen

Cultural approaches: Early detection, use of clean planting material and resistant cultivars are the major methods for managing CBSD.

Susceptibility to cassava brown streak viruses, of which there are two similar species, varies. Some cultivars are tolerant: they retain the virus but don't develop strong symptoms. Tolerant varieties, such as Garukunsubire and Seruruseke in Rwanda, limit losses locally but remain an important source of inoculum for new outbreaks. Cultivars described as resistant to CBSD are used in several countries, for example Kibaha, Rangi Mbili and Kasala in Tanzania. It is still not clear if these remain largely virus-free ('true resistance') or whether they are tolerant and still carry the disease.

Transgenic varieties resistant to CBSD and/or whiteflies are being developed and show promise. The added complication for plant breeders is that many of the existing CMD resistant cultivars are susceptible to CBSD.

Control – what to do after signs are seen

Cultural approaches: The removal of plants with typical CBSD leaf symptoms, known as roguing, is a useful means of limiting disease spread when only a few plants are affected. Symptomless plants may still contain the virus however, and roguing is no guarantee of successful elimination of CBSD.

It is important that neighbouring farmers collectively scout their fields, particularly when CBSD has been recently reported in an area. Effective surveillance and rapid responses are high priorities for ensuring that CBSD does not spread to new areas (e.g. West Africa). Making best use of advisory services and extension workers is part of a balanced strategy to improve early detection and responses.

Chemical approaches: Integrated control strategies include chemical vector control, but this will always be difficult and of limited benefit to farmers. First, controlling whiteflies is time-consuming and expensive. Second, it must be done regularly and effectively. Third, the greatest risk is in planting already infected material, for which there is no treatment. Once the virus is present in the plant there is no way to control the disease.

CAUSE

There are two species of cassava brown streak viruses, both belonging to a type of potyvirus known as ipomoviruses. One is known as *Cassava brown streak virus* and the other as *Uganda cassava brown streak virus*. Both are transmitted by whiteflies and in infected planting material, which may show no symptoms of CBSD.

The pattern of spread of CBSD and CMD differs. CMD moves steadily in a distinct front, up to 100km in a year, similar to the ripples caused by throwing a large stone into a pond. CBSD has arisen in isolated areas (hot spots) then spread outwards, similar to the ripples from throwing several, smaller stones into a pond. This suggests the separate introduction of infected planting material across large areas.

The genetic diversity of CBSD viruses is not fully characterized and it is still unclear if the current epidemic is due to a new strain or species or some other as yet unidentified factor (see Impact).

IMPACT

CBSD has a devastating effect on cassava production. Tubers are traditionally stored in the ground for long periods until needed, and the unexpected discovery that they are unusable greatly affects families expecting to eat or sell their crops. Losses of up to 60% in crop production (70% for the most susceptible varieties) have been reported from Malawi. The super-abundance of whiteflies has also directly led to major crop losses

CBSD was observed in the 1940s and 1990s at a few locations in Uganda yet there was no apparent spread of the disease and certainly no pandemic. These earlier disease introductions (on breeding material) appear to have been successfully intercepted by researchers. It is possible that an increase in development efforts to mitigate the widespread effects of entrenched poverty and persistent conflict have inadvertently assisted in the distribution of infected planting material in East and Central Africa.

Whiteflies, the vectors of CBSD, appear less effective in passing the disease on to healthy plants compared to transmission of other virus diseases of cassava.

DISTRIBUTION

CBSD has been confirmed from Tanzania, Mozambique, Kenya, Uganda, Rwanda, Burundi, Malawi, Equatorial Guinea, Zimbabwe and eastern DR Congo. Unconfirmed reports suggest it is present in Bas Congo. Mapping the distribution and spread of CBSD is made more difficult by the lack of distinct, above-ground symptoms.

FURTHER READING AND OTHER RESOURCES

Plantwise Knowledge Bank (www.plantwise.org/cpc)

Crop Protection Compendium (www.cabi.org/cpc)

FAO (2010). Cassava diseases in Africa: a major threat to food security. Strategic programme framework, 2010-2015. (available from: http://www.fao.org/emergencies/resources/documents/resources-detail/en/c/171103/)

Legg JP, Jeremiah SC, Obiero HM, Maruthi MN, Ndyetabula I, Okao-Okuja G, Bouwmeester H, Bigirimana S, Tat-Hangy W, Gashaka G, Mkamilo G, Alicai T, Lava Kumar P, 2011 Comparing the regional epidemiology of the cassava mosaic and cassava brown streak virus pandemics in Africa. Virus Research 159, 161-170

Cassava bacterial blight

Xanthomonas axonopodis pv. manihotis

Photo: Fen Beed

Yellow patches spread outwards from the leaf spots as the leaf withers.

Photo: Fen Beed

Bacteria block movement of water and nutrients in the woody stem; the leaves above the blockage wilt without developing any leaf spots.

SUMMARY: Cassava bacterial blight is perhaps less well known in Africa than cassava mosaic disease and cassava brown streak. It deserves more attention, however, given the continuing risk of major outbreaks and the large losses due to the disease in the past, particularly in DR Congo. The disease is introduced in symptomless cuttings which fail to grow. The bacteria are then spread by rain splash and physical contact. Varieties with good tolerance to the disease are available though they may not be widely known.

KEY SIGNS

When an infected cutting starts to grow (primary infection), the new shoots wilt and the stem dies back. Secondary infections occur through leaf 'breathing' holes (stomata), or directly through wounds and leaf scars on the woody stems. On the leaves, the first symptoms are small dark-green to blue leaf spots. Minor veins initially restrict the development of the leaf spot, which is angular and water-soaked. Later, the spots enlarge and join up, often along the leaf edge. Yellow patches spread outwards from the leaf spots as the leaf withers and droops. On young leaves this can occur in two to three days under humid conditions.

High humidity favours movement of bacteria in infected plants, leading to production of a sticky ooze containing bacteria, most noticeably on stems but also from infected leaves. Dark areas (lesions) develop along the leaf stalks and woody stems. Bacteria block movement of water and nutrients in the woody stem, causing leaves to wilt without developing any leaf spots. The soft tissue at the top of the growing shoot dies, giving plants a characteristic 'candle' appearance. New leaves grow out lower down the stem but they will also eventually die.

MANAGEMENT

Prevention – what to do before signs are seen

Cultural approaches: In Brazil, where the disease was first seen, the development of integrated control strategies has greatly reduced losses of a once catastrophic disease. The most important part of this strategy is the selection of healthy cuttings and use of resistant or tolerant cultivars.

Although much research has been done on resistance it is difficult to find information on named cultivars. One publication from the year 2000 for West Africa lists 'tolerant' cultivars such as: TMS 4(2)1425 and TMS 30572 from IITA; Afisiafi and Abasa fitaa from Ghana; and NR 8082 from Nigeria. Check locally for availability of cultivars with known resistance or tolerance to the disease.

Infected cuttings do not show any symptoms of bacterial blight. Great care should be taken therefore to obtain planting material from trusted sources or areas free from the disease.

Control – what to do after signs are seen

Cultural approaches: Once the disease is present, careful cleaning of tools will limit spread to healthy plants while animal and human movement within the crop should be restricted to prevent physical transfer of bacteria. The disease spreads less rapidly during dry periods. Cutting out most of the above ground stems of infected plants may limit losses in varieties with some resistance to the disease, but success also depends on how long the plant has been infected and the spread of the bacteria within the plant.

If only a few plants in a field show symptoms, speedy removal and safe disposal of infected plants may reduce further spread.

Chemical approaches: There is no chemical treatment for controlling the disease.

CAUSE

The disease is caused by the bacterium *Xanthomonas axonopodis* pv. *manihotis*. In the past it was also known as *Xanthomonas campestris* pv. *manihotis*.

This is an important and serious bacterial disease of cassava. It is spread in cuttings and in seed. Within fields, the bacteria are spread by rain splash, cutting tools and by people and animals brushing against infected plants.

When new cassava is planted at the same time as harvesting mature plants, machetes and pangas can spread the disease to uninfected cuttings.

IMPACT

Cassava leaves are an important food in many countries and bacterial blight greatly limits their availability. The failure of cuttings to grow can be catastrophic; in the early 1970s a serious outbreak in Zaire led to starvation when up to 75% of the fresh roots were lost. Outbreaks in Uganda and Nigeria caused yield losses of 75-100% in some areas.

Fungal diseases can also attack plants with bacterial blight and further increase losses.

The elimination of varieties of cassava susceptible to bacterial blight, following progressive outbreaks of the disease in several countries, has apparently helped to contain the disease.

DISTRIBUTION

In eastern and central Africa, bacterial blight occurs in Congo, DR Congo, Central African Republic, Sudan, Uganda, Rwanda, Burundi, Tanzania and Kenya. In southern Africa the disease has been reported from Malawi and South Africa but not from Zambia, where strict quarantine measures were introduced to prevent its introduction. In West Africa, bacterial blight occurs in Cote d'Ivoire, Mali, Ghana, Niger, Benin, Togo, Nigeria and Cameroon.

FURTHER READING AND OTHER RESOURCES

Crop Protection Compendium (www.cabi.org/cpc)

An old but still useful paper from 1986 is available online and gives a good summary of the disease: www.apsnet.org/publications/plantdisease/backissues/Documents/1986Articles/PlantDisease70n12_1089.PDF.

For information on 'tolerant' cultivars see:

Msikita W, James B, Nnodu E, Legg J, Wydra K, Ogbe F (2000). Disease Control in Cassava Farms. IPM Field Guide for Extension Agents. IITA. Available at: http://old.iita.org/cms/details/ipm/Disease%20control.pdf

Cassava mosaic disease

Cassava mosaic geminiviruses

Photo: Eric Boa, CABI, CC BY 4.0

Advanced mosaic and distortion of leaves on an infected cutting.

Photo: Eric Boa, CABI, CC BY 4.0

Although chlorotic (yellow) areas are well developed the limited leaf distortion suggests infection via whiteflies after planting.

SUMMARY: Cassava mosaic disease (CMD) is widespread, occurring in all countries in Africa where cassava is grown. This viral disease has been present in Africa for over a hundred years yet caused relatively little concern until the 1980s, when an outbreak of a damaging epidemic began in Uganda and now affects all neighbouring countries. The disease is spread in cuttings, which may be symptomless, and by whiteflies. Disease management has concentrated on the use of resistant cultivars. While this is an important strategy to reduce yield losses, more attention should be paid to the use of clean planting material, early detection of CMD in fields and removal of plants.

KEY SIGNS

The main symptoms of CMD are patches of discoloured (chlorotic) areas on leaves, which vary from light green to yellow, accompanied by distortion of leaves, often severe. CMD symptoms are readily detected in the field though symptomless infections also occur. Mosaic symptoms vary greatly in overall pattern and intensity depending on the strain of virus, susceptibility of cassava cultivars and growing conditions. The more extensive the development of leaf symptoms, the greater the overall reduction in plant growth and yield.

Two types of mosaic have been noted: yellow mosaic shows a strong contrast between the discoloured areas and healthy tissue; in green mosaic the colour changes in leaves are less intense and the border with healthy leaf areas less distinct. The leaf distortion occurs because the chlorotic areas expand less rapidly. Leaf stalks may also be affected and have a characteristic S-shape, though this is not a key symptom.

In severe infections leaves fall off while tubers continue to show no obvious symptoms. However, the effect of CMD on tuber production is often profound, with fewer, smaller and even no tubers produced. Rotting is due to secondary diseases, such as *Armillaria*, a widespread fungus, in plants already severely affected and weakened by CMD.

Cassava mosaic geminiviruses (CMGs) are spread in cuttings and by whiteflies. The first and subsequent leaves from infected cuttings show characteristic mosaics and distortion. Mother plants selected for cuttings may show little or no evidence of CMD. Healthy cassava cuttings also become infected by whiteflies carrying the virus but do not show symptoms for some time.

CMD symptoms can been confused with green mite feeding damage. This feeding results in a speckling of the leaf surface and some distortion, though much less pronounced than CMD. The damage caused by the mites develops more uniformly in groups of plants compared to the greater variation in CMD development.

MANAGEMENT

Prevention – what to do before signs are seen

Cultural approaches: CMD is an internal disease: the virus lives in the host, where it multiplies and spreads, disrupting development and reducing growth and yields. There are no treatments for virus-infected plants, hence the importance of selecting material for propagation from a reliable and disease-free source. Particular attention should be given to training extension workers, farmers and others involved in sourcing healthy cuttings for propagation.

CMD-resistant cultivars are widely available. Their acceptability to farmers will depend on an overall assessment of other key crop characteristics. Extension services and research organisations are the best source of advice on resistant cultivars and how to obtain them. Extra care is needed in selection of planting material following the spread of another cassava virus disease, cassava brown streak disease (CBSD), beginning in the early 2000s in East Africa. Many of the CMD-resistant cultivars are, unfortunately, susceptible to CBSD.

Chemical approaches: Vector control is complicated by the difficulty in killing enough insects quickly (and regularly) to limit the spread of CMD in fields. The cost of chemicals and labour is likely to be high and difficult to afford for poor farmers. The reported 'super-abundance' of whitefly populations in Uganda raises further barriers to effective vector control.

There are no proven biocontrol options and preventing movement of whiteflies from infected areas is next to impossible. The overall conclusion is that vector control offers few if any benefits in managing CMD.

Control – what to do after signs are seen

Cultural approaches: Removal of diseased plants from fields is part of an integrated strategy for managing CMD. This is only effective if done early, before many plants are affected.

CAUSE

There are 8-9 cassava virus species which cause CMD. All are geminiviruses and share similar characteristics, though they interact with cassava in different ways. All cassava geminiviruses (CMGs) are closely related. Some have distinct common names, such as *African cassava mosaic virus* and *East African cassava mosaic virus*, reflecting particular patterns of disease attack.

CMD was of relatively minor importance until the 1980s, when a significant epidemic began in Uganda. CMGs are constantly changing with new variants favouring niches that existing types are unable to survive in.

IMPACT

Cassava infected by CMD is readily visible throughout East Africa, yet the full impact of the disease on yields may not always be fully appreciated. Reduction in tuber production has a profound impact on farmers who depend on a crop which is a traditional bulwark against drought and famine. Loss of cassava leaves is also important where they are a traditional food.

The contiguous production of cassava across Uganda, Tanzania, Kenya and beyond has allowed new and more damaging virus strains to spread rapidly. Coordinated actions by extension services and farmers over large areas are essential to detect new outbreaks and limit disease spread.

Changes in the behaviour, biology and abundance of whiteflies have also been important in increasing the impact of CMD in eastern Africa.

DISTRIBUTION

CMD is present in all countries in Africa that grow cassava, from Senegal to South Africa and also on island nations such as Seychelles and Madagascar. CMD also occurs in India and Sri Lanka.

FURTHER READING

Plantwise Knowledge Bank (www.plantwise.org).

Crop Protection Compendium (www.cabi.org/cpc)

Legg JP, Jeremiah SC, Obiero HM, Maruthi MN, Ndyetabula I, Okao-Okuja G, Bouwmeester H, Bigirimana S, Tat-Hangy W, Gashaka G, Mkamilo G, Alicai T, Lava Kumar P, 2011 Comparing the regional epidemiology of the cassava mosaic and cassava brown streak virus pandemics in Africa. Virus Research 159, 161-170

Thresh JM, Cooter RJ, 2005. Strategies for controlling cassava mosaic virus disease in Africa. Plant Pathology 54, 687-614

Leaf petiole and stem blight disease of sweet potato

Alternaria bataticola

Photo: Carlos Lopes, EMBRAPA

Black sunken spots with grey centres on stems.

Photo: Carlos Lopes, EMBRAPA

Damage to leaves and stems causing a blight.

SUMMARY: Leaf petiole and stem blight disease of sweet potato (also called sweet potato Alternaria blight), is caused by the fungus *Alternaria bataticola*, and results in spots on leaves, petioles (the leaf stalks) and stems, causing leaves to fall and vines to die. It is present in several African countries but is especially serious in the cool, moist environments of central and southwestern Uganda. Management is through the use of varieties selected or bred for resistance or tolerance, careful choice of planting material, and hygiene measures, principally the destruction of vines after harvest.

KEY SIGNS

At first, small dark brown to black oval spots occur on the fully-grown leaves and show a pattern of rings. On the underside of the leaves the veins turn black. The spots grow up to 5 mm in diameter, frequently join together and are often surrounded by yellow halos. Later, the infected leaves turn yellow and fall off. In severe cases they create a carpet of dead blackened leaves over the soil. On petioles and stems the spots are grey at first, later becoming black and sunken. If they grow right around the petiole and stem they will kill them.

MANAGEMENT

Prevention – what to do before signs are seen

Cultural approaches: Leaf petiole and stem blight disease varies among varieties. The selection and breeding of resistant or tolerant varieties is the main method of managing the disease, together with cultural practices of crop hygiene, involving the destruction of the infected crop debris.

Before planting, check the availability of NASPOT[1] varieties released from the Ugandan breeding programme; many of these have been distributed to other countries in sub-Saharan Africa. Apart from high yields and acceptable taste, some have been selected for their orange flesh and also for their resistance to sweet potato virus disease. In 1999, NASPOT varieties 3, 5 and 6 with resistance were released. These were followed by NASPOT 7 to 11. NASPOT 11 is of most interest, a seedling selection from a farmer participatory breeding programme, with acceptable storage root shape, high dry matter, good to excellent consumer acceptance and moderate to high field resistance to sweet potato virus disease and leaf petiole and stem blight. Two orange-fleshed varieties with moderate resistance have been released from the same programme (Kakamega and Ejumula).

Selection of planting material should be done carefully, avoiding any vine cuttings with symptoms on leaves, petioles and stems.

Chemical approaches: Chemical control is not an appropriate method of managing this disease. Although fungicides

[1]Nasmulonge sweetpotato

would be effective, they are too expensive for most smallholders and often they are unavailable. If required in commercial planting, mancozeb or copper compounds would be suitable choices.

CAUSE

The fungus *Alternaria bataticola* is the cause of leaf petiole and stem blight of sweet potato. Although a number of *Alternaria* species (e.g. *A. alternata, A. brassicae* and *A. solani*) have been found causing spots on sweet potato leaves, *A. bataticola* is the only one that attacks the whole vine (leaves, petioles and stems) throughout the crop cycle. Surveys in Uganda, for instance, showed the predominant species were *A. bataticola* (55% of isolates) and *A. alternata* (40%). However, it is not the only fungus that causes similar symptoms. Recent reports from South Africa have shown that a disease similar to leaf petiole and stem blight was mostly associated with another fungus, from the *Phoma* genus. An *Alternaria* was found occasionally, but it was not typical of *A. bataticola. Phoma* is a common soil-borne fungus that causes a pink rot of the storage roots, but had not previously been reported on vines in South Africa.

Leaf petiole and stem blight of sweet potato is also known as *Alternaria anthracnose.* Anthracnose means 'coal disease'; it is a word used to describe diseases caused by fungi that produce dark spots on leaves, petioles, stems and fruits. The disease on sweet potato is also known as *Alternaria* leaf and stem blight.

The disease is spread over short distances by airborne spores and is also carried on wind-driven rain. Over long distances the disease is spread on cuttings used for planting. High relative humidity is required for spore germination, infection and spore formation.

Survival between crops is in plant debris remaining after harvest. Sweet potato is the only known host.

IMPACT

Most work on impact has been carried out in Uganda where yield loss depends on variety, region and cropping season. All commonly grown and preferred varieties are susceptible. The disease is serious in crops at mid and high elevations, those in the cool, moist southwestern highlands (altitude above 1500 masl and annual rainfall 900-1350 mm), and in parts of the central Lake Crescent Region, but less so in the drier regions of eastern and northern Uganda. In places where conditions favour the disease, losses of storage root yield of 50-90% are reported, especially where leaf petiole and stem blight and sweet potato virus disease occur together. Such is the importance of these diseases that they are the focus of breeding programs at the National Crops Resources Research Institute, Namulonge, Uganda, in collaboration with the International Potato Center (CIP), Peru.

DISTRIBUTION

The disease is important in Eastern and Central Africa (Burundi, Ethiopia, Kenya, Rwanda and Uganda) and also in Brazil. It is also reported from Japan and Papua New Guinea.

FURTHER READING

Adebola PO, Lane CR, Smith J (2010) Molecular characterisation of Alternaria species of Sweet Potato and development of a host resistance screening protocol. Aspects of Applied Biology 96:309-313. (http://www.cabi.org.ezproxy.library.uq.edu.au/cpc/FullTextPDF/2010/20103346646.pdf).

Ames T, Smit NEJM, Braun AR, O'Sullivan JN, Skoglund LJ (1997) Sweet potato: major pests, diseases, and nutritional disorder. International Potato Center, Lima, Peru. (http://cipotato.org/wp-content/uploads/publication%20files/books/002435.pdf).

Mwanga ROM, Odongo B, Turyamureeba G, Alajo A, Yencho GC, Gibson RW, Smit NEJM, Carey EE (2003) Release of six sweet potato cultivars ('NASPOT 1' to 'NASPOT 6') in Uganda. Hortscience 38(3):475-476. (http://hortsci.ashspublications.org/content/38/3/475.full.pdf).

Mwanga ROM, Odongo B, Niringiye C, Alajo A, Abidin PE, Kapinga R, Tumwegamire S, Lemaga B, Nsumba J, Carey EE (2007) Release of two orange-fleshed sweet potato cultivars, 'SPK004' ('Kakamega') and 'Ejumula' in Uganda. Hortscience 42(7):1728-1730. (http://hortsci.ashspublications.org/content/38/3/475.full.pdf).

Mwanga ROM, Niringiye C, Alajo A, Kigozi B, Namukula J, Mpembe I, Tumwegamire S, Gibson RW Yencho GC (2011) 'NASPOT 11', a Sweet potato Cultivar bred by a Participatory Plant-breeding approach in Uganda. Hortscience 46(2):317–321. 2011.(http://hortsci.ashspublications.org/content/46/2/317.full.pdf).

O'Sullivan J, Amante V, Norton G, van de Fliert E, Vasquez E, Pardales J (Undated) Sweet potato DiagNotes: A diagnostic key and information too for sweet potato problems. (http://keys.lucidcentral.org/keys/sweetpotato/key/Sweetpotato%20Diagnotes/Media/Html/FrontPage/Index.htm).

Sweet potato virus disease

Sweet potato chlorotic stunt virus & sweet potato feathery mottle virus

Photo: Dr Richard Gibson

Two plants (in the foreground) affected by sweet potato virus disease, with a healthy plant behind.

Photo: (Top) Grahame Jackson, CABI, CC BY 4.0.
(Bottom) Gerald Holmes, California Polytechnic State University at San Luis Obispo, CC BY-NC 3.0 US, www.bugwood.org

Narrow bands around the storage root, symptomatic of sweet potato feathery mottle virus (top). Feathery mottle virus symptoms (russet crack strain) on storage roots causing fissures (bottom).

SUMMARY: Two virus species, one spread by aphids, the other by whiteflies, cause sweet potato virus disease when they occur together. When just one virus is present, often no symptoms are seen. The disease is especially severe in East Africa: plants become stunted, with vines bearing narrow yellow leaves and a lack of storage roots. The most important control measures are the use of tolerant varieties, careful selection of cuttings, removal of infected plants in the first month from planting and leaving at least 15 metres between plantings.

KEY SIGNS

Infection by sweet potato chlorotic stunt virus (SPCSV) can result in a mild yellowing or reddening of older leaves and stunting, as its name suggests. Infection by sweet potato feathery mottle virus (SPFMV) can result in yellow spots or purple ringspots and, occasionally, feather-like patterns bordering the major leaf veins. On the storage roots, some strains, on certain varieties, cause networks of small cracks in the skin, or longitudinal fissures, in circular bands one or more centimetres wide. Inside, the storage roots may show black spots. These symptoms are known as russet crack and internal cork, respectively.

Often, however, either virus on its own does not produce any symptoms in sweet potato, or the symptoms occur only when plants are under stress and growing slowly; for instance, when there is not enough rain. If growing conditions improve, the rapidly growing vines often appear healthy.

By contrast, when the two viruses occur together in susceptible sweet potatoes they cause SPVD. Infected plants have yellow, narrow leaves, often with deformed edges, and vines are severely stunted. Yields of storage roots are generally low, but it depends when infection occurs; if it is early, storage roots fail to develop.

MANAGEMENT

Prevention – what to do before signs are seen

Cultural approaches: Because of the severity of this disease, unrestricted international movement of sweet potato plants should be avoided. Transfers should only be made as pathogen-tested plants growing as sterile tissue cultures, following the FAO/IBPGR (1989) Technical Guidelines for the Safe Movement of Sweet Potato Germplasm[1].

In Uganda, where the disease is particularly severe, varieties occur with resistance to SPVD. These include New Kawogo, Nderera and Munyeera. These are rarely affected by the disease, although yields are poor in comparison to the potential yields of susceptible varieties. New Kawogo is particularly popular because of its tolerance to SPVD. Varieties with moderate field resistance from the Namulonge breeding programme are NASPOT 1 to 6, released in 1999, and NASPOT 11, released in 2010, with improved resistance to SPVD. Check to see if these varieties are available locally.

Farmers should be encouraged to use these varieties, not only for their tolerance to disease, but also because growing

[1] http://www.bioversityinternational.org/e-library/publications/detail/sweet-potato

large amounts will reduce infection of susceptible varieties if grown nearby. However, in the absence of SPVD, they are lower yielding than susceptible varieties.

Farmers should also be encouraged to grow tip cuttings in nurseries, for later planting in the field after checking for the disease, rather than taking cuttings from the last crop, which may have unseen SPVD infections. After harvest, collect vines and burn, bury or compost them. Do not allow discarded storage roots to sprout; collect and feed to livestock or bury them.

Control – what to do after signs are seen

Cultural approaches: During crop growth, remove plants with SPVD, especially during the first month after planting. The early removal of infected plants can significantly reduce the likelihood of the remaining crop developing the disease. In addition, new plantings should be at least 15 metres away from existing plantings where diseased plants might be present; this gap will stop the whiteflies (which transmit SPCSV but do not travel far from their host plant) from spreading the virus to new plants.

CAUSE

Sweet potato virus disease (SPVD) is caused by two different viruses infecting plants at the same time. On their own these viruses do relatively little damage. Although SPVD was first reported in the scientific literature in the 1940s, it was not until 30 years later that the cause was known.

The two viruses involved, SPCSV and SPFMV, belong to different virus groups. Both exist as different strains in East Africa, and there are also differences between East African strains and those elsewhere. As a result of these differences, varieties resistant to SPVD in West Africa became severely diseased when grown in Uganda.

Severe SPVD symptoms occur because the presence of SPCSV allows SPFMV to reach much higher concentrations (up to 600 times) than if it were alone. Symptoms may become even worse if a third virus infects the plants: there are over 30 different types of virus that infect sweet potato.

Spread of SPCSV and SPFMV occurs in three ways. First, they are spread between plants by insects: SPCSV by the whitefly *Bemisia tabaci* and SPFMV by aphids. The viruses are picked up as the insects feed on plant sap. Once aphids have the virus in their mouthparts, they can then infect healthy plants immediately, but this ability to infect is lost quickly. By contrast, whiteflies take a few hours before they are ready to spread SPCSV, but they can continue to infect for a few hours. Secondly, the viruses are spread in cuttings used for planting. Thirdly, the viruses are spread in storage roots sent to markets; buyers often take the roots and grow sprouts from them for planting; this is a way of introducing a new variety of virus. Survival of the viruses between crops or cropping seasons occurs in vines left in the field after harvest, in storage roots discarded in the field or kept as a source of planting material, or in wild *Ipomoea* species such as morning glory.

IMPACT

SPVD is the most serious disease of sweet potato in Africa and perhaps the world. It is particularly severe in East Africa, with losses of 50-90% in the yield of susceptible plants. However, because of the disease, farmers now mostly plant resistant varieties, and in most districts of Uganda and Kenya virus symptoms only occur in 10-20% of plants, with about a fifth to a third of those infected with both SPFMV and SPCSV. Therefore, the real impact of the disease may not be a direct loss due to SPVD, but an indirect one because farmers are forced to grow low-yielding varieties that are resistant to SPVD.

DISTRIBUTION

The disease occurs throughout sub-Saharan Africa; it is particularly common in the Great Lakes region. Both SPCSV and SPFMV occur worldwide in all the sweet potato regions of Asia, North and South America and Oceania.

FURTHER READING

Clark CA, Davis JA, Abad JA, Cuellar WJ, Fuentes S, Kreuze JF, Gibson RW, Mukasa SB, Tugume AK, Tairo FD, Valkonen JPT (2012) Sweet potato viruses: 15 years of progress on understanding and managing complex diseases. Plant Disease 96(2):168-185. (http://apsjournals.apsnet.org/doi/pdfplus/10.1094/PDIS-07-11-0550).

Internal cork disease of sweet potato (Sweet potato feathery mottle virus). Plantwise Knowledge Bank. (http://www.plantwise.org/KnowledgeBank/Datasheet.aspx?dsid=50963).

O'Sullivan J, Amante V, Norton G, van de Fliert E, Vasquez E, Pardales J (Undated) Sweet potato virus disease. Sweet potato DiagNotes: A diagnostic key and information tool for sweet potato problems. (http://bit.ly/1URrLY7).

Sweet potato virus disease. CABI Crop Protection Compendium. (http://bit.ly/1HXv4Y7).

Moyer JW, Jackson GVH, Frison EA (eds.) (1989) FAO/IBPGR Technical Guidelines for the Safe Movement of Sweet Potato Germplasm. Food and Agriculture Organization of the United Nations, Rome/International Board for Plant Genetic Resources, Rome. (http://www.bioversityinternational.org/uploads/tx_news/Sweet_potato_503.pdf).

Yam dieback

Colletotrichum gloeosporiodies

Photo: Grahame Jackson, CABI, CC BY 4.0

Brown irregular leaf spots with yellow margins, expanding or joining together to cause a blight or early leaf fall.

Photo: Grahame Jackson, CABI, CC BY 4.0

Mature leaves blackened during heavy rains followed by sunny days.

SUMMARY: Yam dieback (*Dioscorea alata*), also known as anthracnose of water yam, is caused by the fungus *Colletotrichum gloeosporioides*. It results in dark leaf spots, blotches, defoliation and stem dieback of young shoots, blackening of older foliage and reduced tuber yields. Sources of infection are spores from other crops, weeds and infected tubers. Spread is via wind and rain, and diseased tubers. The disease is managed by using tolerant varieties and by planting early, ahead of the heaviest seasonal rains.

KEY SIGNS

Anthracnose means 'coal disease'. It describes diseases caused by fungi that produce dark spots on leaves, petioles, stems and fruits. Yam anthracnose is a disease of water yam, also known as the greater yam, *Dioscorea alata*, although there are suggestions that it is widespread and severe on most cultivated yam species, including *D. rotundata*. This fact sheet describes the disease on water yam.

Symptoms vary according to the age of the leaf, the variety and the amount of rain. On young leaves, small dark brown spots occur with yellow margins (2-20 mm diameter) that expand rapidly as leaves approach full size. Sometimes the spots run together to form large irregular blotches, and 'shot-holes' occasionally develop as the centres fall out. Infected leaves usually fall off. On more resistant varieties, only the young leaf veins are infected; in this case the leaves become cup-shaped as they grow. On older leaves, pinpoint spots are present that do not expand.

Epidemics can occur when there are several days of heavy rains. Leaves and stems of newly emerged shoots are infected and killed, and dieback results; other shoots develop from the planting piece, and these too are killed. Small tubers from these shoots are seen at harvest. On older plants, young growth hanging down from the tops of supporting poles is similarly infected and dies back. During epidemics, mature leaves of susceptible varieties rapidly turn black, a host plant response to massive numbers of germinating spores and exposure to sunlight. Stems, too, develop similar symptoms of intense but superficial blackening on the sides facing the sun. The blackening is so uniform and sudden on susceptible varieties of yam that farmers in some regions say the yams have been struck by lightning.

MANAGEMENT

Prevention – what to do before signs are seen

Cultural approaches: Use of tolerant varieties is an important part of any management strategy for yam anthracnose, and this may mean the sharing of varieties between countries. However, the unrestricted movement of varieties of yam from one country to another could spread strains of yam anthracnose that are not uniformly distributed: only pathogen-tested plants growing as sterile tissue cultures and following the FAO/IBPGR (1989) *Technical Guidelines for the Safe Movement of Yam Germplasm*[1] should be moved.

[1] http://www.bioversityinternational.org/e-library/publications/detail/sweet-potato

Many cultural practices, such as planting maize around the crop, early staking, weeding, avoiding damage at harvest, and also use of fungicides have been suggested for the control of anthracnose but convincing evidence that they are useful is lacking. However, two measures are highly recommended: use of tolerant varieties and early planting.

No variety is totally resistant to anthracnose, but there are some with greater tolerance. Selections have been made and distributed by international and national research organisations in West Africa. Some performed well and have shown stable anthracnose response across different agroecological zones; for example TDa 87/01091, TDa 95/00197, TDa 95/ 00010, TDa289, TDa291, TDa 294, TDa297, TDa 95/ 00328 and TDa1425. The varieties Belep (New Caledonia), Kinabayo (Philippines), Oriental (Barbados) and Plimbite (Haiti) have shown tolerance to anthracnose in many countries. Farmers should check to see if any of these selections are available.

In all countries, early planting, so that vines reach the tops of their supporting poles ahead of the storm season, is strongly recommended as it improves the performance of all varieties against anthracnose.

It is essential to check each planting piece, whether whole tubers or sections, and discard or cut out parts with rots. This should be standard practice by all smallholders, not only to remove anthracnose infections, but also rots caused by nematodes.

During crop growth, remove weeds that may be alternative hosts, but avoid moving through the crop when the foliage is wet as this might spread spores of the anthracnose fungus.

After harvest, vines should be collected, buried or burnt. It is possible that the fungus can remain alive in the vines and produce spores for at least 4 months, so it could be a source of infection for new plantings.

Chemical approaches: Planting setts should be dusted with ash, or captan fungicide if this is affordable and available.

Foliar applications of fungicides are not recommended. They can delay but not prevent epidemics.

CAUSE

The fungus *Colletotrichum gloeosporioides* is the cause of yam anthracnose; this is the asexual state; the sexual state is known as *Glomerella cingulata*. There are many strains of *C. gloeosporioides* infecting a wide range of crops and weeds, some of which infect yam. In Nigeria, four strains have been described from yam, based on colour, spore characteristics, growth in culture, molecular analysis and aggressiveness, one of which may be a new species. Recent reports suggest that the fungus is also the cause of outbreaks of anthracnose on white yam (*D. Rotundata*), but this needs confirmation. It is possible that other fungi are present and are the main cause of anthracnose epidemics. Another unanswered question is whether more than one *Colletotrichum* species is involved; this is possible as symptoms are varied and it is known that this fungus has many strains.

Spread of the fungus between plants and nearby plantings is via spores in wind-driven rain. Over longer distances, it occurs as tuber infections, the potential of which have been recognised in recent years in the Caribbean, Pacific and West Africa. Survival of the fungus between seasons is not well understood, but sources of infection are likely from weeds, other crops (e.g. citrus and mango), yam tubers and possibly dead vines from the previous season's harvest.

IMPACT

Although *D. alata* is not the most important yam of West Africa, nevertheless, it is popular because of high yield potential, ease of propagation, early vigour and ability to store well. However, yam anthracnose is an economically damaging disease, with epidemics reducing the yield of early, high-quality varieties by up to 80% in West Africa, the Caribbean and the Pacific. Losses of this kind impact food security and also threaten genetic resources. Invariably, surveys have shown that farmers rank yam anthracnose as a limiting factor on production.

DISTRIBUTION

Anthracnose is found wherever *D. alata* is grown in the humid tropics. Across the yam belt of West Africa, the disease is reported from Benin, Cameroon, Cote d'Ivoire, Ghana and Nigeria. In Nigeria, where 75% of the world yam production occurs, the disease is particularly common in the humid forest agroecological zone.

FURTHER READING

Brunt, A.A., Jackson, G.V.H. and Frison, E.A. (eds.) (1989) FAO/IBPGR Technical Guidelines for the Safe Movement of Yam Germplasm.Food and Agriculture Organization of the United Nations, Rome/International Board for Plant Genetic Resources, Rome. (http://ecoport.org/Resources/Refs/IPGRI/yam.pdf).

Amusa NA, Adegbite A.A, Muhammed S, Baiyewu RA (2003) Yam diseases and its management in Nigeria. African Journal of Biotechnology 2(12):497-502. (http://www.ajol.info/index.php/ajb/article/viewFile/14878/58624).

Mathew MA, Winter S, Mignouna HD, Green KR, Asiedu R (2003) Molecular taxonomic, epidemiological and population genetic approaches to understanding yam anthracnose disease. African Journal of Biotechnology 2(12):486-496. (http://www.ajol.info/index.php/ajb/article/view/14877).

Jackson GVH, Newhook FJ, Winch J (revised Wright JG, Peters J (2000). Yam anthracnose. Pest Advisory Leaflet. Secretariat of the Pacific Community, Noumea, New Caledonia. (http://bit.ly/1HwveVr).

[1]http://www.bioversityinternational.org/e-library/publications/detail/yam

Dry rot of yam

Scutellonema bradys

Photo: IITA, Flickr, CC BY-NC 2.0, http://bit.ly/1LKoMgW

Damage by the nematode Scutellonema bradys *on white yam tubers.*

Photo: IITA, Flickr, CC BY-NC 2.0, http://bit.ly/1FV6y4g

Dry rot beneath the skin of white yam tubers. A healthy tuber is on the right.

SUMMARY: Dry rot, common to all food yams, is caused by the microscopic nematode *Scutellonema bradys*. It attacks tubers in the field and in storage resulting in loss of edible parts, lower quality and reduced market value. Tuber infections are important for the survival and spread of the nematode. Management is by using nematode-free setts (achieved by carefully inspecting planting material or treating with hot water to kill the nematodes), by reducing soil populations using legume fallows or rotating yams with crops resistant to attack, and by removal of crop debris.

KEY SIGNS

The nematode causing dry rot of yam has a wide host range, infesting all edible and wild yam species, and many other crops including some legumes (e.g. cowpea), cereals, vegetables and weeds.

The feeding of the nematodes produces cavities. These become darker as the rot progresses, normally to a depth of less than 2 cm. Externally, cracks appear in the skin, which becomes crinkly, and parts flake off revealing the dark rot beneath. Rots continue in storage and can lead to complete decay of the tuber as other rot-causing organisms take over. There are no above ground symptoms.

Sometimes external symptoms are not obvious on the tubers. Extensive rots can develop without cracking and flaking of the skin, and are only seen when the skin is scraped away.

MANAGEMENT

Prevention – what to do before signs are seen

Cultural approaches: As there is evidence of different strains of *S. bradys* and the unrestricted movement of varieties of yam from one country to another could spread them, transfers should only be made as pathogen-tested plants growing as sterile tissue cultures, following the FAO/IBPGR (1989) *Technical Guidelines for the Safe Movement of Yam Germplasm*[1].

No varieties of either *D. cayenensis/rotundata* or *D. alata* are known to be resistant. The main methods of controlling dry rot are by cultural methods, the most important of which is the use of clean, nematode-free planting material.

Before planting, carefully check each planting piece for dry rot symptoms by scraping away the skin; it is easier to check if tuber cuttings or mini-setts are used rather than whole tubers. Wiping the knife used to cut the setts with a cloth containing bleach is recommended, especially if the knife has cut infested tubers.

Coating the setts in wood ash is a traditional method that should be followed, even though it will not significantly reduce nematode numbers, although it may add beneficial potassium. But be careful with fertilizer applications. Adding cow dung

[1]http://www.bioversityinternational.org/e-library/publications/detail/yam

into the mound reduces nematode populations, as do applications of NPK fertilizer and phosphorus alone, but nitrogen alone increases populations.

Ideally, plant yams after a fallow period, although this is often difficult as pressure on land is intense and increasing; nor is it always obvious to farmers what crops to use as *Scutellonema* has a wide host range. However, restorative fallows using legume cover crops reduce nematode populations and the following are recommended: *Aeschynomene histrix, Puerariaphas eoloides, Mucuna puriens* and *Centrosema* spp.

Crops that should be avoided prior to planting yams or as yam intercrops are cowpea, sesame, green gram, pigeon pea, kenaf, okra, tomato and melon. Preference should be given to maize, groundnut, chilli, Indian spinach or sorghum.

Weeds should be kept to a minimum, especially those that are hosts of yam nematode, e.g. *Eupatorium, Synedrella* and *Chromolaena*.

After harvest, all undersized and rotten tubers should be collected and buried deeply or burnt.

Chemical approaches: Chemical control is not appropriate for the control of this disease as most nematicides are dangerous to use without training. They are also expensive and mostly unavailable to smallholders. By contrast, hot water treatment is inexpensive and beneficial. Yams should be immersed in water at 51°C for 10 minutes. As a thermometer is needed to ensure the water is kept at the required temperature, this may not be a viable method for smallholders unless they are trained. If used, farmers should test it on a few setts first, as a method to produce nematode-free setts for the next season's crop. Treatment should be applied near the end of dormancy but before shoots develop, not immediately after harvest.

CAUSE

Dry rot disease is caused by *Scutellonema bradys*. Morphological and molecular characteristics of the nematode differ across the West African yam belt, and there are also differences in pathogenicity. Samples from Benin showed the greatest variability.

Nematodes enter the developing yam tubers at the growing point, at places on the tubers where the shoots and roots emerge and also through cracks and damaged areas in the skin. They inject a hollow needle-like mouthpart, called a spear, into cells of the tuber and feed on their contents. Both males and females live in the tubers and soil around them; eggs are laid and the young develop into adults after several moults.

Spread of *Scutellonema* over short distance is in water within and above ground, and in soil on tools and shoes. However, the main method of spread over long distances is in setts used for planting. Survival of the nematode occurs on the roots of other crops and weeds, and in stored tubers.

IMPACT

Scutellonema causes four main problems on yams: (i) a reduction in the weight of diseased tubers, up to 30% less than healthy ones at harvest; this is due to moisture loss, and is worse in late harvested tubers in dry soil; (ii) lower quality tubers with reduced market value; (iii) a loss of edible portions, which increases the longer the yams are stored; and (iv) a loss of planting material. Long-term losses are estimated to be 50%; when wet rots in storage follow dry rots, losses can be 80-100%.

DISTRIBUTION

Although of African origin, *S. bradys* is now present on several continents. It has restricted distribution in Asia, Central, North and South America, but is widely distributed in the Caribbean. In Africa, it is present in Benin, Burkina Faso, Cameroon, Côte d'Ivoire, Gambia, Ghana, Mali, Nigeria, Senegal, Sudan and Togo.

S. bradys is common in West Africa. Surveys sampling tubers in markets across the yam belt have shown that highest populations occur in D. rotundata in the mid-altitude savannah, followed by the southern Guinea savannah. In Nigeria, these surveys have shown that approximately half the tubers at markets are infested with the nematode.

FURTHER READING

Brunt, A.A., Jackson, G.V.H. and Frison, E.A. (eds) (1989) FAO/IBPGR Technical Guidelines for the Safe Movement of Yam Germplasm. Food and Agriculture Organization of the United Nations, Rome/International Board for Plant Genetic Resources, Rome. (http://ecoport.org/Resources/Refs/IPGRI/yam.pdf).

Scutellonema bradys. CABI Crop Protection Compendium. (http://www.cabi.org.ezproxy.library.uq.edu.au/cpc/datasheetreport?dsid=49315).

Yam nematode (Scutellonema bradys).Plantwise Technical Factsheet. (http://www.plantwise.org/knowledgebank/datasheet.aspx?dsid=49315).

Bridge J, Coyne DL, Kwoseh CK (2005) Nematode parasites in tropical root and tuber crops (excluding potatoes). In: Luc M, Silora RA, Bridge J (eds) Plant parasitic nematodes in subtropical and tropical agriculture (second edition). CABI Publishing, Wallingford, UK, pp 221-258. (http://bit.ly/1LX3XvF).

Yam mosaic disease

Yam mosaic potyvirus

Photo: IITA, Flickr, CC BY-NC 2.0, http://bit.ly/1L09rc5

Plant with yam mosaic disease, showing narrow yellow leaves.

Photo: IITA, Flickr, CC BY-NC 2.0, http://bit.ly/1TunWXa

Yellow and green patterns, distorted margin, and backward curling of a leaf infected with yam mosaic virus.

SUMMARY: There are at least four viruses infecting edible yams in West Africa, of which yam mosaic virus is the most important in distribution, incidence and effect on plant growth. Infected plants show mosaics, distortions and, in severe cases, strap-like leaves, poor growth and smaller tubers than those from healthy plants. Spread is by tubers used for propagation and also by aphids. The most important management techniques are use of new varieties from breeding programmes, controlling weeds and on-farm selection of healthy plants as 'seed'.

KEY SIGNS

Yam mosaic virus is the most important virus of yams infecting all the edible species, including the *Dioscorea cayenensis-rotundata* complex*, D.alata, D. esculenta* and *D. trifida.*

A variety of symptoms are produced depending on the species and variety. Commonly, plants show yellow and green patterns, called mosaics; these are mostly between the veins or in narrow green strips bordering them, in which case the symptom is called vein-banding. In more severe cases the foliage shows shoe-string symptoms, so called because the leaves are long, thin and strap-shaped, and the plants may also be stunted.

A common feature of the disease is the apparent recovery of some plants soon after infection when symptoms disappear. The virus is, however, still living in the plants; those infected may be slower to sprout and show poor vigour.

MANAGEMENT

Prevention – what to do before signs are seen

Cultural approaches: As there is evidence of different strains of yam mosaic virus, and the unrestricted movement of varieties of yam from one country to another could spread them, transfers of yams should only be made as pathogen-tested plants growing as sterile tissue cultures, following the FAO/IBPGR (1989) *Technical Guidelines for the Safe Movement of YamGermplasm*[1]. There are ELISA-based methods for detection of the virus as well as PCR methods using specific primers.

No varieties of either *D. cayenensis-rotundata* or *D. alata* are known to be resistant, but varieties have been bred or selected at the International Institute of Tropical Agriculture, Ibadan, Nigeria, from the world's germplasm for good performance: these should be sought for testing in areas where viruses are of concern.

Growers can also make selections on farm. Yams for propagation should be from the healthiest plants, invariably those that produced the largest tubers. 'Seed' yam producers should always choose tubers this way, perhaps using the mini-sett technology to accelerate multiplication.

[1] http://www.bioversityinternational.org/e-library/publications/detail/yam/

Control – what to do after signs are seen

Cultural approaches: Weeds should be kept to a minimum, as many species are hosts to aphids. Aphid populations build up on weeds and then the insects migrate, probing plants as they go for suitability as new hosts. Although the aphids may not remain on the yams, they can still spread the virus as they travel through the crop.

After harvest, all plant debris should be collected and destroyed. This includes undersized tubers, which should be eaten rather than kept for planting.

Chemical approaches: Chemical control is not appropriate for the management of this disease. Insecticides can kill the aphids that spread the virus, but that does not necessarily prevent virus infection. This is because the time between an aphid sucking up the virus when it feeds on a diseased plant, and spreading the virus as it feeds again on a healthy plant is short; by the time the insecticide has killed the aphid it has already spread the virus.

CAUSE

The virus was first isolated and characterised from *Dioscoea cayenensis* from Ivory Coast in 1979: from serological, molecular and epidemiological analysis it is now known to be a genetically diverse potyvirus. Several aphids spread the virus, including *Aphis fabae, A. craccivora, Rhopalosiphum maidis, Toxoptera citricidus, Myzu spersicae* and *A. gossypii*; however, the relative importance of each of these species is unknown. Spread by aphids is done in a non-persistent way; this means that they acquire the virus on their mouth parts after a short feed on an infected plant (less than a minute), infect a healthy plant after another short feed, but then lose the ability to infect.

Spread of the virus by aphids is important, but so too is spread by vegetative propagation. The virus passes from the planting sett to the developing plant and then to its tubers. Farmers may inadvertently help in the process by eating or selling the largest tubers and saving the smallest for 'seed' for next season's crop: the smallest may have not grown well because of virus.

IMPACT

Yam production is adversely affected by virus diseases. Infection reduces the number and size of tubers and also their starch content. Tests have shown that a 40% loss of yield is possible in susceptible varieties. Apart from affecting the growth of plants, the virus also restricts the international movement of germplasm.

The virus rarely occurs alone and is often associated with, for example, *yam mild mosaic virus, yam badnaviruses* and *cucumber mosaic virus*. All four viruses occur in Nigeria, Ghana, Benin and Toga; tests have shown that 100% of tubers and nearly 70% of leaves contained at least one of the four viruses and 30% of the tubers had mixed infections. The importance of *yam mosaic virus* among the four is supported by surveys in the Guinea savannah of Nigeria that found the virus to be more widespread than the others, occurred in more mixed infections and more often in *D. cayenensis-rotundata*, the most important species.

DISTRIBUTION

The virus spreads in *D. cayenensis-rotundata* and *D. esculenta* throughout West Africa – Nigeria, Benin, Ghana, Ivory Coast, Benin and Burkina Faso – and is occasionally found in *D. alata* in Nigeria. It is also reported from Guyana in South America and Jamaica, Barbados, Guadeloupe and Puerto Rico in the Caribbean. The report of the virus in the South Pacific needs confirmation.

FURTHER READING

Asala S, Alegbejo MD, Boniface, Olalekan Kashina B, Banwo OO, Asiedu R, Lava-Kumar P (2012) Distribution and incidence of viruses infecting yam (Dioscorea spp.) in Nigeria. GJBB, 1(2): 163-167. (http://bit.ly/1PoXvQx)

Brunt AA, Jackson, GVH, Frison EA (eds) (1989) FAO/IBPGR Technical Guidelines for the Safe Movement of Yam Germplasm. Food and Agriculture Organization of the United Nations, Rome/International Board for Plant Genetic Resources, Rome. (http://ecoport.org/Resources/Refs/IPGRI/yam.pdf)

Eni AO, Hughes J d'A, Rey MEC (2008) Survey of the incidence and distribution of five viruses infecting yams in the major yam-producing zones in Benin. Annals of Applied Biology, 153: 223-232. (http://bit.ly/1FgYn6S)

Legg JP, Gibson RW, Hughes J d'A (Undated) Virus diseases of root crops in Africa: an overview. FAO, Rome. (http://www.fao.org/docs/eims/upload/agrotech/2005/sistrc_04_legg.pdf)

Offei SK (Undated) Virus and viral diseases of sub-Saharan Africa: analysis of responses to questionnaires by scientists in sub-Saharan Africa.(http://bit.ly/1L8XLSQ)

Séka K, Etchian AO, Assiri PK, Toualy MNY, Diallo HA, Kouassi NK, Aké S (2014) Yield loss caused by yam mosaic virus (YMV) and cucumber mosaic virus (CMV) on the varieties of Dioscorea spp. International Journal of Agronomy and Agricultural Research, 5(2): 64-71. (http://bit.ly/1gvBlA5)

Jackson GVH, Newhook FJ, Winch J (revised Wright JG, Peters J (2000). Yam anthracnose. Pest Advisory Leaflet. Secretariat of the Pacific Community, Noumea, New Caledonia. (http://bit.ly/1HwveVr).

Late blight of Irish potato

Phytophthora infestans

Illustration: Margaret Senior, NSW Department of Primary industries, http://bit.ly/1IMAEdo

Composite of early lesions on leaves and further development of larger, dark brown patches as foliage withers. Note darkening of stem and rotting of tuber.

Photo: Eric Boa, CABI, CC BY 4.0

The disease develops rapidly once spores infect leaves and late blight takes hold, eventually killing the whole plant.

SUMMARY: Late blight of potato, caused by a fungus-like organism, is a constant threat wherever potatoes are grown. Whole fields can be wiped out in a short time. Selection of clean, healthy and preferably certified tubers is essential, as is removing left-over tubers from previous potato crops. It is difficult to avoid the use of fungicides given the risk of losing whole crops. Most fungicides are used as protectants: some act systemically and others on contact with foliage, with the common aim of preventing disease establishment. Resistant varieties are an important part of an integrated disease management strategy but the risk of disease outbreaks is too high to rely only on this approach.

KEY SIGNS

Symptoms are usually first seen on leaves before spreading to all other parts, including the tubers, and killing the plant. If infected tubers are planted then symptoms appear low down on the main stems.

Diagnosis of early symptoms can be tricky. The leaf symptoms vary according to growing conditions and potato variety. They begin usually as irregularly shaped small leaf spots, pale to dark green, which spread rapidly, unrestricted by the leaf veins. Look out for characteristic browning of veins on the lower leaf surface on young lesions. Leaves dry up and go brown. Pale green to yellow halos develop around the dying (necrotic) areas but note the rapidity with which plants are killed. Black or brown lesions occur on stems and leaf stalks. Infected tubers go brown outside and rot inside. Other fungi and bacteria spread the rot in stored tubers.

Under humid conditions a white, furry growth may occur at the edge of dying leaf areas, indicating spore production. Potato powdery mildew also produces a similar growth but without the rapid death of leaves and decline of the plant. Other leaf diseases produce similar symptoms though generally lack the rapid progress and blackening of the whole plant. If unsure about the presence of late blight, put leaves in a plastic bag overnight and look for fluffy growth (sporulation) the next day.

If a small patch of blight appears, remove and destroy plants by burying in the ground where they are planted. Remove soil and wash tools used before drying off. Great care should be taken to avoid moving spores to new areas.

MANAGEMENT

Prevention – what to do before signs are seen

Cultural approaches: Late blight resistant varieties are available such as Asante and Tigoni in Kenya, Meru, Tengeru 97 and Shengena in Tanzania and Cruza in Rwanda. Check on new resistant varieties and which ones are available in the local area. New cultivars regularly appear.

Always use certified seed potato. Before planting, examine tubers for brown patches, though these are difficult to see in

darker varieties of potato. If uncertain about the health of seed potatoes, test a small batch by letting them sprout for 10 to 15 days at 15 to 20°C. Diseased tubers will rot.

Mound the potatoes in fields and keep the tubers covered to limit infection by spores washed down from leaves. In some countries, farmers grow potatoes during periods of low rainfall to avoid late blight, but yields are much lower.

Phytosanitation and the use of healthy and resistant material is important but late blight can still develop and cause major damage. Stay alert for cool, moist conditions favourable to disease development and any announcements on radio or through extension officers about the risk of late blight.

Chemical approaches: Late blight is a devastating disease, one which takes advantage of any opportunity to infect and invade plants quickly and spread to new areas. The choice and use of fungicides is complex and controversial because the pathogen and fungicide options are constantly changing.

Some active ingredients used against late blight are systemic though the majority work on contact with plant surfaces, preventing air-borne spores that land on leaves from growing. Some are translaminar – these penetrate the leaf but not the leaf stalk and stems. A popular recommendation is to use commercial products which contain two active ingredients with different properties, usually either systemic + contact, or contact + translaminar.

Cymoxanil and dimethomorph are systemic and have curative properties. They are often combined with mancozeb, a commonly used contact fungicide. But the curative effect only works one or two days after infection. The spraying schedule for fungicides must be rigorously observed since any gaps allow the late blight pathogen to enter the crop. Metalaxyl, a systemic fungicide is still widely used but should be avoided because of pathogen resistance. Fungicides with cymoxanil, which kills spores ('stop effect'), should be applied at 5-7 day intervals. Other fungicides are more commonly sprayed at 7-10 intervals.

The maximum number of applications varies from four (cymoxanil by itself) to ten (zoxamide and mancozeb) according to recommendations in the UK. Alternate fungicides containing active ingredients from different chemical groups to avoid inducing resistance. Further information on fungicides is available from various sources listed below.

Control – what to do after signs are seen

Cultural approaches: Remove infected tubers and piles of potatoes rejected after harvest to minimise carryover of the pathogen to the next crop.

CAUSE

Late blight is caused by *Phytophthora infestans*, an oomycete. It is not a true fungus, even if it is still referred to as one; a better term is fungus-like or water mould.

Late blight also attacks tomato and other members of the Solanaceae (potato family), but the strains of *P. infestans* are different and do not cross-infect. There are many *Phytophthora* species which attack many different types of plants, including trees. Many species cause huge damage.

The disease is spread by wind-borne spores and by water splash. Spores are washed into the soil where they can infect tubers. Potato plants are most at risk from late blight under cool and moist conditions.

IMPACT

Late blight of potato is a devastating disease and a major threat to potato production. Once it takes hold it is difficult to control. Whole fields are affected, with total loss of production. The threat and risk from the disease is highest in damp and cool conditions, most often found at higher altitudes in tropical countries.

DISTRIBUTION

Present in all potato growing countries in sub-Saharan Africa.

FURTHER READING

Plantwise (www.plantwise.org)

Crop Protection Compendium (www.cabi.org/cpc).

Infonet-Biovision has details of Irish potato varieties available in Kenya (www.infonet-biovision.org).

CIP (www.cip.org) and the Roots, Tubers and Bananas research programme (www.rtb.cgiar.org).

There are many sources of extension literature on potato blight, particularly from North America and Europe. Be cautious in applying knowledge to Africa since growing conditions, cultivars etc will vary. See: University of Massachusetts (http://extension.umass.edu/vegetable/diseases/late-blight) and Potato Council (www.potato.org.uk/knowledge-hub/publications/late-blight-publications-and-research-reports)

Rats and mole rats of sweet potato

Mastomys, Arvicanthis & Tachyoryctes species

Photo: William T. Stanley, Negaunee Collection Manager, Mammals, The Field Museum of Natural History

Natal multimammate rat, Mastomys natalensis.

Photo: Jonathan Beilby

Grass rat, Arvicanthis *species.*

SUMMARY: Rats and burrowing mole rats both attack sweet potato, but rats are the more serious pests. Although 12 species of rat are crop pests, only the multimammate and grass rat are distributed widely. Rapid population outbreaks, in East Africa at least, may be related to rainfall in December and January that encourages early breeding. Control relies on community action and early intervention, mostly using cultural methods. Chemicals are a potential danger to human health and the environment, as well as being costly and mostly unavailable to smallholders.

KEY SIGNS

Rats feed on sweet potato storage roots and can cause serious damage by digging through mounds or ridges to eat them, or by attacking them when exposed above ground. Mole rats burrow into the soil, eating storage roots from below.

MANAGEMENT

Prevention – what to do before signs are seen

Cultural approaches: A major difficulty with managing rats and mole rats is that farmers mostly wait until they see damage before taking action and, by then, the damage is far advanced and irreversible. There is also the problem of ineffective or dangerous methods being used, such as flooding burrows and use of toxic chemicals:

To overcome these problems, ecologically-based rodent management (EBRM) has been developed. This has been necessary because rats have become resistant to chemicals used previously, and also due to a better understanding of their risks to human health and the environment. The new approach aims to give farmers an appreciation of rodent biology and ecology and the factors influencing population changes of rats.

EBRM is based on two important approaches: (i) community action and (ii) early intervention. It uses the traditional methods devised by farmers, applying them when they will have greatest impact based on knowledge of rodent biology and ecology. This will be different for each species.

It may be possible to alter the time of planting so that harvests occur before maximum rat populations; there is also the possibility of selecting early-maturing varieties in localities where rats are a recurring problem.

During crop growth, procedures likely to reduce rat and mole rat populations and protect the growing crop include setting traps (snap, snare or live), although care must be taken to protect livestock and children from being hurt by them.

Encouraging owls is a practical way to control rats, but needs to be done with respect to community beliefs, as farmers may be superstitious (some communities consider owls evil omens, symbols of death and bad fortune). The reason for placing owl nesting boxes around the village and fields should be carefully explained. Experience shows that farmers will quickly see the wisdom of attracting owls compared to the damage done by rats. Contrary to most people's belief, cats are not very effective; they do not kill many rats, but only frighten them away.

Weeds should be removed from within and around sweet potato fields. Nile rats forage during the daytime and also nest above ground; removing weeds exposes them to predators. Previous outbreaks involving this species and also the multimammate rat have seen more damage in weedier plots of rice; the same may hold true for sweet potato.

Where losses are likely, early harvesting is an obvious solution and should be promoted.

Some other cultural control remedies used by farmers in Kenya, Uganda and Zambia, include:

- Planting the legume *Tephrosia vogelii* (commonly known as fish bean) randomly throughout the field and along the borders. This shrub contains rotenone, a fish poison and insecticide, so be careful when disposing of it.
- Using a mixture of cow dung and pepper placed in the burrows and burnt to smoke out the rodents.
- Pouring one-week old fermented cattle urine into the burrows to chase away mole rats.
- Digging deep ditches around sweet potato plantings to stop rodents from tunnelling straight into the field.

After harvest of sweet potato crops, collect the debris and bury or burn it; importantly, do not leave immature or broken storage roots in the field for rats to eat. Also, protect the harvest in storage from rat infestations.

Chemical approaches: Chemical control is not an appropriate method of managing this disease. Although fungicides would be effective, they are too expensive for most smallholders and often they are unavailable. If required in commercial planting, mancozeb or copper compounds would be suitable choices.

Control – what to do after signs are seen

Cultural approaches: A home remedy is based on use of the legume shade tree, *Gliricidia sepium* (it means 'rat killer'). Pound young leaves or bark, and mix with cooked rice, maize or other bait, or boil the *Giricidia* with rice or other cereal grains. Bacteria convert chemicals in the leaves to substances similar to brodifacoum (an anti-coagulant used as a rat poison). These are less toxic than brodifacoum, so larger amounts must be eaten. Change the bait daily and protect children and pets by placing it in bamboo sections or tins.

CAUSE

Rats feed on sweet potato storage roots and can cause serious damage by digging through the mounds or ridges to eat them, or by attacking them when they are exposed above ground. Rats live above ground and nest on or in the ground or in trees, depending on the species. Species that cause outbreaks have high reproductive capabilities. The gestation periods of the multimammate rat and the grass rat are only 23 and 18 days respectively; as soon as they give birth they can conceive again. Mole rats burrow into the soil, eating storage roots from below. Mole rats live and breed in underground burrows. For mole rats, gestation is longer at about 7 weeks.

The following reasons have been suggested for rat outbreaks: (i) a long rainy season that provides more food and cover, allowing better survival (ii) a reduction in competition from other rat species, predators and disease, when there is a return to rains after consecutive dry years (iii) early breeding, when unusually heavy rains occur during the rainy season and the progeny join the main breeding season population later in the year.

Research in Tanzania has found that cumulative rainfall for December and January could be used to predict an outbreak 6 months in advance. Furthermore, calculations showed that if rainfall exceeded 366mm for December and January then control actions would be cost-effective, taking into account the amount of damage that was likely.

Africa has almost 400 rodent species but only about 5% are crop pests. Of these *Mastomys natalensis* (multimammate rat) and to a less extent *Arvicanthis* species (grass rats) are the dominant rodent pests of sub-Saharan Africa, and most often involved in rodent population explosions. Other species attacking sweet potato are the burrowing orange-toothed mole rat (also known as the East African mole rat, African mole rat or root rat) (*Tachyoryctes splendens*) and the greater cane rat (*Thryonomys swinderianus*), which is relatively slow breeding.

IMPACT

There were 32 recorded outbreaks of the multimammate rat in Africa from 1925-2005. M. natalensis was the most frequently involved, either alone or in a few instances with other Mastomys species or with Arvicanthis species. Most outbreaks occurred in East Africa, although a few were in southern Africa and West Africa, with a major outbreak across the Sahel in 1975-76. Most attention has been paid to impacts on maize, rather than root crops, so the loss of yield under normal circumstances and in outbreak years is not well documented.

DISTRIBUTION

Multimammate and grass rats are widely distributed and common. *M. natelensis* occurs over most of the continent, with other *Mastomys* species more locally present. *Arvicanthis* species live in the northern half of the continent. Both live in grasslands and wooded savannah, cultivated areas and in villages. By contrast, the orange-toothed mole rat occurs in the moist uplands of Ethiopia, Somalia, Kenya, Tanzania, Uganda and eastern Democratic Republic of the Congo.

FURTHER READING

A better rat trap. Journey to Forever. (http://journeytoforever.org/at_rats.html).
FARM: Forum for Agricultural Risk Management in Development. (http://bit.ly/1URsLLQ).
Fiedler LA (1994) Rodent pests management in eastern Africa. FAO Plant Production and Protection Paper 123. Rome: Food and Agriculture Organization of the United Nations. (http://bit.ly/1K2sdwp).
Singleton GR, Hinds LA, Leirs H, Zhang Z (1999) Ecologically-based management of rodent pests. ACIAR Monograph No. 59. 494p. (http://bit.ly/1O3SonP).
Singleton GR, Belmain SR, Brown PR, Hardy B, (2010) Rodent outbreaks: ecology and impacts. Los Baños (Philippines): International Rice Research Institute. 289p. (http://bit.ly/1Hzwz0p).

7. The major pests and diseases of bananas

This section covers the most important pests and diseases that affect bananas in Africa. Table 7.1 summarises the main signs associated with these diseases. In the following pages more details on the signs, management options, cause and impact are provided.

Table 7.1: Key signs of the most important pests and diseases of bananas in Africa.

Key signs	Possible pest or disease	See page
Weak or dying suckers. Yellow floppy leaves. Small bunches. Tunnels caused by grubs in corms.	Banana weevil	122
Red-brown streaks at edge of leaf blade. Dead areas either side of leaf midrib.	Black sigatoka	124
Yellowing of margins of older leaves. Leaves turn brown, dry and collapse.	Fusarium wilt	126
Leaves small, crowded together, erect not arching outwards. Dark green streaks on minor veins in leaves.	Bunchy top	128
Premature ripening of fruit. Young leaves go yellow, fold in centre and collapse.	BXW	130

Banana weevil

Cosmopolites sordidus

Photo: Scot Nelson, Flickr, CC BY-SA 2.0, http://bit.ly/1allhxe

Damage caused by banana weevil.

Photo: Scot Nelson,Flickr, CC BY-SA 2.0, http://bit.ly/1IJWY7t

Adult banana weevil.

SUMMARY: The banana weevil is a serious pest to bananas worldwide. A combination of clean planting material, destruction of crop residues and neem can be used to reduce weevil populations; however, movement of banana weevils from neighbouring farms can reduce the efficacy of any management options.

KEY SIGNS

The banana weevil is a major pest that attacks all varieties of bananas and plantains (*Musa* spp.) as well as ensete (*Ensete* spp.).

Adult weevils are about 10-16 mm long and are black or dark brown in colour. Adult weevils are commonly found in the soil around the base of the plant, between leaf sheaths and under plant debris and mats. Adults have a long life (about two years) and can survive for long periods of time without food. The adults are most active at night and have the ability to fly, although they rarely do.

The larvae (or grubs) are creamy-white with a reddish-brown head, legless and about 12 mm long. The grubs cause the most damage to the plant by boring into the base of the pseudostem, rhizome (or corm), suckers and roots. Tunnelling into the corm causes the most severe damage including fungal infection and reduced nutrient uptake, stem growth and stability.

Symptoms include tunnels, weak or dying suckers, yellow floppy leaves and smaller bunches. Plants that are heavily infested will show extensive damage to the pseudostem and a severe infestation can cause plants to fall over. Infested corms will be full of tunnels up to 1.5 cm in diameter. To identify the pest, cut open the corms with tunnels to see if larvae are present. Stunted plants and plants displaying early withering of leaves should also be inspected for larvae and adult weevils. Cut them with a knife at or just above ground level and search for larvae or larval tunnels.

Adults are attracted to freshly cut corms and pseudostems which can be used as traps for monitoring. To make a pseudostem trap, cut small pieces of pseudostem in half lengthwise (about 25 cm long) and place the cut pieces on the ground near the base of the plant, with the cut surface facing down. After 5 days, check the traps and count the weevils on each trap (and kill the weevils). If more than 2-5 weevils per trap are found, control is necessary. These traps are best used when the weather is warm and wet. About 50 traps/hectare are needed to provide good coverage, and the traps should be set once per month.

MANAGEMENT

Prevention – what to do before signs are seen

Cultural approaches: Strict regional quarantine measures to ensure the availability and use of clean planting material are important to protect new plantings and prevent the spread of the insect.

It is extremely important that farmers use clean planting material. Farmers should inspect the corms and suckers for damage and discard any that are infested. Take one or two slices from the planting material and look for tunnels, larvae or pupae. If infested, destroy the planting material.

If clean planting material is not available, then trimming the material to remove the parts infested with eggs and larvae, and using a hot water treatment are the next best options. Remove the outer leaf sheath of suckers to get rid of weevil eggs. Submerge the clean trimmed suckers in a hot water bath (52 to 55°C) for 15 to 27 minutes before planting. Neem is also effective for cleaning infected planting material. Dip suckers in a 20% neem seed solution just before planting.

Entomopathogenic fungi, such as *Beauveria bassiana* and *Metarhizium anisopliae*, and other biological control agents (e.g. arthropods such as ants) are being tested and developed and may provide more options in the future.

Control – what to do after signs are seen

Cultural approaches: Destroy all infected materials and crop residues after harvest to reduce populations.

Do not replant previously infested areas while old corms remain in the ground. Remove all the corms after the final harvest, cut them into 4-8 pieces and allow them to dry to prevent larval development in harvested plants. Allow three months for the weevil population to die out, before replanting a field.

Chemical approaches: Neem powder can be used to control banana weevils. Apply 60-100 grams/mat of neem seed powder or neem cake around the base of the plant. Apply at planting and again every four months.

Chemical control is often used in large plantations, but is often beyond the means of small-scale farmers. The banana weevil is reported to have developed or shown signs of resistance to several pesticides, including cyclodienes (BHC, dieldrin), organophosphates (chlorpyrifos, ethoprophos, pirimiphos-ethyl and prothiophos) and carbamates (carbofuran). Treatments used to control nematodes often control weevils too. Active ingredients that are effective against banana weevils include fosthiazate, azadirachtin, fipronil and imidacloprid. If the bananas are for export, any chemicals used must abide by the rules and regulations in the importing country.

CAUSE

The banana weevil (*Cosmopolites sordidus*) is also known as the banana borer or banana root borer. The insect is spread between plantations, districts and countries by the transport of infested parts of banana plants, especially the corms. Adults can easily move from one banana plant to the next.

IMPACT

The banana weevils can decrease root growth, reducing nutrient uptake and plant vigour, leading to small fruit bunches and yields, and weakening the overall stability of the plant. Infestations in newly planted fields can lead to crop failure. Highland bananas and plantains are more susceptible than dessert or brewing varieties.

DISTRIBUTION

The banana weevil originated in Malaysia and Indonesia, and is currently found in all major banana producing regions of the world. It can be found throughout East and West Africa including Burkina Faso, Ghana, Mali, Nigeria, Tanzania, Uganda and Kenya.

FURTHER READING

Plantwise Knowledge Bank www.plantwise.org/knowledgebank

Gold, C., Pena, J., and E. Karamura. Biology and integrated pest management for the banana weevil Cosmopolites sordidus (Germar) (Coleoptera: Curculionidae). Integrated Pest Management Reviews, 2001, volume 6, issue 2, pp. 79-155.

PIP Guide to Good Crop Protection Practices for production of Bananas. 2011. www.coleacp.org/pip

Bioversity International http://www.bioversityinternational.org/

ProMusa www.promusa.org

Black sigatoka of banana

Mycosphaerella fijiensis

Photo: Grahame Jackson, CABI, CC BY 4.0

Brown streaks with yellow areas between; the spots have joined together at the leaf margin causing a blight.

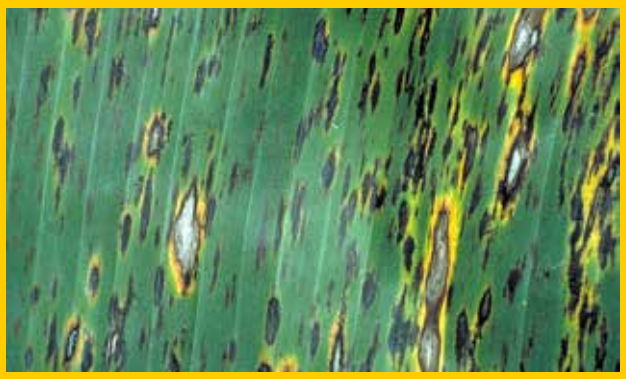


Photo: Grahame Jackson, CABI, CC BY 4.0

Close-up of brown elongated spots, most with yellow margins, and some with grey centres.

SUMMARY: Black Sigatoka, caused by the fungus Mycosphaerella fijiensis, is a leaf disease of banana and plantain worldwide. Spores are spread in wind and rain, and leaves die rapidly after infection, reducing fruit weight by 30-40% – less for plantains. Management is by using tolerant or resistant varieties. Some plantains are little affected, and resistant dessert and/or cooking varieties with Cavendish characteristics have been bred. Fungicides – protectant and systemic – exist for commercial plantations, but expense, availability and strategies to prevent fungal resistance, complicate their use by smallholders.

KEY SIGNS

The first sign of the disease is red-brown streaks, parallel to the veins, about 1-5 mm long by 0.25 mm wide. They are most noticeable on the underside of the third or fourth youngest leaf, especially along the edge of the leaf blade, which is first exposed as the leaf emerges. The streaks expand and become noticeable on the upper surface, darkening and later developing grey, slightly sunken centres with black margins and bright yellow halos. As the streaks join together they form bands of dead areas several centimetres wide, on either side of the midrib, and the leaves collapse and die.

MANAGEMENT

Prevention – what to do before signs are seen

Cultural approaches: The control of this disease is extremely difficult and is best done through use of tolerant or resistant varieties, and attention to some cultural practices that reduce the length of time that leaves are wet and can be infected by spores.

For planting, choose areas that have good drainage, avoiding those with heavy clay soils where surface water remains for some time after rains. Avoid areas where there is shade from trees. If planting on hillsides, choose sites with morning sun to dry leaves rapidly and always plant so that the rows run in the direction of the prevailing wind, to allow air into the plantation to dry the leaf surface, to prevent infection, or to reduce the number of spores. Planting at wide spacing, about 2.5 m apart (1600 plants to the hectare), will also help.

Use tolerant or resistant varieties. Many plantains are either tolerant or resistant (e.g. Mysore, Saba and Pisang Awak). These are dessert or dessert/cooking bananas with resistance to black Sigatoka. Yangambi Km5, a dessert banana from West Africa, also has black sigatoka resistance. Check whether these varieties are available locally.

Control – what to do after signs are seen

Cultural approaches: Collect infected leaves from the mother plant after harvest of the fruit and take them out of the plantation and burn them.

Chemical approaches: Deleafing and fungicide applications are necessary if Cavendish varieties are grown for the market. Deleafing (the removal of infected leaves) is done before spraying. Remove infected leaves, or parts of leaves. If less than 30% infection, remove only part of the leaf; if more than 40% remove the entire leaf. Take the leaves out of the plantation and burn them.

- Protectant fungicides (these stay on the surface of leaves): mancozeb (in oil or oil/water emulsion) and chlorothalonil (in water) during times when the disease is relatively low.
- Systemic fungicides (these move inside plants): triazoles (e.g. propiconazole, fenbuconazole and tebunconazole) and strobilurins (e.g. azoxystrobin).

It is important to rotate the fungicides in the different groups to prevent the build up of resistant strains of the fungus. No more than two applications of the same systemic fungicide should be made before changing to another group. In drier times, mancozeb can be used alone.

CAUSE

Black Sigatoka is caused by the fungus, *Mycosphaerella fijiensis*. It produces two types of spores: ascospores and conidia. Ascospores are produced when different strains fuse together (a form of sexual reproduction); conidia are produced by all strains without a sexual process. Ascospores are thought to be the most important, discharging from the upper surface of the leaves, infecting the new leaves as they unfurl and spreading the fungus to new areas in wind-driven rain.

Spores are produced in the dead, grey areas on the upper leaf surface. They are released during rains or during periods of high humidity and spread in wind and rain to leaves nearby or even to those in plantations far away. They land on the underside of emerging leaves, infect them and so the cycle begins again.

The fungus is also spread on infected planting material and on old banana leaves.

Another, similar fungus, called yellow Sigatoka (*Mycospeaella musicola*) also exists. This was the most important leaf disease before black Sigatoka was identified in the 1960s, although it had probably been present in parts of Asia and the Pacific long before. Today, black Sigatoka has replaced yellow Sigatoka in most regions of the world.

IMPACT

Black Sigatoka is the most serious leaf disease of banana and plantains, and is widespread throughout the tropics.

Leaves die early; instead of lasting 200 days as they should, they last only about 50 days. The result is fewer than 5-7 leaves at flowering, the minimum number needed to produce bunches of acceptable weight and quality.

The impact of black Sigatoka on banana production can be measured in different ways depending whether they are grown commercially or principally for household consumption, and whether bananas or plantains are the crop of concern. Estimates will vary in different regions of the world, but generally losses in Cavendish plantations due to black Sigatoka are 30-40% in terms of reduced yield and premature or uneven ripening of the fruit.

To overcome the potential losses, fungicides are applied which amount to about 30% of the costs of production, less if plantains are the commercial crop. Similar figures for smallholder production are hard to come by. However, it is known that worldwide black Sigatoka has caused growers to abandon the crop because they cannot afford fungicides. This in turn has resulted in loss of an important staple food, increased market prices because of reduced supply and loss of genetic resources as growers abandon the crop. The result is a threat to food security, of particular concern in Africa where 50% of the world's plantains are grown.

DISTRIBUTION

East, South and Southeast Asia, East and West Africa, Central and South America, the Caribbean and Pacific islands. It occurs on several islands in the Torres Strait, but not mainland Australia.

FURTHER READING

Black Sigatoka disease management (2012) Plantwise: Factsheets for farmers (ed Phillip Taylor) (http://www.plantwise.org/FullTextPDF/2013/20137804482.pdf).

Black Sigatoka. Fact sheet. Plant Health Australia. Queensland Government (http://www.planthealthaustralia.com.au/wp-content/uploads/2013/01/Black-Sigatoka-FS.pdf).

Mycosphaerella fijiensis. CABI Crop Protection Compendium. (http://www.cabi.org.ezproxy.library.uq.edu.au/cpc/datasheetreport?dsid=35278).

Ploetz, RC (2001) Black Sigatoka of Banana. The Plant Health Instructor. (https://www.apsnet.org/publications/apsnetfeatures/Pages/blacksigatoka.aspx).

ProMusa. (http://www.promusa.org/tiki-custom_home.php).

Fusarium wilt of banana

Fusarium oxysporum f.sp. cubense

Photo: Denis Persley, Department of Agriculture and Fisheries

Leaves turn yellow, first at the margins and later hang down.

Photo: Denis Persley, Department of Agriculture and Fisheries

Water-conducting parts of the leaf turn red, brown or black.

SUMMARY: Fusarium wilt of banana and plantains is a fungus that invades the roots and stem, blocking the water conducting channels and causing leaves to turn yellow, dry and collapse. It is spread through movement of soil, on equipment and especially on contaminated planting material. There are four strains: three can be contained by cultural methods and resistant varieties, but a recent variant (TR4) attacks dessert bananas and plantains, putting both industry and smallholder production at serious risk. Recently, the fungus has spread from southeast Asia to Australia, Jordan and Mozambique.

KEY SIGNS

The first sign is a yellowing at the margins of older leaves, advancing towards the midrib. Leaves turn brown, dry and eventually collapse. Disease symptoms move progressively from older to younger leaves until only a few of the youngest leaves remain green and erect, with the older ones forming a 'skirt' around the stem. Eventually, all the leaves will collapse. On some varieties the stems split as well. Internally, brown, red and yellow rings occur in the stem, at first at the centre and later, in cases of severe infection, spreading throughout the stem. Suckers may also show symptoms. Eventually, all parts above and below ground will die and rot.

MANAGEMENT

Prevention – what to do before signs are seen

Cultural approaches: Management of fusarium wilt is extremely difficult as the fungus remains alive in the soil for many years and there are no fungicides or cultural controls that can be usefully applied against it. The only solutions are to: (i) keep it out in the first place; (ii) establish methods for early detection; (iii) observe strict hygiene measures; and (iv) use resistant varieties.

The following advice applies to all strains of fusarium wilt. However, such is the threat from TR4 that national and regional biosecurity authorities need to ensure that the fungus does not spread within Africa (or elsewhere in the world) but, should a new introduction occur, have the necessary authority to apply quarantine regulations to limit its impact. Transfers of varieties between countries should follow the *Technical Guidelines for the Safe Movement of Germplasm*.

Tolerant and resistant varieties do exist. Some FHIA (Honduran Agricultural Research Foundation) varieties (e.g. 01, 02, 18, and 25) have resistance to TR4 and, in Taiwan, Cavendish variants from tissue culture have also proven resistant. Check whether these are available locally.

Never use suckers for planting from plants that show symptoms of fusarium wilt, even though they may appear to be healthy, as the fungus may be in the roots. Use clean planting material, preferably from disease-free certified plants. If these are not available, growers should be encouraged to take suckers only from areas that have not shown symptoms of the disease, preferably from growers' own farms which have been monitored for disease.

Spores of the fungus can be transferred in soil, so avoid sharing farm machinery and equipment (e.g. shovels, knives and ladders) with other growers, and remove debris and soil from machinery, equipment, vehicles and footwear before entry to any

farm, even farmers' own farms. Ensure that all members of the family (and other workers) know about the disease, and the hygiene rules to keep it out of the farm and/or under control.

Control – what to do after signs are seen

Cultural approaches: If an infected plant is detected, disturb the soil as little as possible. Dig out the plant with its root mass and suckers, and burn everything on the spot. Do not chop the plant into pieces. Use a herbicide (e.g. glyphosate) to hasten death of infected plants, then leave plants to die in place. Consider removing a ring of plants around the diseased one. This is done in case the disease has already spread to adjacent plants via root-to-root contact – never mind that they look healthy, they may develop the disease later.

If possible, fence infected areas so that people do not spread the disease in soil on footwear, and clean the tools used to remove plants with sodium hypochlorite bleach. As a future caution, dig drains to divert surface run-off (and irrigation) water if it flows through infested areas. Furthermore, do not replant with the same or another fusarium wilt susceptible variety, and monitor the remaining plants every two weeks.

CAUSE

Fusarium wilt, also known as Panama disease, is the most serious disease affecting bananas. It is caused by the soil inhabiting fungus *Fusarium oxysporum* f.sp. *cubense*. It only infects banana and relatives.

The fungus grows in the soil and invades bananas through the fine (hair) roots. Spores are produced inside the water-conducting channels of the root and stem (xylem), and these and the growth of the fungus block the flow of water and cause a wilt. Fusarium wilt can remain alive in soil for long periods – perhaps indefinitely – as resistant spores, in infested plant debris or in the roots of other plants that are hosts.

Spread of fusarium wilt over short distances is by root-to-root contact, in surface run-off water, in soil attached to vehicles, tools, footwear and in unsterilized potting composts. Over longer distances, both within and between countries, spread occurs in infected planting material.

There are four distinct races of this fungus and one is divided into two strains. Tropical Race 4 (TR4) is the most serious as it affects a large number of varieties, including the popular Cavendish. All four of the races invade roots by travelling through the water-conducting tissues (xylem).

- Race 1 infects Gros Michel, but not Cavendish, and some plantains
- Race 2 generally infects cooking bananas
- Race 3 infects only Heliconia species
- Race 4 infects most varieties including Cavendish. There are two important strains: i) Subtropical Race 4 produces symptoms in Cavendish after a period of cold stress, and ii) Tropical Race 4 attacks Cavendish in tropical and sub-tropical conditions.

IMPACT

Race 1 forced the banana export industry to change from Gros Michel to Cavendish in the 1950s when fusarium wilt decimated production. Other races have appeared since then. It is estimated that 80% of global production is now under threat from TR4, which, if it spreads to Latin America, the Caribbean and West Africa, will have dire social and economic consequences. Not only will it devastate production, but also the livelihoods and food security of millions of smallholders who grow more than 85% of the crop. Additionally, there are potential environmental and biodiversity impacts as uncontaminated land would be cleared for cultivation, and difficult-to-grow varieties abandoned.

DISTRIBUTION

Fusarium wilt probably originated in Southeast Asia, but was first reported from Australia in 1876. Subsequently, it spread globally and is present in most parts of Asia, Africa and the Americas. It is now spreading in Pacific islands. The aggressive race TR4, which was first detected in Asia in the 1990s, it is now found in Taiwan, Indonesia, Malaysia, the Philippines, China and northern Australia. Outbreaks were recently reported from Mozambique (2013) and Jordan (2014).

FURTHER READING

Diekmann M, Putter CAJ (1996) FAO/IPGRI Technical Guidelines for the Safe Movement of Germplasm. No. 15. Musa. 2nd edition. Food and Agriculture Organization of the United Nations, Rome/International Plant Genetic Resources Institute, Rome. (ftp://192.156.137.116/ipgri/Publications/pdf/502.pdf).
Fusarium oxysporum f. sp. cubense. CABI Crop Protection Compendium. (http://www.plantwise.org/knowledgebank/datasheet.aspx?dsid=24621).
Fusarium wilt of bananas (Panama disease).Agnote. (http://www.nt.gov.au/d/Content/File/p/Plant_Pest/786.pdf).
García-Bastidas F,Ordóñez N, Konkol J, Al-Qasim M, Naser Z, Abdelwali M, Salem N, Waalwijk C, Ploetz RC, Kema JGH (2014) First Report of Fusarium oxysporum f. sp. cubense Tropical Race 4 associated with Panama disease of banana outside Southeast Asia. Plant Disease 98 (5): 694. (http://apsjournals.apsnet.org/doi/abs/10.1094/PDIS-09-13-0954-PDN).
New banana disease found in Mozambique (Fusarium oxysporum f.sp. cubense Tropical Race 4). (http://bit.ly/1IRS7EB).
Panama disease. Fact sheet. Plant Health Australia. (http://bit.ly/1HTTzHy).
ProMusa. Fusarium wilt of banana. (http://www.promusa.org/Fusarium+wilt).
WBF Fighting against banana threats. World Banana Forum.Food and Agriculture Organization of the United Nations. (http://bit.ly/1JtSBwy).

Bunchy top of banana

Banana bunchy top virus

Photo: Eric Boa, CABI, CC BY 4.0

Erect leaves, narrower and smaller than healthy leaves, with yellow edges.

Photo: Eric Boa, CABI, CC BY 4.0

Adult banana weevil.

SUMMARY: Bunchy top of banana is a viral disease that gets inside the plant and stays there. Infected planting material appears healthy. An aphid that is present in all banana growing countries helps to spread the disease over short distances. The bunches of erect leaves caused by the disease are more than a curiosity: the plant does not fruit and production is severely affected. All bananas are susceptible and there is no chemical treatment. However, careful selection of healthy planting material can prevent the introduction of the disease to new countries and early detection of symptoms enables its spread to be limited.

KEY SIGNS

The development or appearance of the symptoms depends on how the plant becomes infected. The first leaves (suckers) from an infected stool or mother plant (primary infection) are small and crowded together. They stay erect and do not arch outwards as in healthy banana plants. The leaf edges are a lighter colour (chlorotic), going brown towards the centre as they decline. New leaves are successively narrower and smaller, giving a distinct and typical bunchy appearance. Suckers from an already infected stool rarely produce fruit bunches.

Secondary infections occur when the aphid introduces the virus to a healthy plant. Symptom development is at first less dramatic and more difficult to see when compared to infected suckers. The second leaf produced after virus inoculation develops dark green streaks on minor veins that look like dashes, though these may be difficult to distinguish. The veins are hooked (J-shaped) as they join the middle of the leaf (midrib). The 'J' hooks and dashes are best seen on the underside of a leaf held against the light. Dashes may also occur on midribs and the main trunk of the banana.

The initial symptoms of a secondary infection can be mistaken for a nutrient deficiency or abiotic (physical not biological) stress. Bunchy top becomes more evident with the production of subsequent leaves that are successively smaller. Bunchiness is less pronounced, though still visually detectable, if secondary infection occurs at a later stage of banana development. A banana plant with a secondary infection may produce one set of fruit but these will be small and distorted. New suckers will contain the virus and produce leaves that are affected from the outset.

MANAGEMENT

Prevention – what to do before signs are seen

Cultural approaches: : The virus moves throughout the plant and may occur in parts which do not show symptoms, hence the importance of ensuring that material used for propagation, such as corms and suckers, is virus-free. It is crucial to screen material used in tissue culture so that plantlets are virus-free.

Where the disease is absent (e.g. Uganda) but present in neighbouring countries (e.g. DR Congo), it is vital that plant health

inspectors and extension officers recognize the symptoms of bunchy top so that farmers can be advised on how to select healthy planting material. Healthy planting material should be made available to farmers.

There is no biocontrol option for the banana aphid.

There is some evidence from central African countries to suggest that plantains (AAB or BBB group) are tolerant to bunchy top. For all practical purposes, however, all bananas should be considered at risk from infection.

Control – what to do after signs are seen

Cultural approaches: Constant vigilance (for example through plant clinics) and regular surveys by extension staff are essential to ensure that any suspected outbreaks are detected early and swiftly dealt with. In such cases affected banana plants should be removed and cut into small pieces to prevent new suckers growing.

Once the disease is well-established, the number and proximity of smallholders growing bananas and the difficulty in coordinating crop destruction in a timely manner makes it unlikely that local eradication campaigns will succeed without strong official enforcement. Such a campaign would also involve cutting down healthy banana plants.

Chemical approaches: There is no chemical control for the virus, either as a preventative or curative measure. Chemicals can, however, be used against the aphids which spread the disease: infected plants can be sprayed, particularly the underside of leaves, forcefully with water or a water-soap solution (about 2% by volume) to kill the aphids and thus help to contain an early outbreak. Insecticidal oils can also be used, such as those containing paraffin or neem extract (2% by volume).

CAUSE

Banana bunchy top virus is a type (species) of nanovirus. The disease is initially spread in planting material. Once established, the virus is spread locally by an aphid, *Pentalonia nigronervosa*, a sucking insect which is widely distributed and already present in countries still free from the disease.

Isolates of virus taken from across a wide geographic area of five central African countries, including DR Congo and Malawi, are genetically identical. The widespread distribution of the same strain of virus shows how local movement of banana planting material, from farmer-to-farmer, as well as aphid transmission, has helped to spread the disease. The virus is not mechanically transmitted so cleaning of tools will not affect spread to new plants.

IMPACT

Bunchy top is one of the most serious diseases of banana. In severely infected plants total yield loss can occur. An outbreak in Malawi in the mid-1990s affected 3500 hectares and is estimated to have destroyed 800 hectares. Outbreaks cause great concern because of the difficulty in managing the disease once established, followed by rapid spread through exchange of infected planting material. The presence of the aphid vector in countries currently free of the disease, such as Uganda, heightens the risk of banana bunchy top virus to a vital food crop.

DISTRIBUTION

Bunchy top of banana is found in West Africa (Benin, Nigeria and Cameroon) and in East and Central Africa (Burundi, Rwanda, DR Congo, Malawi and Zambia). It also occurs in Mozambique and several other African countries. The disease has not been recorded from Uganda or Tanzania.

FURTHER READING

www.rtb.cgiar.org/rtb-centers-and-partners-prepare-to-battle-banana-bunchy-top-disease-across-sub-saharan-africa/

Plantwise Knowledge Bank (www.plantwise.org).

CABI Crop Protection Compendium (www.cpc.org/cpc).

Promusa also has information on the disease (www.promusa.org).

For a recent review of bunchy top virus and overview of the disease:

Lava Kumar P, Hannab R, Alabic OJ, Sokod MM, Obena TT, Vangue GHP, Naiduc RA (2011). Banana bunchy top virus in sub-Saharan Africa: Investigations on virus distribution and diversity. Virus Research 159, 171-182

Banana xanthomonas wilt

Xanthomonas campestris pv. *musacearum*

Photo: Eric Boa, CABI, CC BY 4.0

BXW produces a distinctive premature ripening of fruit, which eventually rots.

Photo: Eric Boa, CABI, CC BY 4.0

BXW can be confused with Fusarium wilt, caused by a fungus. The bacterial wilt affects younger leaves, though, and leaves collapse inwards.

SUMMARY: Banana xanthomonas wilt (BXW) is named after the bacterium that infects the plant and eventually kills it. The disease has surged through Uganda since it was first found in 2001 and is now widespread in the region. All banana types are susceptible, though research has produced some promising results for future resistant varieties. The main management option for now is sanitation: planting healthy material, using clean cutting tools and removal of male buds to limit infection by bacteria-carrying insects as they collect nectar. BXW does not occur in West Africa and the other major bacterial wilt on banana worldwide, caused by *Ralstonia solanacearum* (Moko disease), is absent from all of Africa.

KEY SIGNS

The most distinctive feature of BXW is the premature ripening of fruit: individual bananas start to go yellow while others in the bunch remain green. When cut open, the bananas have dark stains, including those that are green. The bananas quickly blacken and eventually the whole bunch decays.

The first signs of infection, however, are when the purple leaves (bracts) of the male flower bud shrivel, go black and die. Also look out for young leaves in the crown that go yellow, fold in the centre and then collapse. Cut the fruit stalk and, once the milky juices stop oozing, look for small yellow dots. These indicate that the bacteria are blocking the flow of water and nutrients. It may, however, be difficult to see these yellow dots.

A fungus disease of banana, known as Fusarium wilt or Panama disease, also causes yellowing but on older leaves. The leaf blade does not become limp and the yellowing is much brighter compared to BXW. The fruit develop normally. The most distinctive symptom of Fusarium wilt is a dark staining inside the trunk, which is absent in bananas with bacterial wilt.

MANAGEMENT

Prevention – what to do before signs are seen

Cultural approaches: BXW is spread in infected planting material and by insects which introduce bacteria through banana flowers. The bacteria can also be transferred on cutting tools. All types of banana are susceptible though it is more difficult for the bacteria to infect some cultivars in the field because of their flowering characteristics. These cultivars can still be infected through contaminated cutting tools.

The key to successful management is careful selection of healthy planting material and keeping cutting tools used for cutting banana fruit bunches and leaves clean and free from bacteria.

Carefully select planting material from areas where the disease is absent. Obtain suckers from trusted sources. Do not use suckers from banana plants affected by BXW, even if the suckers appear healthy.

Removal of the male bud by hand or with a forked stick (to reduce the risk of moving bacteria on cutting tools) will limit the risk of infection via pollen and nectar feeding insects that have previously fed on infected plants. Timing of removal is important; male buds need to be cut off quickly after the fruit has been set. The drawbacks are that the method is time-consuming and some farmers believe the male bud is essential for good juice production in beer types of banana.

Chemical approaches: Cutting tools can be cleaned with household bleach (one part to four parts of water), solutions containing local plants with antibiotic activity, such as tobacco or chilli, or by passing through a flame. Bleach is the most effective method for killing bacteria though few farmers appear to use this method.

Control – what to do after signs are seen

Cultural approaches: Initial advice when BXW was first discovered in Uganda was to dig up and burn the whole banana mat. We now know that bacteria do not completely invade the plant. The current advice is therefore to remove those trunks (pseudostems) showing symptoms of BXW and dispose of these carefully. Single stem removal will reduce the amount of bacteria capable of infecting new plants, but this will not eliminate the disease. Selection of healthy planting material and cleaning of tools remain the key pieces of advice to follow.

Chemical approaches: Once the plant is infected there is no treatment to control the disease.

CAUSE

This is a bacterial disease caused by *Xanthomonas campestris* pv. *musacearum*. The 'pv.' stands for pathovar and indicates a particular type of *X. campestris* that only infects banana and its close relative ensete. Previously it was known as *X. musacearum*.

The bacterium can be spread by insects feeding on flowers of infected plants and then feeding on clean plants, and also via cutting tools contaminated with bacteria.

Globally, there are several bacterial wilts that cause similar symptoms and behave in similar ways to BXW. These wilts are associated with different types of the bacteria *Ralstonia solanacearum*, which does not attack banana in Africa.

IMPACT

BXW is a highly damaging disease affecting all types and varieties of bananas. Plants and fruits are destroyed. The disease can be spread over long distances, presumably in planting material and possibly plant debris: the strong demand for bananas in Kampala attracts imports from far away. Most information about impact comes from Uganda, where the disease has caused the biggest losses and had a major impact in stimulating research and extension efforts to contain and manage outbreaks.

DISTRIBUTION

Wilt symptoms were seen on ensete in Ethiopia, probably as early as the 1930s, but not confirmed as *X. campestris* pv. *musacearum* until the 1960s. Ensete is closely related to banana and is cultivated for food in Ethiopia. It grows wild in other countries in East and Central Africa but there are no recorded wilt symptoms on ensete outside Ethiopia.

BXW appeared on bananas in Uganda in 2001 and in a remote area of North Kivu, DR Congo around the same time. Neither introduction has been fully explained but, since these first recorded outbreaks, the disease has spread to all banana growing areas of East Africa.

In order of confirmation, from 2001 onward, BXW has been found in: Uganda, DR Congo (North and South Kivu), Rwanda, Tanzania, Kenya and Burundi.

FURTHER READING

CABI Crop Protection Compendium (www.cpc.org/cpc).

Promusa (www.promusa.org).

Plantwise Knowledge Bank (www.plantwise.org).

There are several useful reviews of BXW with the most recent being:

Blomme G, Jacobsen K, Ocimati W, Ntamwira J, Sivirihauma C, Ssekiwoko F, Beed F, Nakato V, Kubiriba J, Tripathi L, Tinzaara W, Mbolela F, Lutete L, Karamura E (2014) Fine-tuning banana Xanthomonas wilt control options over the past decade in East and Central Africa. European Journal of Plant Pathology (in press)

An abstract of Blomme et al. (2014) is available at www.banana.go.ug, which as updates on banana research in Uganda.

Check the Roots, Tubers and Banana project website for current activities on BXW (www.rtb.cgiar.org).

This manual is an output of the Africa Soil Health Consortium (ASHC), which is coordinated by CABI.

This guide was first published in 2015 by ASHC
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