Wild Weather Versus Good Disease Management

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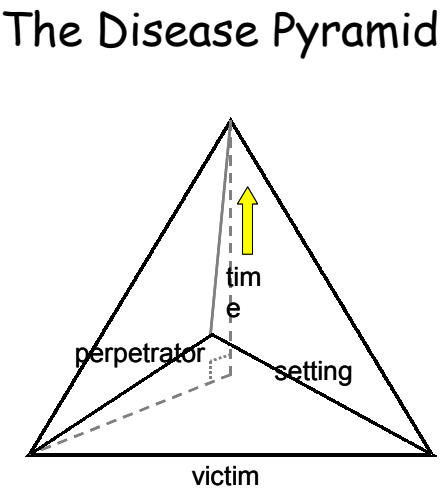
Introduction

The Planning Committee for the Soils and Crops Workshop was united in its desire to include a special topic considering **field crop diseases** – a broad ranging discussion to shed some light on the abundance and intensity of disease seen in crops grown in Saskatchewan in 1999. Although there was more disease in a wide range of crops, we have decided to limit our focus to **pulse diseases**. Many of the talks that follow in this session will also cover pulse diseases, and in greater detail. Our intention is to set the stage – to give you, in layperson's terms, a perspective on the strategies farmers employ to combat disease and their prospects for success.

A Disease Triangle



The first concept that we present is that of the **disease triangle**, the three pre-conditions for disease to develop in the first place. Let's consider disease as a "crime" against crops. You cannot have disease without a suitable *victim*, an aggressive *perpetrator*, and the ideal *setting*. Take any of these away and the crime is far less likely to occur. A few diseases, such as the fusarium head blight and ergot, occur suddenly and might be considered violent crime. The majority, however, are more like the white-collar crime of fraud, that is they occur over a longer period of time. The longer the fraud continues undetected or unchecked, the more serious its consequences. By adding *time* as a fourth dimension, the triangle becomes a pyramid.

Before we spend too long in the world of analogy, let's consider a specific disease to delve into the mysteries of the disease pyramid: ascochyta blight in chickpea. The **perpetrator** is a highly aggressive fungus by the name of *Ascochyta rabei*. Not all fungi are bad; in fact many are useful in breaking down plant material into organic matter. *A. rabei* though is one malicious character. All of the recommended varieties of chickpea are rated as very good for their resistance to ascochyta blight. In normal circumstances, Sanford (kabuli) and Myles (desi) should not fall **victim** to this perpetrator. In '99 however, the cards were stacked against chickpea. Frequent precipitation delayed seeding and cool soils delayed emergence. Even the earlier maturing desi type had a long season ahead of it. Then, rainfall continued in earnest through June and July,

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giving the perpetrator every opportunity to build up and overwhelm the crop – an ideal **setting** and plenty of **time**.

Fighting Back: Management Tools

"Aha!" you say. We even used a weapon (fungicide) against this character and still we suffered losses. It turns out there was more than one criminal stalking chickpeas (the others were *Sclerotinia* and *Botrytis*). Before we get to those, let us examine all of the tools we can use to **prevent** disease. Plant pathologists, like medical pathologists, only participate in **intervention** by providing accurate diagnoses – identifying the offending perpetrator. Otherwise, we preach **prevention** because with crops, as with people, "the best offense [against disease] is a good defense." Many of the following tools are employed to prevent infection from happening in the first place.

Here is a list of defensive weapons. On the right we list which side of the disease triangle each affects:

- Genetic resistance – beefing up the immune system *host*
- Clean seed – keeping the culprit off your farm *pathogen*
- Crop rotation – allowing natural selection to wear down the opposition *pathogen*
- Use of fungicides – attacking the weak link in the enemy's armor *pathogen*
- Agronomic practices – safeguarding the neighborhood *environment*
- Location – staying out of harm's way *environment*
- Time of planting – avoiding a confrontation *time*

As you might suspect, not all of these tools is equally effective against each disease. We are going to explore them as **key** *management options* relating to specific pulse diseases, in which instances they may let us down, and which others will bolster our defenses.

1. Genetic Disease Resistance

To keep with our crime analogy, we can consider genetic disease resistance as **changing the locks** so that the pathogen is unable to enter the host plant and cause an infection. Genetic resistance entails modifying the genetic structure of the host plant so that it recognizes the pathogen and can isolate it. Breeding for disease resistance can be compounded by the fact that there are usually many **races or strains** within a pathogen population. Thus, the best defense against disease is to **pyramid genes** from multiple sources of resistance. Breeding for resistance is a continuous process because it puts selective pressure on the pathogen population and favours the development and survival of new strains that can overcome the plant's resistance. An example of this would be breeding for leaf and stem rust resistance in cereals. Even with good sources of resistance, every once in a while we experience a rust outbreak as new strains attack our cereal varieties and breeders then need to find new sources of resistance. However, since this presentation is limited to pulse diseases, we will avoid discussion of the 'cereal' killers.
A main goal for the pulse breeders has been to improve genetic resistance to ascochyta blight in all pulses, as well as to powdery mildew in pea and anthracnose in lentil. Advances have been

made in the development of new lentil lines with **resistance to anthracnose** (*Colletotrichum truncatum)*. An anthracnose resistant variety, CDC Robin, is now registered. A limitation with this new variety is that resistance was selected for using anthracnose strains from one location, so this resistance may not bear up elsewhere. The Crop Development Centre in Saskatoon is actively developing markers for anthracnose and ascochyta resistance in lentil and ascochyta resistance in chickpea.

2. Clean Seed

The rationale for testing pulse seeds is not entirely based on disease risk assessment. Since seed costs are proportionally higher than for many field crops, a determination of seed viability is useful in determining the appropriate seeding rate to achieve an optimal plant stand. With field peas, for example, the range of seed size expressed as thousand kernel weight (TKW) is very large. Following is a formula to help determine the appropriate seeding rate (SR) as a function of seed size:

Calculation of Seeding Rate:

$$SR = \frac{10 \times TKW \times \text{plants per square foot}}{\%\ \text{projected emergence}}$$

$$202\ \text{lbs./ac.}\ (\sim 3.4\ \text{bu}) = \frac{10 \times 260 \times 7}{90}$$

Many of the diseases of pulse crops are seed-borne so it is recommended that producers use seed with acceptable disease levels in addition to good germination. One way to look at the importance of having seed tested is to think of it as **testing for fingerprints**. The seed may look healthy but there may be evidence of the pathogen within the seed.

The risk of planting diseased seed depends on a number of factors including:

- Disease pathogen present
- Type of pulse seed
- The cost and availability of seed
- Use and availability of seed treatments
- Location and weather conditions
- Scouting and fungicide application in the field

For an example, let's look at what a **2% ascochyta seed infection** means for the various pulse crops. In field pea, a 2% infection is safe to plant as the seed-to-seedling transmission of the disease is not that high. Furthermore, inoculum on infected pea residue from surrounding fields is usually a more important source than what is brought in on the seed. In lentil, a 2% infection is acceptable to plant in the brown soil climatic zone, but in areas with higher moisture such as the dark brown or moist dark brown soil zones, the farmer may consider a fungicide seed treatment (Crown) for the control of seed-borne ascochyta. In chickpea, the level of risk is even greater, regardless of soil climatic zone, as the seed-to-seedling transmission of the disease is high.

An ascochyta seed infection of 0% is recommended for chickpea. Even a 0.25% ascochyta seed infection in chickpea could potentially result in 400 infected plants per acre, or 1 infected plant every 10 sq.m. Therefore, even a low seed infection could result in an infected seedling in the field that serves as an inoculum source for infecting neighbouring plants. In growing seasons with frequent rainfall, the aggressive *Ascochyta rabei* pathogen can result in severe ascochyta blight development under a lush chickpea canopy. Another reason for the zero tolerance of ascochyta in chickpea seed is that there are currently no seed treatments registered for the control of the seed-borne phase of this disease.

Even if farmers are using clean seed it is important that they learn how to scout early in the season for symptom development when weather conditions are favourable and be prepared to apply a foliar fungicide application of Bravo®.

3. Crop Rotation

Crop rotation is the process of allowing time for the natural decomposition of fungal structures surviving on crop residue or in the soil. In regard to our crime analogy, it can be referred to as a **clean-up-the-neighbourhood campaign.** Unfortunately, crop rotation is not a silver bullet as the pathogen can spread or be brought into a field by other means. In general, a crop rotation of 3 to 4 years between the same types of crop is recommended.

However, this is not practical in the case of ***Sclerotinia* stem rot** caused by the pathogen *Sclerotinia sclerotiorum*. Many broadleaf plants besides pulses, including canola and even weeds, are susceptible to this disease, making it impractical to include a broadleaf crop only once every 4 years in rotation. Therefore, diseases such as *Sclerotinia* and also *Botrytis* (grey mould) can be considered as **opportunistic pickpockets** as they will be prevalent in most areas in years where the weather conditions favour their sporulation and spread, regardless of crop rotation.

Also, some pathogens have an **air-borne spore stage** that can move into a crop from adjacent infected fields. Examples of such diseases include ascochyta blight of pea, anthracnose of lentil and *Sclerotinia* of broadleaf crops. It becomes nearly impossible for the farmer to plan his rotations so that the crop is not planted next to infected residue of the same crop. Furthermore, determining crop rotation is a decision that is not based solely on disease management but also on weed control, soil fertility, residue management and economics.

4. Scouting for Disease and Fungicides

Farmers need to have training by the **FBI (Fungal Blight Investigators)** as to what they should be watching for in their crops. They need to anticipate disease by learning what conditions favour them and also how to look for "incriminating evidence" or symptoms of the disease. An effective **scouting routine** will provide farmers information on what diseases are present and potential crop loss if left untreated. Identifying disease potential in advance will allow the farmer time to make a decision and to ensure the fungicide is applied at the right time.
For diseases such as **ascochyta blight** in chickpea, lentil and pea and for anthracnose of lentil, symptoms of the disease and the environmental conditions conducive to disease development

should be present before an application of Bravo 500® is made. There is a Fungicide Decision Support System available for anthracnose and ascochyta blight in lentil and for ascochyta blight in field pea to aid farmers in making this decision:

http://paridss.usask.ca/specialcrop/pulse_diseases/index.html

Conversely, with a disease such as ***Sclerotinia* stem rot** which requires an external nutrient source (= petals) to start an infection, a fungicide application has to be made to protect against this primary infection. Therefore, the most effective timing hinges on predictive methods for forecasting whether the conditions are there to favour disease development. Once the farmer observes *Sclerotinia* stem rot symptoms, it is too late to apply fungicides. Currently, foliar fungicides for *Sclerotinia* stem rot control are only registered in bean and canola.

An understanding of **how fungicides work** is also important. For example, Bravo 500® (chlorothalonil) does not have systemic movement within the plant but has contact activity. Consequently, it is used in a preventative fashion as it will not stop infections already started but protects against the next infection cycle. Good coverage and penetration of the plant canopy is essential with contact fungicides.

Fungicides may also be employed earlier in the life of the crop as seed treatments. Refer to Management Tool #2.

5. Agronomic Practices (Fertility, Tillage and Crop Density)

While we may not be able to affect the weather, we **can** have an impact on the micro-climate within the crop. A lush, uniform, closed crop canopy may be appealing to the eye yet a real hazard to the onset and development of disease. Nutritional imbalance may contribute to the creation of a disease-conducive micro-climate. With peas, lentils and chickpeas, *Rhizobium* inoculation is responsible for providing the bulk of the crops' nitrogen (N) requirements. Thus an excess of N fertilizer is rarely a problem. The risk of phosphate (P) fertilizer in the seed row may limit farmers' ability to deliver adequate P to crops that are heavy users. An appropriate N:P balance is important in ensuring even and early maturity.

While many pulse diseases are residue borne, the destruction of crop residues through tillage is counterproductive to soil conservation goals. Continuous cropping and reduced tillage, however, are practices that go hand-in-hand with a greater emphasis on crop rotation, both for residue management and for disease management.

Beans are highly susceptible to white mold. The perpetrator is the same fungus that cause stem rot in canola and field peas, *Sclerotinia sclerotiorum*. Widening row spacing or careful plant population selection in solid seeded beans can delay canopy closure to a time when infection is less likely to occur. Plant architecture, *i.e.* crop height and branching pattern, may also be utilized to modify the crop canopy, allowing air circulation.

Some varieties of lentils and chickpeas that were subject to above average rainfall suffered from overly dense canopies that led to a high incidence of *Sclerotinia* and *Botrytis*. Since these crops

are poor competitors with weeds, a reduction in seeding rate is not a practical management tool. Weed infestations, however, can influence the intensity of disease. For example, volunteer cereals in lentil have been known to "prop up" lentils allowing air movement beneath the canopy and **reduce** the incidence of *Botrytis*. Dense patches of susceptible broadleaf weeds in any of the pulse crops can **increase** the incidence of *Sclerotinia*.

6. Location

In light of current commodity prices, it may be difficult to decide what to plant. Conversely, it may be tempting to pick a winner and bet the farm, *e.g.* chickpeas are a brighter spot on the commodity price horizon. Can chickpeas be grown in all of our soil climatic zones? The answer is NO. The chickpea varieties currently available are not suitable for planting in soil climatic zones that experience shorter growing seasons or are more likely to receive frequent rainfall. Chickpeas are best adapted for the brown soil zones or in the province's south-west. In some years, farmers may be able to get away with producing pulse crops outside the area of best adaptation BUT they are increasing their risk to yield loss as a result of disease and fall frost. Field pea is best adapted to the moister soil regions such as the black, moist black and grey soil climatic zones. Lentil is best adapted to the soil climatic zones in between chickpea and pea, such as the dark brown and moist dark brown zones. If the risk of disease is too high, it is advised to **move to a safer neighbourhood** and avoid the ideal setting for a disease crime.

7. Time of Seeding

To examine time of seeding as a cultural method of managing disease we focus on powdery mildew on field peas. You may have seen less of it in 1999. Powdery mildew, unlike some of the diseases we've mentioned does not thrive in cool, wet weather. Infection is favored by weather that causes dew formation, namely hot, dry days and cool nights. Such conditions normally occur in late July or early August. Planting as early as soil temperatures allow avoids excessive heat during flowering. It also minimizes the impact of powdery mildew in susceptible crops by allowing the crop to mature prior to the onset of disease-conducive conditions.

Summary Comments

Looking at the title of our presentation you may have expected that we would just throw up our hands and say, "Let's blame it on the weather." Our goal, however, was to convey that weather (environment) is just one element of a three part phenomenon. We have to examine whether we have done all we can to avoid being victims of circumstance. We **do** have a major role to play in that regard. We cannot depend solely on one strategy (*e.g.* resistance) and knowing as much about the perpetrators as we can helps us to plan our defenses most effectively.