Sulfonylurea spray contamination damage to canola crops

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Abstract

Every year large areas of canola are damaged by non target crop herbicides resulting from boomspray contamination. Sulfonylurea herbicides such as triasulfuron, metsulfuron-methyl and chlorosulfuron are used in farming systems to control a range of grass and broadleaf weeds in cereals. While these herbicides are useful in the farming system they can cause considerable damage to non target crop species due to the difficulty in decontaminating all boomspray components and the potential for crop damage at low concentrations.

Research has shown that minute amounts of sulfonylurea herbicides can result in significant damage to sensitive crops such as canola. Piper and Bell (1999) showed that 20 milligrams per hectare of triasulfuron (for example Logran®) applied to canola seven weeks post sowing reduced yield by 45% when compared to the untreated control.

Piper and Bell (1999) determined that low concentrations of sulfonylurea applied post emergent to seedling canola results in a range of plant symptoms. They concluded that canola can probably outgrow symptoms of sulfonylurea-induced leaf yellowing but once sulfonylurea-induced leaf distortion occurred severe yield losses were likely.

Sulfonylurea damage in canola is easily confused with a range of other diseases and nutrient disorders because plant symptoms for both are similar. Diagnosis of sulfonylurea damage to canola usually occurs as a process of elimination. There are some paddock signs that distinguish sulfonylurea damage from other diseases.

Sometimes the symptoms of damage appear clearly where the boomspray has started and they fade further into the paddock. Other cases of crop damage can occur after several susceptible crops have been sprayed with no apparent damage. The delayed effects of crop damage usually occur when a solvent based insecticide or herbicide strips a deposit of sulfonylurea residue from a component where it has accumulated, such as a hose end or a filter screen.

Sulfonylurea herbicides

Sulfonylurea herbicides, or amino acid synthesis inhibitors as they are also known, inhibit cell division in the roots and shoots of plants (Powley 2003). Sulfonylurea herbicides include:

- chlorosulfuron (for example Glean®)
- sulfo sulfururon (Monza®)
- metsulfuron-methyl (Ally®)
- triasulfuron (Logran®)
- thifensulfuron-methyl (Harmony®)

Sulfonylurea herbicides act by binding to a specific enzyme to prevent the production of amino acids that are essential for normal plant growth and development. Sulfonylureas are rapidly taken up and translocated but are not persistent in the plant (Tomlin 2000). They are widely used in cereals as they control a broad range of weeds and are relatively inexpensive to use.

Sulfonylureas are characterised by their efficacy at low application rates and their persistence in soil (Powley 2003). Sulfonylureas are particularly effective in controlling broad leaf weeds, which means they are also damaging to broad leaf crops, such as canola. Because they are efficacious at low rates of active ingredient, any residues left in a boom spray from controlling weeds in cereals can be very harmful if sprayed out on other crops, particularly canola. Western Australian research has shown that less than one thousandth of the concentration used to control broad leaf weeds in winter cereals will reduce yield of canola by up to 45% (Piper and Bell 1999).

Symptoms of sulfonylurea damage

Symptoms of post emergence sulfonylurea herbicide damage to canola usually take 10 to 14 days to develop and include:

- stunting of plants
- clustering and distortion of new growth
- cupping of the existing leaves
- yellowing of the youngest leaves starting at the leaf margins
- production of thin strap-like leaves
- reddening or purpling of the leaf veins
Reddening and purpling of leaves may also occur because plants produce anthocyanin, a red or purple pigment, in response to stress.

Symptoms of sulfonylurea damaged canola plants are consistent with a range of plant disorders. In canola, viral diseases such as beet western yellows virus can cause stunting and clustering of new leaves, direct insect feeding can stunt new leaf growth, manganese toxicity typically causes yellowing of the outer leaf margins, boron deficiency can cause convex cupping of leaves and moisture stress can cause leaf discoloration stunting.

Sulfonylurea damage can occur over the whole crop, when residue is dislodged directly into the tank, or in strips within a crop where residues are confined to individual nozzles or hoses. In paddocks where the majority of the crop appears damaged, strips of plants without symptoms may be seen. The undamaged plants usually occur where the boomspray has driven wide of the previous run. Untreated areas are useful when diagnosing damage and can often be found around trees or fence-lines.

Decontamination, residues and appearance of damage symptoms

Sulfonylurea herbicide labels contain extensive information about decontaminating application equipment after using sulfonylurea herbicides. To decontaminate, chloride and water are flushed through equipment to neutralise the SU herbicide remaining in the equipment. If decontamination does not involve the physical removal of accumulated residues in certain components and from surfaces of equipment then flushing with chloride and water may not adequately remove residues. This becomes of particular concern where sulfonylurea residues are allowed to dry and crust.

Some boomspray components, such as hollow end caps on boom lines and hoses to pressure compensators valves, are readily identified as residue accumulation points. Replacing such components with easy-to-clean parts or exchanging them, after sulfonylurea use, with an extra set of components may help to minimise the risk of crop damage. Some contractors and farmers have gone to the extent of purchasing two separate boomsprays, one for sulfonylurea herbicides and one for other herbicides, in order to avoid the contamination problem (Sutherland pers. comm.; Parker pers. comm.).

The irregular occurrence of non-target crop damage is due to the unpredictable timing of dislodgement of residues. Many cited cases of sulfonylurea damage to non target crops have occurred after spraying hundreds of hectares of susceptible crop with the same chemicals without the appearance of any damage symptoms at all (Sutherland pers. comm.; Stoll pers. comm.).

The variable solubility of sulfonylurea herbicides in different carriers, the complexity of design of some spraying equipment and the ability of the herbicide to accumulate in minor components appears to be the cause of such irregularity.

While there is often no apparent reason for the occurrence of sulfonylurea damage to non target crops it regularly occurs after insecticides or herbicides with an emulsifiable concentrate formulation are used. Sulfonylurea herbicides are more soluble in some organic solvents, which act as the carriers in emulsifiable concentrate formulations, than in water. Further, sulfonylurea herbicides vary in their solubility in water depending on pH (Tomlin 2000).

A current recommendation to minimise the risk of sulfonylurea herbicide damage to sulfonylurea susceptible crops, based on this information, is to physically remove sulfonylurea residues from components identified as potential accumulation points, decontaminate equipment using chlorine and to spray an emulsifiable concentrate formulated product in a non-sulfonylurea susceptible crop prior to spraying an sulfonylurea susceptible crop.

Analysis of plant tissue for sulfonylurea damage

Plant tissue can be analysed to confirm exposure of sensitive plants to sulfonylurea herbicides but due to the level of sophistication of the equipment and the technical skill required analysis is expensive (approximately $200 to $300 per herbicide). There are several factors that will influence the validity of plant tissue testing to confirm sulfonylurea exposure. These include the:

- rate of metabolism of the sulfonylurea in the plant
- timing after exposure of collection of the sample
- specificity of metabolites to sulfonylurea exposure
- sensitivity of the test
- history of sulfonylurea use in the paddock

Analysing plant tissue for the presence of the suspected parent compound may not confirm exposure to some sulfonylurea herbicides as they can be rapidly metabolised in the plant. For example, chlorsulfuron is metabolised very little by sensitive plants while triasulfuron is metabolised before symptoms appear (Davy et al. 2001). Within hours of exposure, some plants can significantly reduce the amount of triasulfuron to metabolic by-products (Davy et al. 2001). If sampling does not occur until after symptoms develop then levels of sulfonylurea
may be undetectable. Testing plant tissue for metabolic by-products may provide evidence of exposure to sulfonylurea herbicides but not all are specific to sulfonylurea herbicides. Some metabolic by-products produced after exposure to sulfonylurea herbicide can also be produced in response to natural environmental stress (Davy et al. 2001).

A sensitivity of less than one part per billion may not adequately detect sulfonylurea residues in plant tissue if sampling has not occurred until after plant symptoms are observed due to the rate of metabolism.

If the paddock has a history of sulfonylurea use then it may be necessary to test several samples to distinguish between sulfonylurea presence as a result of soil residue and sulfonylurea presence as a result of foliar application.

Sampling the spray mix for the presence of sulfonylurea traces is possible. It is however, unlikely to confirm a residue if it has accumulated in a spray component without dislodging back into the spray mix.

**Diagnosis**

In order to determine the cause of any crop disorder it is necessary to obtain as much information about the crop, paddock history and growing conditions. Diagnosis of herbicide damage to sensitive crops occurs as a process of elimination. First, all of the possibilities for the cause of crop damage are considered, then, after information is gathered, the least likely causes of damage are discounted and the most likely causes are investigated.

Plant parts such as roots, leaves, growing points, buds, flowers and seeds of plants with damage symptoms should be compared to plants without symptoms. Photos and records of the symptoms can be useful for further reference. Inspection for insects on plants with a hand lens and in the field may assist with diagnosis (Gage and Munro 1979).

In the field, patterns of damage relating to aspect, topography, machinery operations, insect infestation or wind direction may help to discriminate between sulfonylurea herbicide damage and another cause. Observation of weeds in unsown areas and plants on headlands, fencelines and around trees may also provide answers. Inspections of machinery for abnormalities and maintenance history may reveal useful information. The timing of the appearance of the symptoms may relate to the timing a routine management practice (Gage and Munro 1979).

**Conclusion**

Sulfonylurea spray contamination can cause significant yield loss in canola and the cost to canola growers, when it occurs, is high. The risk of sprayer contamination cannot be eliminated completely where there is a reliance on sulfonylurea herbicides in the farming system. Cost effective strategies such as exchanging components identified as potential residue accumulation points and spraying non-sulfonylurea susceptible crops after sulfonylurea decontamination with emulsifiable concentrate formulated chemicals to take advantage of the known solubility of sulfonylureas can however, be employed to reduce the risk of contamination.

**Case Study**

Following is a case study of a paddock in southern New South Wales. Canola plants in certain parts of the paddock developed symptoms of plant damage eight weeks after sowing.

The paddock is situated in southern New South Wales and had not been cropped for 20 years prior to 2003. In 2002 the paddock was a poor pasture paddock consisting mainly of onion grass (*Romulea rosea*), subterranean clover (*Trifolium subterraneum*), silvergrass (*Vulpia spp*), annual ryegrass (*Lolium rigidum*) and pattersons curse (*Echium plantagineum*). There was no paddock preparation prior to February 2002 because a sharefarming agreement was not signed until then.

The paddock was cultivated once with a scarifier in February and sown on the 26th April 2003 at 3.6 kg/ha of Pinnacle canola and 121 kg/ha diammonium phosphate (18:20:0:2) fertiliser. Gypsum was broadcast over the paddock after scarifying. There were no pre sowing applications of herbicide. A tankmix of herbicide and insecticide (Atrazine 600 g/L a.i. at 800 mL/ha plus Simazine 600 g/L a.i. at 2.5 L/ha plus Alpha cypermethrin 100 g/L a.i. at 100 mL/ha plus dimethoate 400 g/L a.i. at 85 mL/ha) was applied on 2nd May 2003 with the farmer’s boomspray.

A further application of dimethoate (400 g/L a.i. at 85 mL/ha) was applied on 16th May 2003 by a contractor. The farmer applied the insecticide because he was sowing and knew that he would not have time to monitor the paddock for insect pests. The farmer used a contractor because he had sprayed sulfonylurea herbicides with his own boomspray and did not have time to decontaminate thoroughly. None of the surrounding paddocks had been sprayed prior to the symptoms appearing.

The contractor had applied 300 hectares of a sulfonylurea herbicide (triasulfuron) prior to the dimethoate application and he decontaminated according to the label directions after using the sulfonylurea. Glyphosate, simazine and atrazine were used after the sulfonylurea application. The symptoms observed (Figure 35 to Figure 39) in the
paddock were not apparent in 20 hectares of canola sprayed prior to this application.

The soil is a red brown earth with soil test values 0–10 centimetres as follows:

- pH 4.8 (CaCl2)
- 5% Aluminium
- 17 mg/kg Phosphorus (Colwell)
- Phosphorus buffer index 46
- 9.4 mg/kg Sulphur (KCl)
- 16 mg/kg Nitrate nitrogen
- 350 mg/kg Potassium
- 1.3% organic carbon

A paddock inspection was made by the farmer on 21st June 2003 when he observed patches of plants with poor vigour. The paddock was inspected and diagnosed by an agronomist on 23rd June 2003. The following photos were taken on the 4th July 2003.

The photos below show the typical pattern of damage in the canola paddock and the range of plant symptoms that occurred.

Table 69: Rainfall records for the property to the end of June

<table>
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<th>2003</th>
<th>Mean</th>
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<tr>
<td>Feb (mm)</td>
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</tr>
<tr>
<td>Mar (mm)</td>
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<td>Apr (mm)</td>
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<td>May (mm)</td>
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<td>Jun (mm)</td>
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<td>58</td>
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<td>Total to end June (mm)</td>
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<td>264</td>
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</tbody>
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Figure 35: strips of severely damaged plants in the middle foreground.
The strips occur in the same direction of travel of sowing and spraying machinery.

Figure 36: Strips of complete plant death.
The strips follow the outline of the crop and appear two metres from the crop edge. On the edge of the strips plants are exhibiting milder damage symptoms.

Figure 37: Death of the youngest plants.
Elongation of leaves can be seen on the older plants.

Figure 38: A canola plant exhibiting the milder symptoms of leaf cupping and yellowing of the outer leaf margins.
Figure 39: Close-up of leaf distortion symptoms. The four plants on the left are exhibiting symptoms of leaf distortion. The two plants on the right exhibit none of the same symptoms. Measurements are in centimetres.

Diagnosis

The symptoms exhibited by the damaged canola plants are consistent with a range of plant disorders including beet western yellows virus, direct insect feeding damage, manganese toxicity and herbicide damage.

Canola plants exhibiting severe symptoms appear in a discriminatory pattern typical of machinery related damage (Figure 35 and Figure 36). There are three distinctive areas of complete crop death in the paddock found approximately two metres in from the crop edge following the outline of the crop (Figure 36).

The pattern of crop damage, the insecticide application history and the areas of complete crop death suggest that disease, insect feeding or nutrient disorder are the least likely cause of the symptoms. The pattern of plant damage is consistent with the width of a spray nozzle and the direction of travel of the boomspray, the two early post emergence insecticide treatments are likely to have significantly reduced the aphid population thereby reducing the chance of direct feeding damage and beet western yellows virus (aphid are the vector of beet western yellows virus) and disease or nutrient disorder rarely cause complete crop death.

This indicates that herbicide damage is the most probable cause of the canola damage symptoms.

Group M herbicides (for example glyphosate) and group B herbicides (for example triasulfuron) can cause similar damage symptoms at low rates. The surrounding paddocks were not sprayed prior to the appearance of the symptoms suggesting that herbicide drift was unlikely. The spray history eliminates glyphosate as a contaminant and leaves sulfonylurea herbicide as the most likely cause of crop damage symptoms.

It is likely that an accumulated residue was dislodged in spray lines during the contractor’s application of dimethoate, an emulsifiable concentrate formulated insecticide.

References


Gage JF and Munro HE (1979) Herbicide effects in crop plants. Queensland Department of Primary Industries, Brisbane.


Personal communications

