Stressed crop plants **provoke herbicide injury**

By Dr Charlie Reinhardt (Photos by Dr Charlie Reinhardt)

Stress in plants can be defined as any external factor that negatively influences plant growth, productivity, reproductive capacity or survival (D Rhodes and A Nadolska-Orczyk, 2001).

Herbicide injury on crops represents an abiotic (non-biological) stress factor that is of synthetic chemical nature. Other abiotic stresses include natural factors such as wind, hail, wounding and physical impedance of roots in compacted soils.

The abiotic stresses that crop plants most commonly suffer are extremes in water supply (drought or flooding), temperature (low or high), irradiance (low or high sunlight), nutrition (deficiency or excess) and soil pH (acidic or alkaline). Biotic or biological stresses include plant pathogens (bacteria, fungi, viruses) and herbivores such as insects and animals.

This article focuses on the interactions between herbicides and other abiotic stress factors, specifically those that can cause a herbicide that is supposed to be safe for use in a crop (i.e. registered for use in that crop) to unexpectedly injure the crop.

Disruptive abilities

Herbicides have the inherent ability to disrupt the metabolism of all plants (crops and weeds) at a physiological level, which can lead to plant injuries manifesting in various forms as symptoms of injury or damage. Ultimately plants can die – in fact, the latter consequence of plant response to a herbicide is the fundamental expectation underpinning chemical (herbicide) weed control.

The ability to select between weeds and crops makes herbicides unique among pesticides (xenobiotics) – the onus on the pesticide not to directly harm the crop, or other desirable vegetation, is nowhere greater than in the case of herbicides.

Stress factors and crop yield loss

Metabolic impairments or injuries caused by herbicides range from inhibition of photosynthesis and protein synthesis, and damage to cell membranes as well as membranes of organelles (e.g. chloroplasts) in cells. Herbicide injury typically progresses in a sequential way as one after the other life process are impaired, and secondary effects of non-herbicidal nature can follow on the primary herbicide effects. Plants weakened by herbicide injury (primary stress) can be predisposed to pathogen attack (secondary stress), and *vice versa*.

In recent growing seasons, field observation of heat/drought and sandblast damage on sunflower and soya bean seedlings coincided with apparent reduced ability to tolerate or resist herbicides that are normally perfectly safe towards the crop.

Understandably, such doubly stressed crop plants are likely to be susceptible to a third stress factor such as pathogens, which gain access through physical lesions (wind and drought/heat damage), and due to a physiologically weakened (herbicide damage) crop plant whose natural defence mechanisms are impaired.

Ultimately it does not matter which factor had the primary effect and which the secondary effect, because crop growth and development are negatively affected by the interaction of stress factors, and serious yield loss is a real risk.

Herbicide and environmental factors

Another example of herbicide interaction with environmental factors that can provoke herbicide injury of the crop, is when the soil-applied herbicides metolachlor and acetochlor are used in maize under a specific set of soil/climate factors. These two herbicides can safely be used in maize for grass and nutsedge control only if a so-called 'safening' chemical is included in the herbicide formulation.

The 'safener' works by boosting an enzyme system in the maize plant, glutathione-s-transferase, which

A sunflower seedling showing symptoms most likely caused by a combination of heat and herbicide injury.

deactivates the herbicide and thus ensures crop safety. Grass and nutsedge weeds possess a less efficient enzyme system and, hence, are controlled.

Under wet, cold conditions, which are common early in the summer rainfall regions, the herbicide is taken up by plants (crop and weed) due to adequate soil moisture and relatively warm days, but during the colder nights the activity of the enzyme system doing herbicide inactivation is reduced and the result can be crop injury.

Moreover, a cool/cold soil slows the maize seedlings' emergence rate and hence allows for uptake of more herbicide than would be the case in warmer soil where coleoptile development would be boosted, and therefore, result in reduced exposure of the crop to the herbicide. This higher than usual uptake of metolachlor or acetochlor can be sufficient to overwhelm the enzyme system doing herbicide inactivation, with dire consequences for the crop.

The coleoptile part of grass seedlings, maize included, is where preferential uptake of herbicides such as metolachlor and acetochlor occur; roots absorb significantly less of these herbicides than the coleoptile.

Herbicide-resistant crops

Selectivity of herbicides (i.e. the ability to be safe towards the crop and injurious towards weeds) relies on the superior ability of the crop to detoxify (deactivate) the herbicide faster than the weed it is expected to kill. Exceptions are herbicide-resistant (HR) crops that have been engineered to resist the harmful effects of specific non-selective herbicides.

The dominating genetic modification (GM) technology, since the turn of this century, remains Roundup Ready™ transgenic maize, soya bean and cotton that have been made tolerant, and fundamentally resistant, to the non-selective herbicide glyphosate. Examples of other technologies available on the South African seed market, which followed different development routes than Roundup Ready™ technology to produce HR crops, are atrazine-resistant canola and imidazolinone-resistant maize and sunflower.

A sunflower seedling with a heat scorch lesion at the soil surface, which is a possible entry point for pathogens. Affected plants can be more susceptible to herbicide injury.

Herbicides that inhibit photosynthesis

Chilling stress, heat shock and water stress are disruptors of electron transport in the photosynthesis process that occurs in all green plants. The energy contained in electrons is built into carbohydrates that are end-products of photosynthesis, and that constitute the basis of all life on earth.

Disruption of electron flow in the photosynthetic pathway results in these 'freed-up' electrons combining with free oxygen (O_2), which is also formed in chloroplasts of plant cells, to produce toxic active oxygen species, including superoxide, hydrogen peroxide (H_2O_2), singlet oxygen and hydroxyl radical.

These reactive byproducts of disrupted electron flow are strong oxidising agents that attack proteins and lipids, first in the chloroplasts where photosynthesis occurs, with the result that chloroplast membranes and chlorophyll (green pigment of plants) are destroyed – symptoms of this type of plant injury is chlorosis (yellowing) of green plant parts.

These destructive effects of the reactive oxygen species are rapidly followed by destruction of cell membranes, with the result that cell function ceases and cell content is lost – a symptom of this type of plant injury is necrosis (browning and death) of affected plant parts.

Herbicides that inhibit photosynthesis, i.e. triazines (e.g. atrazine, terbuthylazine), bipyridyliums (e.g. paraquat, diquat), bromoxynil and bendioxide all cause disruption of electron flow in the photosynthesis process, exactly in the same way as chilling stress, heat shock and water stress. The reactive oxygen species is what really causes the death of plants that are susceptible to these herbicides, and injury symptoms are chlorosis of green plant parts, followed by necrosis.

Obviously, this correspondence in effects of herbicides and climatic stresses can cloud the diagnosis of causes of plant injury. This fact demands the involvement of 'people skilled in the art' when cases of crop injury are investigated. Because the causative factors in such cases are likely to be multi-factorial in nature, the investigative team ideally should be multi-disciplinary in terms of the skills contributed by team members.

Dr Charlie Reinhardt is dean of Villa Academy, extraordinary professor in weed science at Stellenbosch University, and project leader in the SAHRI at University of Pretoria. For enquiries, contact him on 083 442 3427 or dr.charlie.reinhardt@gmail.com.