



Visual symptoms of herbicide drift on grapevine shoots, leaves and fruit

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Introduction

Herbicide drift towards non-target crops is unfortunately a common occurrence in agricultural regions. Grapevines can exhibit specific negative symptoms after exposure to most herbicides used to control weeds around broadacre crops, or next to roads and on lawns. Depending on climatic conditions, off-target drifts can move for several kilometres and can easily reach vineyards on neighbouring farms. Windy conditions, lower relative humidity and/or higher atmospheric temperatures are all factors contributing to the extent of injury from spray drift. However, linking specific symptoms to a particular herbicide can be difficult, making it problematic to identify the drift source and to avoid future incidents.

Some of the most widely used herbicides in Australian and global agriculture include 2,4-dichlorophenoxyacetic acid (2,4-D), 3,6-dichloro-2-methoxybenzoic acid (Dicamba), 2-methyl-4-chlorophenoxyacetic acid (MCPA) and glyphosate. Many plant growth-regulating herbicides, such as 2,4-D, Dicamba and MCPA, are renowned for causing drift issues. Phenoxyacetic acid type herbicides, including 2,4-D and MCPA, are particularly damaging to grapevines. Glyphosate is commonly used in vineyards to control weeds between vines and can therefore easily reach off-target grapevines.

A simulated drift experiment on potted grapevines was recently conducted at the National Wine and Grape Industry Centre (NWGIC) in Wagga Wagga to better characterise grapevine injury symptoms to specific herbicides. Spring exposures (mid-November) to 2,4-D, Dicamba, MCPA and glyphosate were observed visually over several weeks on five-year-old Tempranillo

grapevines at the cessation of flowering. An automated cabinet boom sprayer was used to apply rates of 65 g/ha of the active ingredient of each herbicide to the allocated vines. This rate represents drifts between 7 and 12% of the recommended label rates of the herbicides. The onset of véraison occurred around 20 December 2017, while berry maturity was attained by 30 January 2018. The information below provides a description and images of the development of the obvious visual shoot, foliar and fruit injuries that were triggered by the different herbicides as the season progressed.

Shoot injuries

Herbicide exposure generally caused downward bending of apical (front) shoot components and also entire shoots after 2,4-D, Dicamba or MCPA treatment. Glyphosate exposure, however, resulted in milder shoot injuries. Shoot tip necrosis (death, as evidenced by tissue browning and desiccation) was also obvious as the experiment progressed, and was induced to some degree by all four herbicides. Exposure to 2,4-D, Dicamba and MCPA induced shoot necrosis, in a basipetal direction from the tip over time. Shoot necrosis was particularly severe following Dicamba exposure. Below is a more specific description of the damage caused by each herbicide over time.

The first visible response to 2,4-D was extensive downward bending (drooping) of the top 10–20 cm of the shoot tip from the day after treatment (Figure 51A). These shoots continued to lose their turgor and wilt during the first 3 days following exposure. After about 3 days, shoot tips started curling, presenting a pig's tail appearance, and also becoming necrotic. Additionally, there was necrosis of the tendrils located near the shoot tips. Downward bending of more shoots and necrosis of additional shoot tips and tendrils continued during the second week after 2,4-D application. By week four, shoot necrosis progressed basipetally and considerable senescence (2–5 nodes) was observed by week

five (Figure 51B). This was not universal, with some shoots instead exhibiting a zig-zag growth pattern with short internodes from this period (Figure 51C). Lateral shoot development initiated within the sixth week after 2,4-D treatment, and continued until berry maturation, however, these were stunted and tended to crowd around the primary shoot (Figure 51D).

Dicamba exposure induced the downward bending of shoot tips, clearly visible from the day after spray application (Figure 52A). Leaf petioles also drooped within the first week after treatment, giving these shoots a wilted appearance (Figure 52A). Curling of shoot tips, in the shape of a pig's tail, occurred within the second week after Dicamba exposure (Figure 52B), whereas necrosis

of the shoot tips and tendrils initiated during the same period, and was particularly widespread from the third week after treatment. This was followed with the senescence of the top of the shoot (2–5 nodes) by week four (Figure 52C), while shoot necrosis progressed basipetally for about 2–4 more nodes throughout weeks five to seven (Figure 52D). A few lateral shoots emerged during the last 2 weeks of the experiment, however these appeared normal. The shoots of grapevines sprayed with MCPA also bent down and resembled a wilted appearance from the day after treatment (Figure 53A). Tendril and shoot tip necrosis, in addition to more severe shoot bending, occurred during the second week after MCPA exposure (Figure 53B).

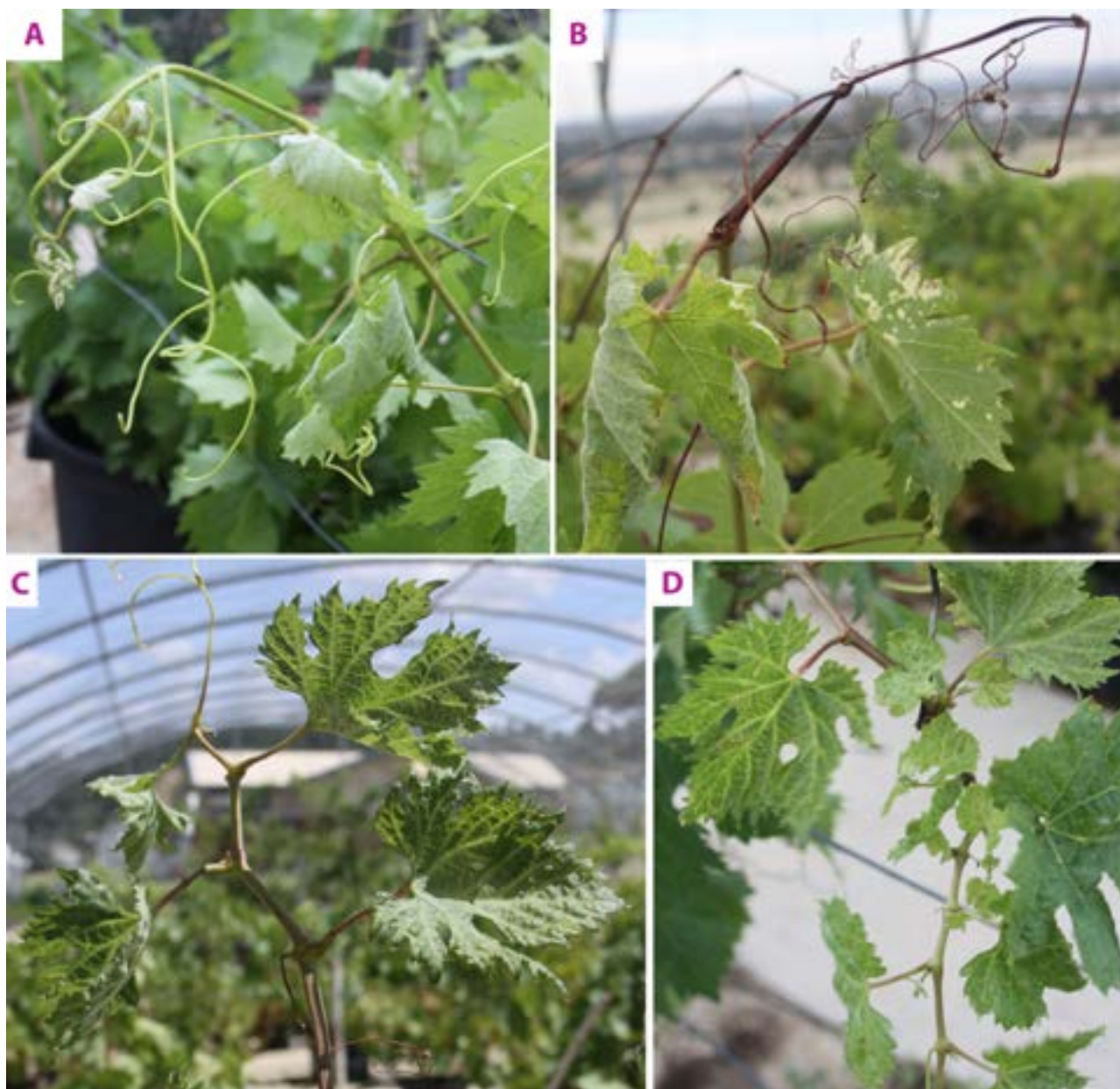


Figure 51. Grapevine shoot appearance after 2,4-D exposure. A: Downward bending of shoots the day after exposure. B: Shoot necrosis, and leaf upward folding and interveinal white chlorotic lesion development, at 29 days after exposure. C: Shoot growth exhibiting a zig-zag pattern, while apical leaves appear unevenly surfaced and cupped, 31 days after treatment. D: Stunted lateral shoots crowding around the primary shoot at 42 days after exposure.

Basipetal progression of shoot necrosis was obvious during week four, continuing for a few weeks (Figure 53C). Normally appearing lateral shoot development initiated from week eight and continued towards berry maturity.

Downward bending of approximately 5-10% of shoots occurred from around 4 days after glyphosate exposure, while only minor shoot tip necrosis also emerged during the same time. Necrosis of a few additional shoot tips and tendrils continued during the second and third weeks after glyphosate treatment (Figure 53D). Normal lateral shoot development became evident from the eighth week after treatment.

Leaf injuries

The timing and symptoms of the leaf injuries were not always herbicide specific. Perhaps most distinct, however, was Dicamba exposure which induced leaf blade rolling in the upward direction in conjunction with the development of yellow and brown interveinal lesions. The leaf injury symptoms of 2,4-D, MCPA or glyphosate exposure were not easily discerned from each other at times. However, 2,4-D exposed vines specifically developed severely deformed lateral shoot leaves, whereas leaf blade margin necrosis was noticeable after MCPA or glyphosate exposure only.



Figure 52. Grapevine shoot appearance after Dicamba exposure. A: Shoot tip drooping, and upward rolling of leaf blades in addition to downward bending of the petiole at two days after treatment. B: Shoot tip curling, and tip and tendril necrosis, 13 days after exposure. C: Shoot necrosis and tip senescence at 22 days after Dicamba application. D: Widespread downward bending of shoots and necrosis at 47 days after treatment.

Emergence of injury signs linked to glyphosate exposure was often delayed, while glyphosate exposure distinctly induced impaired apical lobe development of young apical leaves.

Upward rolling of younger leaf blades was obvious within 24 hours after 2,4-D exposure (Figure 51A). After 3 days, many apical leaf blades appeared shrivelled and continued to roll inward to full leaf blade closure. The shrivelling of these leaf blades subsided slightly by the

second week, however, upward cupping of most apical leaves was visible by this stage. Interveinal white chlorotic lesion development initiated on some of the cupped leaves by 10 days after treatment. In contrast, young leaves near the top of the shoot exhibited a fan-shaped appearance from 12 days after exposure, with small cupped leaf blades, serrated margins (enations) and reduced interveinal spaces apparent (Figure 54A). Prominent white interveinal

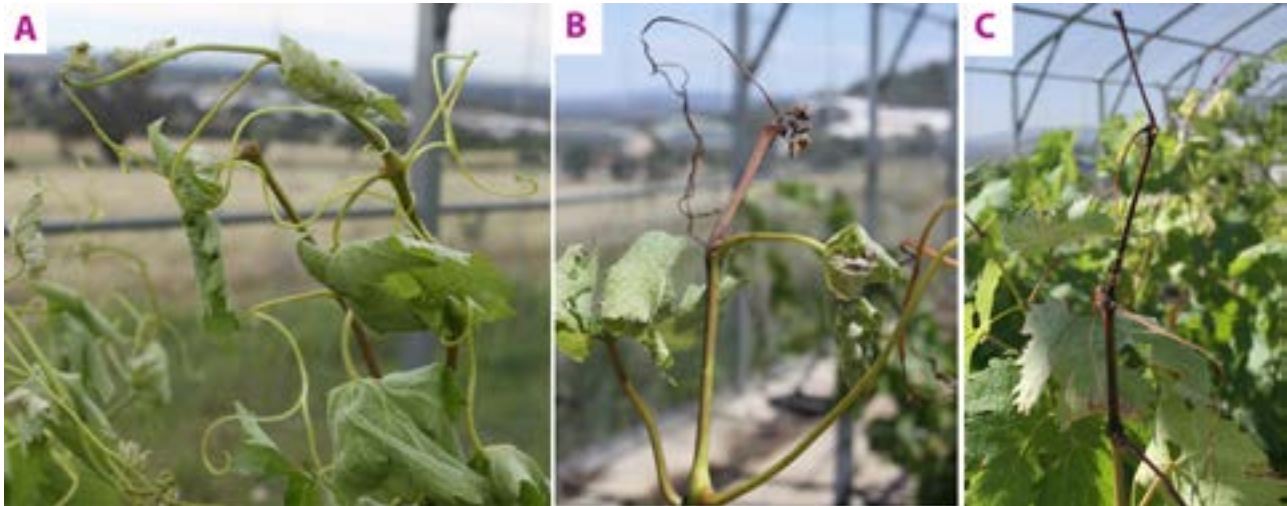


Figure 53. Implications of MCPA exposure on grapevine shoot appearance. A: Downward bending of shoots, in addition to severe upward leaf blade rolling at 2 days after exposure. B: Shoot tip and tendril necrosis, as well as leaf blade upward rolling and margin necrosis at 15 days after treatment. C: Shoot necrosis progressing downward at 22 days after exposure.

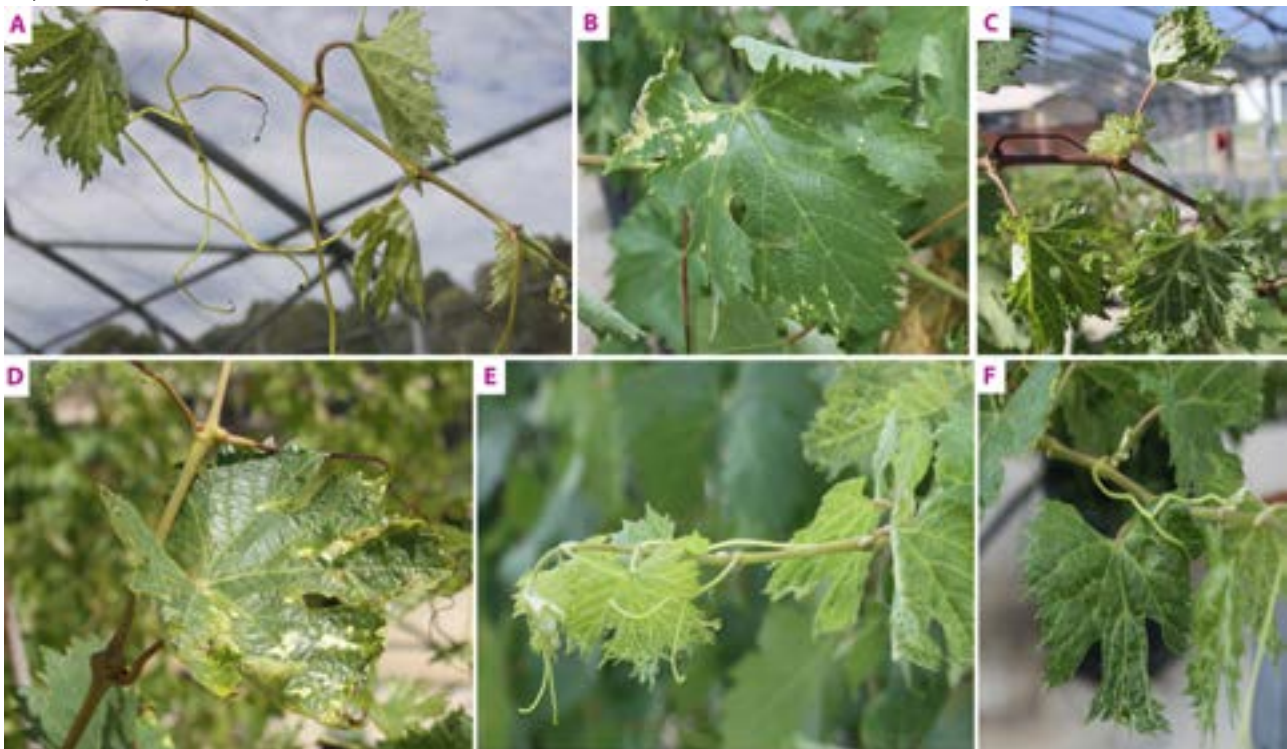


Figure 54. Leaf appearance after 2,4-D exposure. A: Fan-shaped apical leaf, exhibiting cupping, sharp margins and reduced interveinal spaces at 12 days after exposure. B: White interveinal lesion development and leaf margin upward folding at 18 days after treatment. C: Thick apical leaf blades with discoloration around veins and cupping at 50 days after exposure. D: Yellow interveinal lesions and distorted leaf blade shapes at 31 days after treatment. E: Severely deformed small, light coloured lateral shoot leaves at 42 days after treatment. F: Deformed lateral shoot leaves with crowded veins and narrow interveinal spaces at 44 days after treatment.

lesions started to emerge on still expanding leaves a bit further down the shoots within the third week after spraying, in conjunction with mild upward leaf blade folding (Figure 54B). Fan-shaped young leaves displayed thick and uneven, rutted blades, puckered spots and discolouration around leaf veins from the third week after treatment (Figure 54C). By week five, many leaves on different shoot positions exhibited interveinal white or yellow chlorotic lesions and/or distorted blades (Figure 54D). Severely deformed lateral shoot leaves emerged from week six, remaining small and lacking pigmentation to maintain a light green appearance (Figure 54E). Lateral leaves were also very crowded around the shoot (Figure 54E), and exhibited reduced or narrow

interveinal spaces, crowded veins and sharp margin teeth from week seven (Figure 54F). Distinct upward margin rolling of apical leaves initiated from the day after Dicamba exposure (Figure 55A), continuing throughout the first and second weeks after treatment. By 2 days after exposure, many fully closed leaf blades were visible on the youngest region of the shoot (Figure 53A). Younger leaves still exhibited upward rolled margins by 2 weeks after exposure, as well as pale interveinal yellow lesions by this stage (Figure 55B). Interveinal lesion development intensified from week three, with yellow and brown or black lesions appearing on many apical leaves in conjunction with upward leaf margin rolling (Figure 55C).

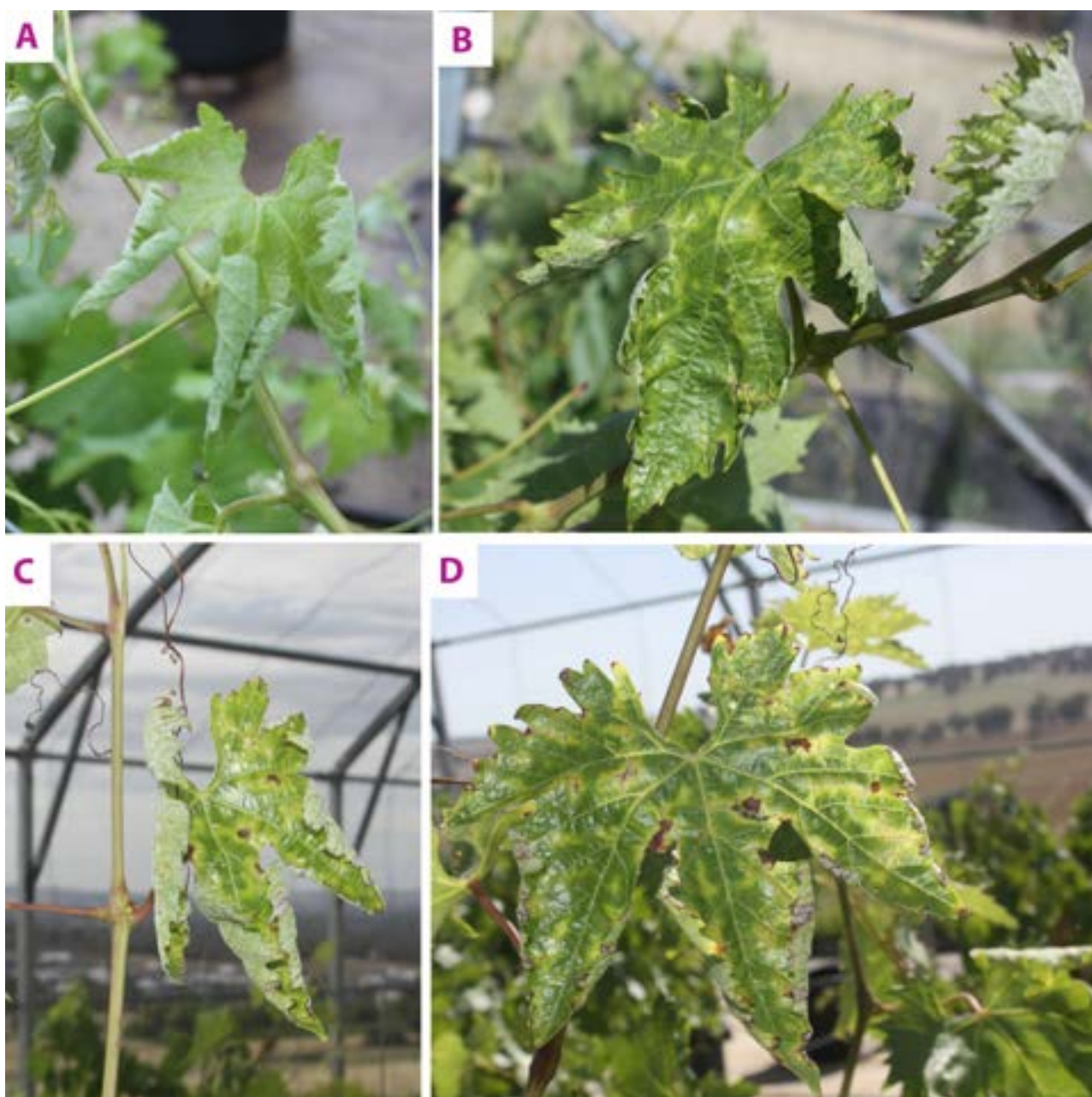


Figure 55. Leaf appearance after Dicamba exposure. A: Upward margin folding the day after treatment. B: Minor upward margin rolling and pale yellow interveinal lesion development at 13 days after exposure. C: Yellow and brown interveinal lesions and upward leaf margin rolling at 19 days after treatment. D: Yellow and brown interveinal lesion and upward margin rolling at 32 days after exposure.



Figure 56. Leaf appearance after MCPA exposure. A: Upward blade folding and petiole epinasty 1 day after treatment. B: Upward folding and cupping of apical leaves at 13 days after exposure. C: White interveinal lesion development, and upward margin folding and necrosis at 18 days after treatment. D: Deformed apical leaves with uneven surfaces and white interveinal lesions at 26 days after treatment. E: Leaf margin necrosis and distorted blade shapes at 36 days after exposure.

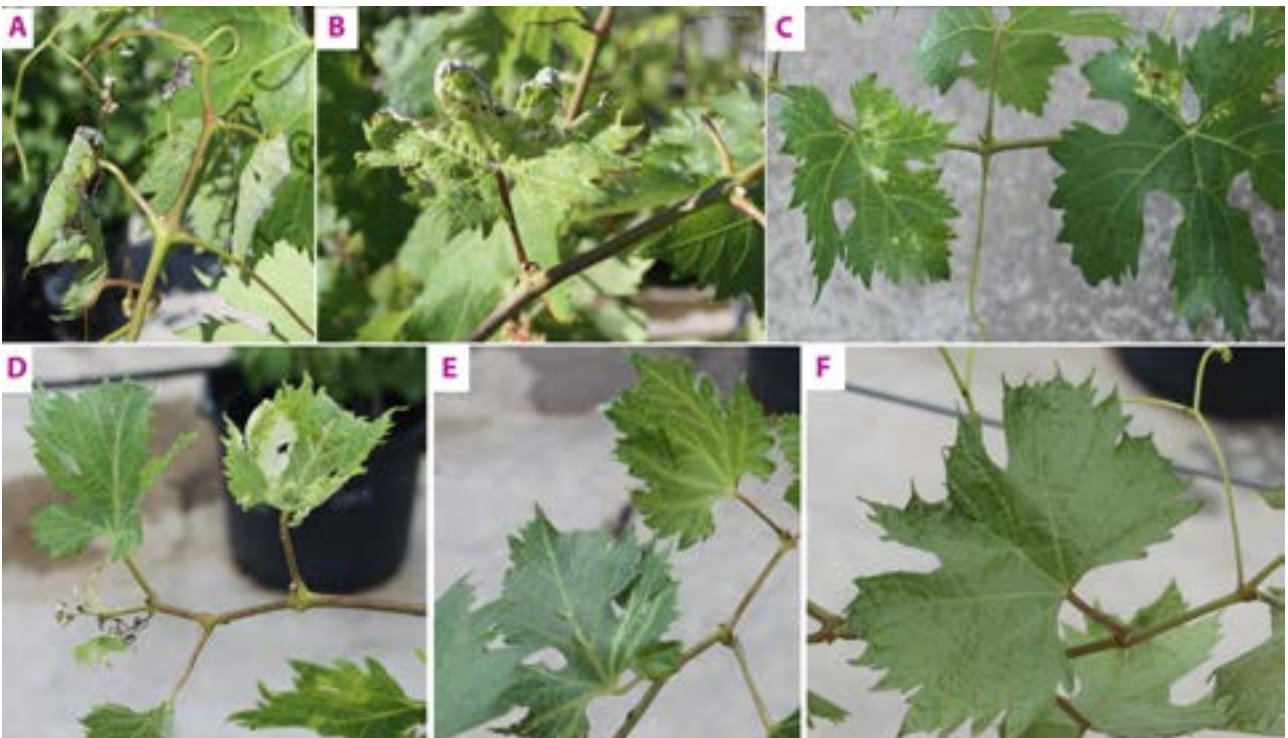


Figure 57. Leaf appearance after glyphosate exposure. A: Upward leaf blade folding and margin necrosis occurring at 4 days after exposure. B: Margin necrosis at 22 days after treatment. C: White or yellow lesions developing on apical leaf blades at 16 days after exposure. D: Cupping of apical leaves, as well as uneven blade surfaces and white stain development at 29 days after exposure. E: Discolouration around leaf veins and crowding of veins apparent at 54 days after treatment. F: Impairment of young leaf apical lobe development at 58 days after exposure.

Development of leaf lesions continued throughout weeks four and five (Figure 55D), and were present until the end of the experiment. Some leaves further down the shoots also started to exhibit upward margin rolling from week five onwards. Lateral shoot leaves emerged from week ten, presenting uneven surfaces and rounded blades.

Exposure to MCPA induced upward blade folding and epinasty of young apical leaves from the day after treatment (Figure 56A). Two days after treatment, young leaf blades were severely rolled up to full closure (Figure 53A), however, the tightness of leaf rolling was reduced by the second week (Figure 56B). Young leaves on the uppermost two to three nodes appeared cupped by the second week after treatment, whereas the blades of expanding leaves further down the shoot continued to roll up. In the third week, leaf margin rolling continued on additional older expanding leaves (5–10 nodes below the shoot tip), whereas on apical leaves distinct white interveinal chlorotic lesions emerged along with margin necrosis (Figure 56C). Deformation of young leaves continued during weeks four and five, resulting in uneven leaf surfaces and rough or sharp leaf margin serrations in addition to more severe margin necrosis (Figure 56D and E). Upward rolling continued further down shoots during weeks six to nine, whereas additional young leaf margin necrosis development also occurred during this period. Lateral shoot development occurred from week eight, with these leaf blades formed in a round shape.

Apical leaf blades on the vines treated with glyphosate started to roll up from about 4 days after spraying, and margin necrosis also set in (Figure 57A). Young leaf margin necrosis continued during the second and third weeks (Figure 57B), along with the emergence of yellow or white interveinal chlorotic lesions on some of these leaves (Figure 57C). Other types of young leaf blade distortion were evident from the fourth week, with the development of cupping, sharp margin serrations, crowded veins and uneven surfaces (Figure 57D). Deformation of young leaves continued during week five, with the onset of distinctive white discolouration near the veins (Figure 57E). By week six, some young leaf margins appeared serrated, while impaired apical lobe development seemed to occur as the leaves expanded (Figure 57F). More discolouration around young leaf veins was observed 7 and 8 weeks after treatment, when apical leaves also appeared fan-shaped with uneven surfaces and crowded veins. Leaf margin necrosis additionally progressed along the older nodes further down

the shoot at around week eight. Lateral shoot leaves emerged from week nine, exhibiting leaf blades with little to no sinus differentiation.

Fruit injuries

Curving of bunch stems was the first and most prominent early sign of bunch injury sign following exposure to 2,4-D, Dicamba or MCPA just after the cessation of flowering. Exposure to 2,4-D resulted in the most severe visual symptoms, including noteworthy berry or whole bunch necrosis. Dicamba exposure was characterised by bunch millerandage ('hen and chicken' appearance), whereas glyphosate related bunch symptoms were mild and generally emerged later than those of the other treatments.

Minor bunch stem curvature was noticeable during the first week after 2,4-D exposure, while necrosis or abortion of individual peppercorn sized berries and pedicels were also observed (Figure 58A). By week three, fruit were pea-sized and more berry necrosis was evident. Full necrosis of some bunches or necrosis of the basal portion of the bunch, including the berries, pedicels and rachis were noteworthy by week five after treatment, just prior to the start of véraison (Figure 58B). By week eight, when the fruit had intermediate sugar levels, ripening appeared uneven with some green berries still undergoing véraison. By week 11 at fruit maturity, various bunches still contained some green berries with berry necrosis widespread on many bunches.

Curvature of bunch stems was prominent within the first week after Dicamba application, with some minor berry abortion that was not evident in the control treatment. Most bunches had a 'hen and chicken' appearance from this period onwards (Figure 59A). By week five, bunches of Dicamba treated vines exhibited distinct millerandage throughout the length of the bunch (Figure 59B). A small number of whole bunches on Dicamba treated vines were necrotic by berry maturity.

Exposure to MCPA resulted in curved bunch stems and noteworthy millerandage a week after treatment (Figure 60A). By week five, full necrosis of some bunches was noted, whereas bunch millerandage was still noticeable by the final harvest when the fruit was mature (Figure 60B).

Glyphosate exposure only had minor effects on bunch appearance. However, mild bunch stem curvature was noted by the fifth week after treatment. Likewise, by week 11 when the fruit had matured, glyphosate treated vines exhibited mild millerandage.



Figure 58. Bunch appearance after 2,4-D exposure. A: Slight bunch stem curving and necrosis of individual berries and pedicels 8 days after treatment. B: Necrosis of whole bunches or basal bunch parts at 34 days after treatment.

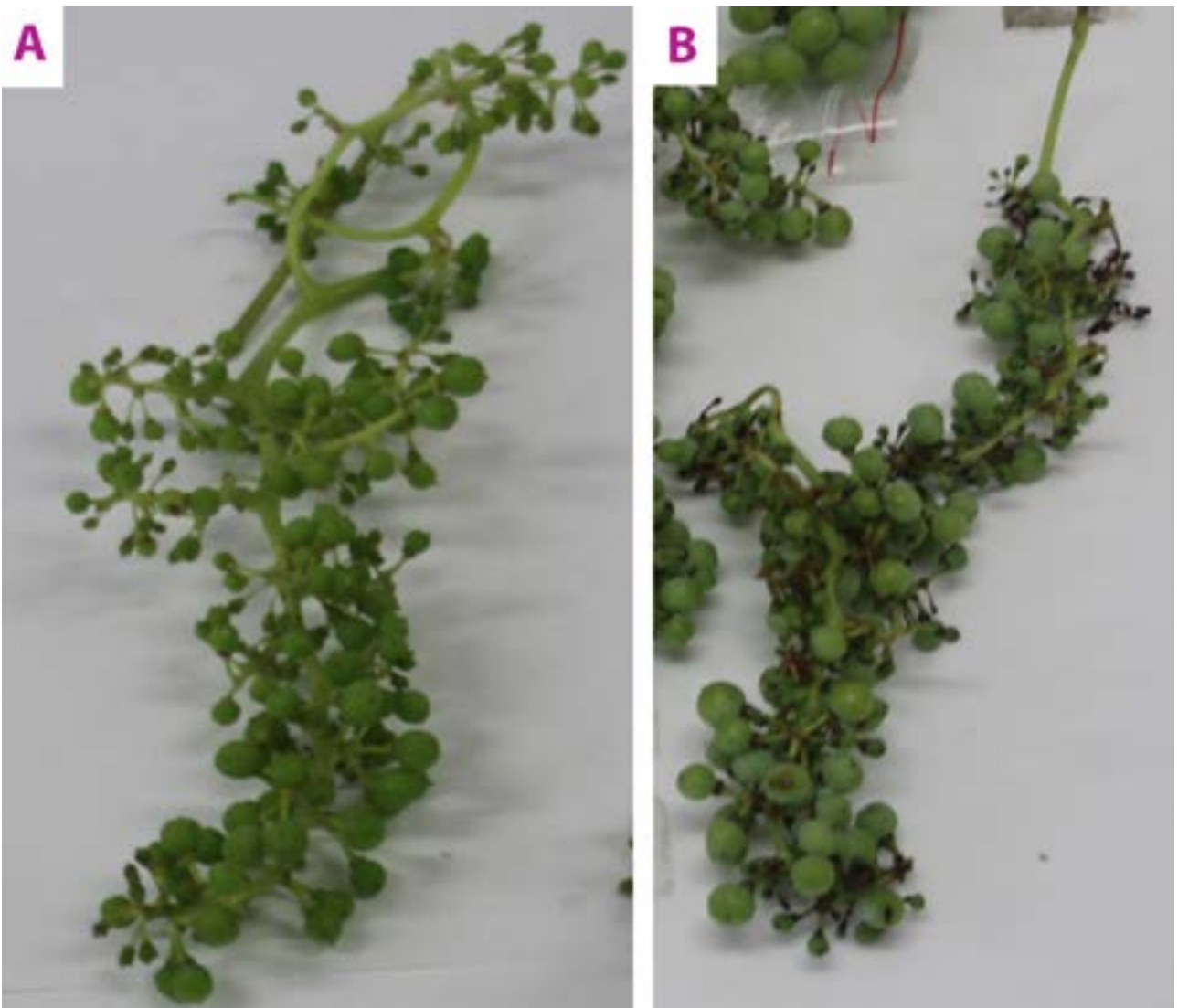


Figure 59. Bunch appearance after Dicamba exposure. A: Mild stem curvature and bunch millerandage at 8 days after treatment application. B: Millerandage visible across the bunch length at 34 days after exposure.



Figure 60. Bunch appearance after MCPA exposure. A: Bunch stem curvature and bunch millerandage present at 8 days after exposure. B: Bunch millerandage at 73 days after treatment.

Conclusions

The visual assessment and identification of grapevine damage related to 2,4-D, Dicamba, MCPA or glyphosate exposure can be confusing. Many injury signs caused by each of the four herbicides are similar and therefore hard to distinguish. However, Dicamba exposure induced unique injury signs, especially upon leaf development.

Vines injured by Dicamba exhibited upward leaf rolling in conjunction with yellow and brown interveinal lesion development. Being chemically similar, 2,4-D and MCPA exposure induced several comparable symptoms. However, unlike MCPA, 2,4-D damage did not exhibit leaf margin necrosis, whereas severely deformed lateral shoot leaves only developed after 2,4-D exposure. Glyphosate related injuries mainly emerged later than those induced by the other herbicides. Impaired development of the apical lobe of young leaf blades was perhaps the most distinct feature of glyphosate damage.

This guide to visually identify grapevine responses to the above-mentioned herbicides can hopefully assist growers in future seasons to promptly recognise and address common herbicide drift related issues in vineyards. Not included in this report, the study also included an assessment of vine physiological and biochemical responses to the herbicides, which will provide further information useful to understand and address herbicide drift issues in vineyards.

Take home messages: what can I do to minimise damage after herbicide exposure?

Avoid or limit cane pruning. Growth regulating herbicides (2,4-D, Dicamba and MCPA) impair bud fruitfulness, especially those on higher shoot positions.

Spur pruning is a safer option. The basal bud health is less affected after exposure to growth regulating herbicides.

Avoid water stress during berry ripening. Young leaf photosynthesis is impaired by phenoxyacetic acid herbicides and glyphosate, and irrigation practices can contribute to the retention and functioning of older leaves. Older leaf functioning subsequently becomes important towards fruit ripening, particularly if herbicide affected vines carry a substantial crop load.

Apply postharvest or late season fertilisation and irrigation especially to younger vines with developing root systems. Growth regulating herbicides impair root growth and stimulation of root development during the postharvest/late season period becomes crucial. Avoiding water constraints and nutritional deficiencies during this period should promote the development of a healthier root system.