From:	Rebecca Murray
To:	[redacted]
Subject:	LGOIMA request for the most recent risk assessment/s of the use of glyphosate herbicides
Date:	Monday, 17 June 2024 4:05:00 pm
Attachments:	HS-022 - Chemicals and Hazardous Materials - Handling and Storing.pdf HS-150 - Weed Spraying.pdf JRA - Granular Glyphosate (2).pdf Herbicides for Vegetation Control Report to Strategy Planning and Engagement Committee - 13 February 2023.pdf Copy of DCC Sealed and Unsealed Road Network Vegetation Control.pdf image001.png
Importance:	High

Kia ora [redacted]

Local Government Official Information and Meetings Act 1987 (LGOIMA) Request

In response to your LGOIMA request received 17 May 2024 requesting the most recent risk assessment/s of the use of glyphosate herbicides in Dunedin, please find attached the following:

- Hazard control sheets for the use of chemicals and hazardous materials, and weed spraying undertaken by the contractor (Delta).

- Job Risk Analysis (JRA) for weed control glyphosate granular herbicide and a copy of information regarding vegetation control as part of DCC sealed and unsealed road network maintenance contracts by the contractor (Fulton Hogan).

Also attached for your information is a copy of the Herbicides for Vegetation Control Report presented to the Strategy, Planning and Engagement Committee on 13 February 2023 which may be of interest.

I trust this answers your request.

Kā mihi

Rebecca Murray
Mana Whakahaere Kairuruku / Governance Support Officer
Governance Group

P 03 4774000 | [redacted] | **E rebecca.murray@dcc.govt.nz**
Te Kaunihera a Rohe o Ōtepoti - Dunedin City Council
50 The Octagon, Dunedin
PO Box 5045, Dunedin 9054
New Zealand
www.dunedin.govt.nz

From: [redacted]
Sent: Tuesday, 11 June 2024 9:36 am
To: Rebecca Murray <Rebecca.Murray@dcc.govt.nz>
Subject: Re: LGOIMA Request 1048459 Overdue

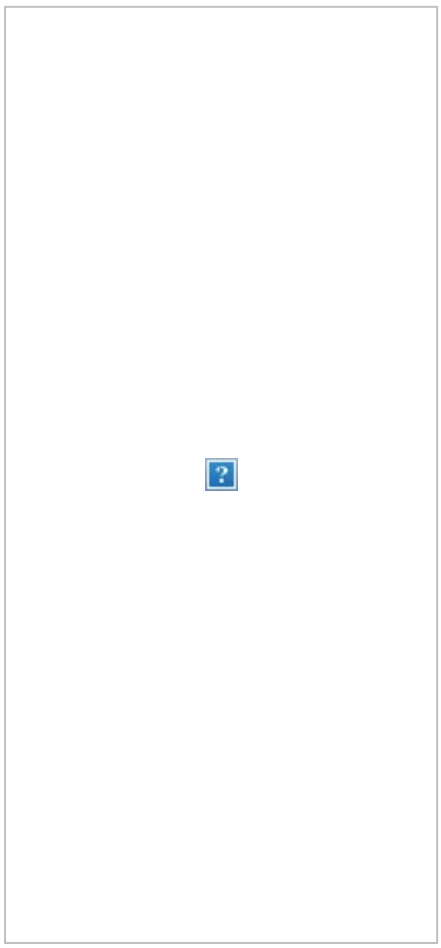
Deepest apologies, I put my date into a working days calculator but obviously must have made a mistake. Sorry for any confusion.

I look forward to hearing from you.

Kind regards,

[redacted]

On Tue, 11 Jun 2024, 09:12 Rebecca Murray, <Rebecca.Murray@dcc.govt.nz> wrote:

> Kia ora [redacted]
>
> Thank you for your email.
>
> The statutory timeframe for a LGOIMA request is 20 working days, your LGOIMA is due date is 17 June 2024 which takes into account King's Birthday (3 June 2024) which is a public holiday.
>
> Below is a link to the Ombudsman's website which provides a 20 working days calculator for LGOIMA due dates. I have also provided below a snippy of the 20 working day due date calculation for your LGOIMA.
>
> **https://www.ombudsman.parliament.nz/**
>
> 

I am in the process of collating and reviewing the information.

Kā mihi

Rebecca Murray
Mana Whakahaere Kairuruku / Governance Support Officer
Governance Group
P 03 4774000 | [redacted] | **E** **rebecca.murray@dcc.govt.nz**
Te Kaunihera a Rohe o Ōtepoti - Dunedin City Council
50 The Octagon, Dunedin
PO Box 5045, Dunedin 9054
New Zealand
www.dunedin.govt.nz

From: [redacted]
Sent: Monday, 10 June 2024 5:47 pm
To: Official Information <officialinformation@dcc.govt.nz>
Subject: LGOIMA Request 1048459 Overdue

Dear Dunedin City Council,

I filed LGOIMA Request 1048459 on Friday May 17. Over 20 working days have surpassed since that date and you have not responded to that request, as you are legally required to.

Please contact me as soon as possible regarding this request.

Kind regards,
[redacted]

[redacted] has submitted a LGOIMA request - 1048459.

Below are the details of the request

Request details:
Could you please provide access to the most recent risk assessment/s of the use of glyphosate herbicides in Dunedin. Please feel free to email or call me regarding my request. Kind regards, [redacted]

File attachment (file name**)**
No file uploaded

Name
[redacted]

Email address
[redacted]

Mailing address
[redacted]

Contact phone number
[redacted]

CHEMICALS & HAZARDOUS MATERIALS – HANDLING & STORING

Personal Protective Equipment:

- Safety footwear
- Chemical resistant gloves
- Safety glasses
- Spray suit/overalls
- Breathing apparatus/face masks

Pre-Work Checks:

- **Prior to using any chemicals/hazardous materials obtain and read the appropriate SDS sheets**
- Wind direction

HAZARD	*RISK RATING*	*CONTROL PLAN* *Action required to minimise*
Contact, handling, inhalation, swallowing	High	a) Follow all safety precautions contained on the MSDS sheets and on the labels b) Wear appropriate protective clothing and safety equipment for the chemical/hazardous material in use c) Ensure all containers are well labelled
Storage / spillage	High	a) Always have sawdust/spill kit in area in case of spillage b) Store in correct locations c) Store to have MSDS and hazard sign in place d) Well ventilated e) Contact Supervisor/Manager immediately after emergency services notified f) Store list of chemicals and amounts off site away from chemical store
Fire / explosion	High	a) Ensure fire extinguisher is available b) No smoking c) Clean up any spillage d) Store in appropriate container/s e) Ensure storage separation
HAZARD ANALYSIS AND CONTROL PLAN TO BE REVIEWED AND AMENDED IF THE SCOPE OF WORK CHANGES		

RISK RATING

Low	Normal work conditions apply, be aware of potential change.
Moderate	A moderate risk exists. Additional care, resourcing may be required.
High	sk exists. A high level of planning is necessary.
Critical	The risk is of an extreme nature. Work should not proceed without supervision; a formalised plan & management sign off.

WEED SPRAYING

Personal Protective Equipment:

- Safety footwear
- Chemical resistant gloves
  Protective Eyewear as per SDS (Safety Data Sheet) for chemical being used
- Spray suit/Overalls
- Respirator when required as per SDS for chemical being used

Pre-Work Checks:

- Ground conditions and work area to ensure it is safe
- Communications are established prior to commencing all site work in case of an emergency
- Wind direction

HAZARD	*RISK RATING*	*CONTROL PLAN* *Action required to minimise*
Contact, handling, inhalation, swallowing	High	a) Prior to using any chemicals/hazardous materials obtain and read the appropriate SDS b) Follow all safety precautions contained on the SDS and on the labels c) Fill Spray containers/units to agreed levels to avoid spillage when being carried on a person d) Avoid spraying in windy conditions & dead calm. A light breeze is most suitable e) Wear appropriate protective clothing and safety equipment for the chemical/hazardous material in use – refer to NZS 8409 f) Ensure all containers are well labelled
Potential harm to the public / pedestrians etc.	Moderate	a) Ensure signage is put in place if using chemicals/hazardous materials in a public situation b) Ensure public are informed and if necessary keep them away from the site Ensure all containers are well labelled
Unskilled / untrained operator as applicator	High	a) Ensure that you have received the appropriate training to use the chemical/hazardous material in question b) Some materials and chemicals/hazardous materials require specialist training and handling
Storage / spillage	High	a) Always have sawdust in area in case of spillage b) Store in correct locations c) Store to have SDS and hazard sign in place d) Well ventilated e) Contact Manager immediately after emergency services notified f) Follow Guidance on SDS if chemical comes into contact with skin/eyes/mouth etc.
Vehicle moving when being filled from water source	High	a) Check ground conditions to ensure it is safe to proceed b) Always Park vehicle side on (parallel) to the edge of the water source c) Ensure hand brake is fully applied d) Place wheel chocks under both rear wheels e) When full move vehicle to a safe level location before retrieving wheel chocks
Disposal of empty containers	High	a) Triple rinse or return to place of purchase

HAZARD CONTROL SHEET

HAZARD ANALYSIS AND CONTROL PLAN TO BE REVIEWED AND AMENDED IF THE SCOPE OF WORK CHANGES	
RISK RATING	
Low	Normal work conditions apply, be aware of potential change.
Moderate	A moderate risk exists. Additional care, resourcing may be required.
High	A significant risk exists. A high level of planning is necessary.
Critical	The risk is of an extreme nature. Work should not proceed without supervision; a formalised plan & management sign off.

VC VEGETATION CONTROL

Vegetation Control shall be completed in accordance with TNZ C21.

General

The inspection, reporting, programming and control of vegetation by chemical and/or non-chemical methods including physical removal and mowing.

This section outlines the specifications required to maintain the vegetation growth within the Principal's road network.

Vegetation Control also includes the following activities:

- Vegetation envelope maintenance,
- Tree pruning and removal,
- Mowing,
- Roadside vegetation spraying and
- Noxious Weed Management.

Standard drawings are illustrated in Standard Drawing AM-002/1, AM-002/2, AM-005 & AM-006/1.

The frequency of vegetation control required shall relate directly to the growing conditions. To maintain the specified level of service, increased vegetation control frequencies shall be required at certain times of year.

Departures from TNZ C21

Any references of "State Highway" shall be replaced with "Local Roads". This specification has precedence over TNZ C21 where any discrepancies exist.

The following clauses override TNZ C21.

Clause 2.1 - Noxious Weeds include:

- Barberry *(Berberis glaucocarpa)*
- Blackberry *(Rubus fruticosus)*
- Bomarea
- Broom (*Cystisus scoparius, Teline monspesulana)*
- Boneseed *(Chrysanthemoides monilifera)*
- Boxthorn *(Lycium ferocissimum)*
- Gorse *(Ulex spp.)*
- Hawthorn (under 2m high) *(Crataegus monogyna)*
- Hemlock *(Conium maculatum)*
- Montpellier Broom *(Teline monspessulana)*
- Nodding Thistle *(Carduus nutans)*
- Cape Ivy *(Senecio Angulayus)*
- Old Mans Beard *(Clematis Vitalba)*
- Privet *(Ligustrum lucidum, L. sinense)*
- Pampas *(Cortaderia selloana, C jubata)*
- Ragwort *(Senecio jacobaea)*
- Spiny Broom *(Calicotome spinose)*
- Sweet Brier *(Rosa rubiginosa)*
- Toadrush *(Juncus Bufonius)*
- Tutson *(Hypernium adraseamum)*

- Whire Edged Nightshade *(Solanum marginatum)*
- Wild Ginger *(Hedychium, gardnerianum, flavescens)*
- Wilding pines (under 2m)
- Woolly Night Shade *(Solanum mauritianum)*
- Any other plants listed on the ORC noxious list

Clause 3.1.2: Table One is replaced with the following table:

Standard Type	Grass height tolerance (mm)	Approx. Quantities	
		Sealed Network	Unsealed Network
Type 1	30 – 100. Plus all cuttings shall be picked up and disposed of off-site by the Contractor	231,538m2	4,704m2
Type 2	50 - 200	240,425m2	13,297m2
Type 3	50 - 300	205,031m2	80,925m2

The locations of the different standard types are shown in the GIS maps provided in Appendix B6.

Add to Clause 3.5:

All noxious plants within the legal road reserve shall be treated with a suitable chemical approved by the ORC and the Engineer, including an anti-drift wetting agent and a coloured dye to show where spraying has been undertaken.

Add clause 3.5.9: No-Spray Areas

The following areas shall be maintained by non-chemical means only:

- Frontages that are maintained by private individuals in a neat and tidy condition.
- Intertidal Areas

The following areas shall not be treated by the Contractor:

- All properties listed in the "No-spray" Register provided in Appendix A4.
- Frontages displaying "No Spray" signs or similar.

Add to clause 3.5.7. Chemicals are not to be used:

- Within 500 metres of schools, play centres, kindergartens or community activity centres an hour before and after they are in use.
- At weekends or statutory holidays unless approved by the Engineer.
- When wind speed exceeds 10 km/hr.

Clause 3.6.1: Unsealed (Metal) Shoulders

Amend clause to read: "The required control standard is illustrated in Appendix 1 attached to this Specification" for treatment around edge marker posts. Between edge marker posts, the treatment shall continue at the same width (at least 1m behind edge marker posts) to ensure all vegetation is be treated, unless explicitly excluded. Where no edge marker posts are present, vegetation stall be treated to a distance of 2m from the edge of seal or pavement (on unsealed roads).

Add to clause 4: Performance Criteria.

That all vegetation covered in the scope of works shall be within the specified level of service at any one time.

All quality control and testing has been completed and submitted in the correct format, and is approved by the Engineer.

Delete Section 5: Basis of Payment

Additional Requirements

6.8.1.4 Advertisement

Public Notice shall be prepared by the Contractor and forwarded to the Engineer allowing it to be placed in the Otago Daily Times "DCC Notice Board" of the intention to apply herbicides at least one week prior to work commencing in any area and once again every month during spraying season. The advert shall include details of where spraying will take place. No spray work shall be undertaken until the Engineer holds a copy of the appropriate advertisement.

6.8.1.5 Resident Notification

A small number of residents are severely allergic to any chemicals and DCC has an agreement to inform them of any spraying undertaken within a 5 km radius of their property. The contact details for these residents will be supplied to the Contractor. The Contractor shall give them at least 7 days notice of their intention to spray near their properties. The Contractor shall cooperate with any reasonable request to adjust the programme.

There are also a small number of commercial produce properties that shall be contacted prior to spraying their property frontages. The contact details for these businesses will be supplied to the Contractor who shall give them at least 7 days' notice of the intention to spray near their properties.

6.8.1.6 Safety Issues

Any vegetation causing safety issues shall be removed within 48 hours.

Ordered Works

Ordered works shall consist of any additional vegetation control required within the network as required by the Engineer.

Job Risk Analysis

	Likelihood				
	A Certain	B Likely	C Possible	D Unlikely	E Rare
Major	E	E	E	H	M
Moderate	E	E	H	M	L
Minor	E	H	M	L	L
Insignificant	H	M	L	L	L

Legend
EXTREME
HIGH
MODERATE
LOW

Weed Control	*Glyphosate Granular Herbicide*

Overview

This Job Risk Analysis (JRA) identifies tasks associated with the application process, the hazards and mitigation process.

A Site-specific job log must be completed daily in conjunction with having read this JRA.

Hierarchy of Controls

The range of control measures must follow the following hierarchy;

- Elimination - the total removal of the hazard.
- Minimise - the impact of the hazard. This may include
  - Substituting (wholly or partly) so the risk is lesser
  - Isolating the person from exposure or contact
  - Engineering controls
  - Administration
  - PPE

Key:

LH = Likelihood H = High
E = Extreme M = Moderate
L = Low EL = Eliminate
S = Substitute I = Isolate
EN = Engineering A = Administration
P = Personal Protective Equipment (PPE)

Product	Information	Identified Hazards	LH Before controls	Control method	ACTION - to Eliminate/Isolate/Minimise	LH After controls
Weed Control - Glyphosate Granular Herbicide	Glyphosate Granular Herbicide (GGH) is a broad spectrum, non-selective, non- residual herbicide that is non-volatile and may be used in agriculture, horticulture and for other uses including general weed control. The herbicide is absorbed by plant foliage and green stems. It is inactivated immediately upon contact with soil and does not provide residual weed control. GGH moves from the point of foliage or stem contact into the root system, controlling the plant above and below the ground. Visible effects may take up to 14 days or even longer depending on growing conditions. These effects are a gradual wilting and yellowing of the foliage which advances to deterioration of above and below ground parts. Apply GGH only to healthy, actively growing plants with adequate leaves for herbicide uptake. Avoid treating plants that are drought affected, frost damaged, silt laden or suffering the effects of previous herbicide application. Rainfall occurring up to 2 hours after application may reduce effectiveness. Apply with well maintained and calibrated spray equipment. (This product should only be mixed, contained in or sprayed by, equipment made from stainless steel, fibre glass, plastic, aluminium, brass or copper) All spray equipment, including pumps, spray tanks, lines, nozzles, and landing gear (aircraft) should be thoroughly washed with water after each day of spraying Record daily all spray jobs on either spray record sheet or Job Log:-record name of chemical used, quantity, general wind speed and area.	• Inappropriate usage	H	A	All operators must be certified – GROWSAFE agrichemical certification (valid 5yr) The guidelines set down in the NZ Standards Growsafe Code of Practice must be adhered to All spraying work done must comply with resource consent conditions Read the manufacturers recommended use and the directions to be well acquainted on safe handling of products before use Weeds should be clean, actively growing and not under stress from drought, frost, cold, water logging, grazing or previous herbicide applications DO NOT apply if rainfall is likely within 2 hours Keep chemicals in their original containers Return chemicals to storage areas immediately after use Read the label and follow instructions carefully Use the right product for the problem Always complete a job log for the task Check spraying equipment - ensure it is in good condition and properly calibrated	L
		• Maybe harmful if swallowed, inhaled or absorbed through the skin	H	p	Treat all chemicals as hazardous - they can enter the body by being inhaled, absorbed through the skin or swallowed Avoid contact with eyes and skin. When mixing or applying, wear overalls, boots, gloves and eye protection. Clean protective clothing daily after work. Do not eat, drink or smoke while using. Remove protective clothing and wash hands and face thoroughly before meals and after work. Assess exposure and use any additional measures to keep airborne levels below any relevant exposure limit Avoid inhalation of dust or spray mist. If airborne mist or vapors are generated, use respiratory protection to a minimum of Organic Vapor cartridge type and/or local exhaust ventilation controls. Use low pressure, air induction nozzles and spray low to the ground, to avoid spray mist Use spray additives, when possible, to keep spray mist down	L

Product	Information	Identified Hazards	LH Before controls	Control method	ACTION - to Eliminate/Isolate/Minimise	LH After controls
		• May damage foliage or green stems of desirable plants	H	A	DO NOT apply in conditions where spray drift may occur, e.g. wind Avoid application in winds which allow drift onto desirable plants. Place nozzles within the air stream and not across it in forced air systems Always spray as close as practical to the target Increase/decrease the flow rates of the application where necessary/recommended	L
		• Contamination of land, air, and water, which can in turn lead to adverse effects on human health and ecosystems	H	A	Ensure safe transportation of chemicals and equipment - transport minimal quantities, secure knapsacks to avoid them tipping, secure lids and tractor couplings Check knapsacks, sprayers before use to insure they are in good working condition - any problems must be rectified promptly Mix chemicals away from water source, to prevent contamination in case of spill - ensure spill kit is available before any mixing proceeds Chemicals should be stored in a secure locked area separate from foodstuffs, seed, animals and other incompatible chemical Always rinse out containers into the tank Mix sufficient chemical for the job - do not leave chemicals standing in sprayers due to leakages, unidentifiable mix, corrosion etc. When spraying make sure that it does not drift onto neighbouring urban areas, houses, schools, public roads and access-ways, crops or gardens Use a drift control additive where recommended/required Check for sensitive areas - accurate spraying methods are required next to streams, rivers, lakes and wetlands	L

HERBICIDES FOR VEGETATION CONTROL

Department: Transport

EXECUTIVE SUMMARY

1 This report responds to the resolution of the Planning and Environment Committee on 6 July 2022 that requested a review of the use of herbicides, especially those containing glyphosate, for weed and vegetation control in Council operations, and what alternative vegetation control methods exist.

2 The report also presents the findings of a staff literature review on the health or other risks associated with herbicide use.

RECOMMENDATIONS

That the Committee:

a) **Notes** the Herbicides for Vegetation Control Report.

BACKGROUND

3 This report responds to the resolution from the Planning and Environment Committee on 6 July 2022 where the Committee resolved:

Moved *(Cr David Benson-Pope/Cr Steve Walker):*

That the Committee:

Requests *that staff review the use, by Council departments and contractors, of herbicides (especially those containing glyphosate) for weed and vegetation control and report on any health or other risk and alternative products, where they exist.*

Motion carried (PLA/2022/011)

DISCUSSION

Herbicide Use

4 The Dunedin City Council (DCC) is responsible for weed and vegetation control within road corridors, parks, reserves, cemeteries, landfills, around 3 Waters infrastructure and DCC property.

5 Herbicide sprays are the primary method used by DCC Contractors to control undesirable weeds and vegetation.

6 Herbicides can be known by brand names, common names and chemical names. DCC contractors use many various product brands, and some may not be listed in the table below. Different branded products regularly contain other additives (often proprietary) to improve the product effectiveness. Contractors may also use additives to improve the sprays' effectiveness for certain weed types.

7 The table below identifies the main herbicides used by DCC contractors.

Common name	Typical Brand Names	General Uses	Approximate Yearly Volume of Usage (undiluted litres)
Glyphosate[1]	· Roundup · KiwiGarden · Yates · WeedMaster · Orion · NuFarm	Broad control of all weeds, primarily using sprays. Used on road shoulders, unlined surface water channels, lawn edges, kerb and channel, around buildings, footpaths.	Transport: 3,500 Parks and Recreation: 1,625 Three Waters: 30 Property: 28
Picloram[2]	· Tordon · Triumph · Vigilant · Ken-Zon	Spraying for pest plants, noxious weeds, woody weeds. Gel form, with brush applicators used for tree stumps/limbs to prevent regrowth.	Transport: 100 Parks and Recreation: 125 Three Waters: <5 Property: <2
Benzalkonium Chloride[3]	· MossBoss · Surrender · Moss and Mould Sorted Commercial	Spraying to control moss and lichen.	<10 combined

Herbicide chemical names
1 N-(Phosphonomethyl) glycine
2 4-Amino-3,5,6-trichloro-2-pyridinecarboxylic acid
3 Alkyldimethylbenzyl-ammonium chloride

8 Glyphosate is the most widely used herbicide in the world, including in New Zealand.

Herbicide Risks

9 The following information on herbicide risks is based on a staff literature review. Staff are not offering expert advice in this area and have not consulted with toxicology or other experts in preparing the information for the Committee. Information from the staff literature review relevant to the Committee resolution, in chronological order, is as follows:

10 The World Health Organisation International Agency for Research on Cancer (IARC) classified glyphosate as "probably carcinogenic to humans" in 2015. The report states "this was based on

'limited' evidence of cancer in humans (from real-world exposures that actually occurred) and 'sufficient' evidence of cancer in experimental animals (from studies of 'pure' glyphosate)".

11 The New Zealand Environmental Protection Authority published a 'Review of the Evidence Relating to Glyphosate and Carcinogenicity' in August 2016. It concluded "glyphosate is unlikely to be genotoxic or carcinogenic to humans and does not require classification under HSNO [Hazardous Substances and New Organisms Act 1996] as a carcinogen or mutagen". Since the August 2016 publication, the EPA has not changed this position.

12 The toxicology risks of glyphosate came to prominence in 2018 and 2019 when American juries began awarding compensatory and punitive damages against Monsanto (the manufacturer of Roundup, which contains glyphosate) for failing to warn consumers that exposure to Roundup weed killer causes cancer. The cases are still in appeal by Monsanto.

13 Following the Monsanto lawsuits, a number of countries, states and districts across the world have either banned or attempted to restrict the use of glyphosate. A list of these can be reviewed via the link: https://www.wisnerbaum.com/toxic-tort-law/monsanto-roundup-lawsuit/where-is-glyphosate-banned-/.

14 In respect to Christchurch City Council's use of glyphosate, in 2020, University of Canterbury Professor of Toxicology Ian Shaw noted "for glyphosate to potentially cause cancer, which has been reported overseas, repeat doses over a long period of time would be needed and the risk of this was small". A paper by Professor Shaw on Roundup, published in the NZ Science Review Journal, is included as Attachment A.

15 The EPA has recently published (May 2022) a Glyphosate in Aotearoa New Zealand Summary Report following a call for information on the herbicide. The EPA Summary Report is a first step in the EPA's process to review the classification of the herbicide. The Summary Report can be reviewed via the link: https://www.epa.govt.nz/assets/Uploads/Documents/Hazardous-Substances/Glyphosate-call-for-information/Glyphosate-call-for-information-summary-report-may22.pdf

Herbicide Application

16 As noted above, glyphosate is the most widely used herbicide in the world. The EPA considers the product to be safe if applied in the accordance with regulations and guidelines.

17 Industry and DCC staff experience show herbicides to be the most effective method for controlling weed and vegetation growth.

18 All herbicide use by DCC contractors is in accordance with product Safety Data Sheets and New Zealand Environmental Protection Agency (EPA) regulations and guidelines.

19 Contractors prepare and adhere to Safe Operating Procedures (SOPs), which identify the controls and procedures for the safe application of herbicides. The SOPs refer to all relevant resources, including personal protective equipment to be used, appropriate application rates, and other procedures to be used by staff, which are based on regulations and industry best-practise. When using herbicides, DCC Contractors take a precautionary approach to manage the associated risks.

20 SOPs also manage 'spray-drift' risk ensuring that herbicide spraying only occurs in appropriate weather conditions to prevent contact with vegetation that should not be sprayed.

21 The New Zealand Agrichemical Education Trust (under the brand Growsafe) is a not-for-profit organisation promoting the safe, responsible and effective use of agrichemicals. Training and qualifications are provided through Growsafe. In addition to complying with EPA regulations and guidelines, DCC require its Contractors to be certified by Growsafe, and that all Growsafe standards are adhered to.

Alternative Methods

22 Alternative weed and vegetation control methods include:

a) Manual pulling of weeds
DCC contractors use this more labour-intensive alternative in garden areas and parks where spraying could affect desirable plants. This method is less cost effective where large areas of vegetation control are needed (such as road verges, surface water channels, edges, etc). Physical health risks from manual handling are a consideration with this method.

b) Mechanical removal using tractor attachments, weed eaters, chainsaws, mowers etc
Contractors routinely use these methods for vegetation control where the preference is to cut rather than kill. In comparison to spraying, a much greater frequency of treatment is required. These activities are used in amenity areas such as parks, road verges and sports fields where spraying (weed killing) is not a suitable control method.

c) Bark nuggets or wood mulch
This method used to prevent vegetation growth is already widely used in Parks and Recreation areas as soil coverage for gardens, and around trees.

d) Thermal means such as steam, hot water, electrochemical and fire
These methods have been trialled by DCC to control roadside vegetation but have proven to be less effective as the application frequency needs to be two to four times more often than herbicide sprays. The equipment also requires the carting of a heating system and the requirement to carry a large volume of water. Some of these methods can also kill worms and insects.

e) Organic herbicides
These are products that have organic and natural properties, that are used in the same way as chemical herbicides. These sprays can be made from pine oils, fatty acids, salt and vinegar. Some of the products such as salt or vinegar can alter soil PH and be harmful to insects. Some products have very strong odours. All products are proven by industry experience to be less effective than glyphosate products.

f) Alternative chemical herbicides
The NZ EPA 2016 report lists the seven frequently used herbicides after glyphosate. Based on the public feedback the EPA concluded “responders did not identify any one herbicide that is as safe, effective, and affordable as glyphosate and that could replace all its uses”.

23 DCC has undertaken trials of alternative weed control methods in the past, as have other local authorities, including Christchurch City Council. Staff are not aware of any reports that summarise the costs, effectiveness and environmental benefits of these trials. Discussions with Christchurch City Council staff, industry professionals and experience from DCC trials indicates that herbicides are the most cost effective method for controlling weed and vegetation growth.

NEXT STEPS

24 The New Zealand Environmental Protection Authority is the regulator of chemicals and hazardous substances in New Zealand. DCC and its contractors will continue to comply with all EPA regulations and guidance.

25 Council departments will continue to use herbicides, taking a precautionary approach to it's use, for weed and vegetation control.

Signatories

Author:	Ben Hogan - Transport Delivery Manager
Authoriser:	Jeanine Benson - Group Manager Transport Simon Drew - General Manager Infrastructure and Development

Attachments

	Title	Page
A	Is it time to round up Roundup?	

SUMMARY OF CONSIDERATIONS

Fit with purpose of Local Government

This decision enables democratic local decision making and action by, and on behalf of communities.

Fit with strategic framework

	Contributes	Detracts	Not applicable
Social Wellbeing Strategy	☐	☐	☒
Economic Development Strategy	☐	☐	☒
Environment Strategy	☐	☐	☒
Arts and Culture Strategy	☐	☐	☒
3 Waters Strategy	☐	☐	☒
Spatial Plan	☐	☐	☒
Integrated Transport Strategy	☐	☐	☒
Parks and Recreation Strategy	☐	☐	☒
Other strategic projects/policies/plans	☐	☐	☒

Herbicide is used in Council operational activities and does not contribute to DCC Strategies.

Māori Impact Statement

Staff have not discussed the use of herbicides in Council operations with Mana whenua.

Sustainability

Staff are not aware of any sustainability implications with the use of herbicides in Council operations.

LTP/Annual Plan / Financial Strategy /Infrastructure Strategy

There are no known implications of noting this report.

Financial considerations

There are no known implications of noting this report.

Significance

The report is considered low in terms of the Council's Significance and Engagement Policy.

Engagement – external

Staff discussed vegetation control experience with Christchurch City Council during the development of this report.

Engagement - internal

Parks and Recreation, 3-Waters, Property, Transport and Council contractors were consulted with in the development of this report.

Risks: Legal / Health and Safety etc.

The potential health risks of glyphosate, and their controls, are outlined in this report.

Conflict of Interest

There are no known conflicts of interest.

SUMMARY OF CONSIDERATIONS
Community Boards The use of herbicides and the effectiveness of vegetation control methods will be of interest to all parts of the city, including those areas covered by Community Boards.

Article

Is it time to round up Roundup®?
The changing science of glyphosate

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Background – glyphosates discovery, toxicity, and approval for marketing in 1974

Glyphosate (Fig. 1) is the active ingredient of the world's most commonly used herbicide (e.g., Roundup®). It was re-discovered by John Franz of Monsanto in the early-1970s (Dill *et al.*, 2010). Franz was investigating organophosphorus compounds and noted the plant toxicity of *N*-(phosphonomethyl)glycine which was later named 'glyphosate' by contraction of its chemical name. Franz's discovery was not the first time glyphosate had been studied; Henry Martin, a Swiss chemist, first synthesised glyphosate in 1950 and its synthesis was patented fourteen years later (USA Patent, 1964). The patent notes glyphosate's metal chelating properties. Strangely, Martin never published his work in the scientific literature, but his observations that glyphosate is a metal ion chelator (Fig. 2), including Ca^{2+}, Mg^{2+}, Cu^{2+}, Mn^{2+} and Zn^{2+} is the basis of one of our more recent environmental impact concerns (Mertens *et al.*, 2018) about glyphosate's extensive use in agriculture; this will be discussed later.

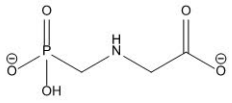

Figure 1. Glyphosate (*N*-(phosphonomethyl)glycine) showing its charges at biological pH.

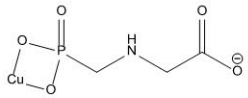

Figure 2 Possible structure of a Cu^{2+}-glyphosate chelate. Other divalent metals (e.g., Zn^{2+}, Mn^{2+}) might react similarly.

Studies on glyphosate's mechanism of herbicidal activity showed that it inhibits the shikimate pathway (Fig. 3; Steinrücken & Amrhein, 1980), a facet of biochemistry unique to plants. This was very encouraging because it suggested minimal, if any, animal toxicity. Glyphosate inhibits the shikimate pathway because of its structural analogy with phosphoenolpyruvate, a substrate for the key enzyme, 5-enolpyruvylshikimate-3-phosphate (EPSP) synthase (Fig. 4). Without the shikimate pathway, plants cannot biosynthesise aromatic amino acids and therefore die.

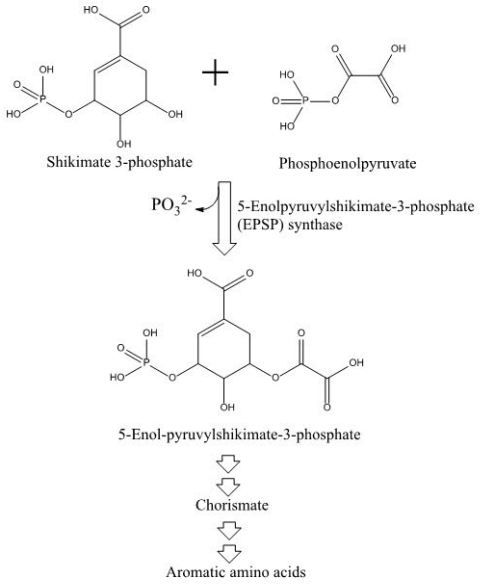

Figure 3. The shikimate pathway showing 5-enolpyruvylshikimate-3-phosphate (EPSP) synthase which is inhibited by glyphosate.

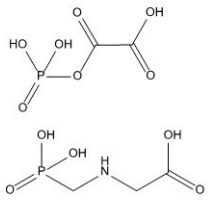

Figure 4. Phosphoenolpyruvate (top) and glyphosate (bottom) aligned to show their structural analogies, which is the basis of glyphosate's inhibition of EPSP synthase.

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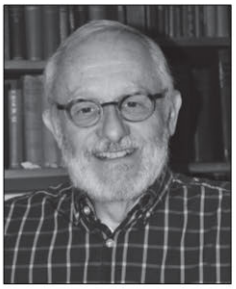

Ian Shaw is Professor of Toxicology at the University of Canterbury. Professor Shaw is an international expert on the impact of environmental contaminants on human health, particularly in a food safety context, and is a passionate communicator of science. He has had over 40 years' experience in academia, industry and government, both in the UK and New Zealand, which includes chairing the UK Pesticide Residues Committee and working as the National Food Safety Programme Manager at the Institute of Environmental Science and Research (ESR), based in Christchurch. He won the NZ Association of Scientists' Science Communicator's Award in 2009, and was awarded a DSc by the University of Bath for his work in toxicology and food safety in 2019.

There was pressure in the 1970s to support the growth of large-scale agriculture with the development of pesticides. The ideal was to introduce pesticides with minimal impact on non-target species (as, of course, it still is). Glyphosate fitted this goal perfectly because of its plant enzyme-based mechanism of action. In this respect it was, indeed, the Holy Grail of herbicides vis-à-vis its mechanism of action.

Further studies on glyphosate enhanced its Holy Grail status. Studies on its environmental fate and behaviour suggested that it was rapidly broken down in environmental systems (e.g., soil) to form a non-toxic degradation product, aminomethylphosphinic acid (AMPA; Fig. 5), which degraded further to form ammonia, carbon dioxide and water (Fig. 5). Its rate of disappearance (half-life, $t_{½}$) from terrestrial systems (e.g., soil) was shown to be very variable – from less than a week to years (Carlisle & Trevors, 1988); this disappearance was interpreted as degradation in the early days of glyphosate. However, it soon became clear that glyphosate's soil kinetics are biphasic. Studies with [^{14}C]-glyphosate in soils showed biphasic evolution of $^{14}CO_2$; this reflects a rapid initial degradation of free glyphosate, followed by slow degradation of soil-bound glyphosate. The time of the second degradation phase is dependent on soil type (i.e., is adsorption capacity) (Nomura & Hilton, 1977).

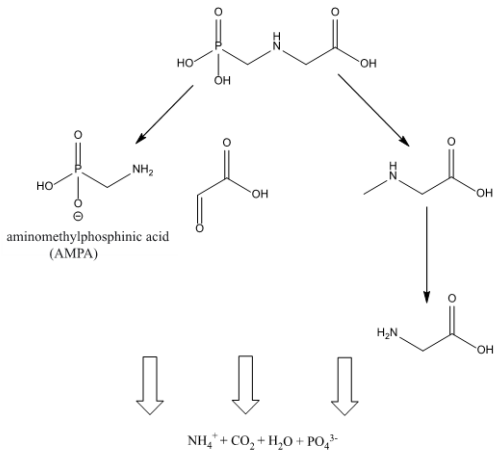

Figure 5. The environmental degradation of glyphosate showing its major degradation product, AMPA and its eventual complete degradation to ammonium, carbon dioxide, water and phosphate (based on Carlisle & Trevors, 1987).

Glyphosate, therefore, was hailed as a non-target non-toxic herbicide with a very short environmental residence time - ideal.

Food residues studies were carried out in a rather unconventional, but pragmatic way (Reding, no date). Instead of measuring glyphosate residues in crops to which the herbicide had been applied, the maximum residue levels (MRLs) for glyphosate in a large number of food crops were used as the worst-case consumption scenario. MRLs in conjunction with dietary intake data were used to calculate MRL-based worst case glyphosate intakes for each crop. Adding all of the individual crop glyphosate intakes together gives a total theoretical glyphosate intake of approx. 0.57 mg/person, which corresponds to a glyphosate dose of 0.0096 mg/kg body weight (bw) for a 60 kg human. At the time of Reding's calculations, glyphosate's acceptable daily intake (ADI) was 0.3 mg/kg bw, which means that the total theoretical glyphosate intake was approx. 3.2% of the ADI. This means that the health risk to consumers was deemed negligible.

Using current New Zealand data would give a much more favourable total glyphosate intake because the glyphosate MRL is currently (2021) set at a default value of 0.1 mg/kg, whereas Reding used MRLs ranging from 20 mg/kg for soybeans to 0.1 mg/kg for rice for her calculations. The current (2021) ADI for glyphosate is 1.0 mg/kg bw (FAO/WHO, 2006), which is higher than that used for Reding's calculations – this gives a glyphosate intake of 0.96% of the current ADI. If the default MRL of 0.1 mg/kg bw is used to estimate glyphosate intake, this would give a total theoretical intake of approx. 0.195 mg (= 0.00325 mg/kg bw for a 60 kg human) based on Reding's consumption data, which equates to approx. 0.33% of the current ADI. These calculations make the clear point that, based on conventional toxicological parameters, glyphosate intake as residues in food results in a negligible health risk.

Therefore, when glyphosate was approved for use in 1974 it appeared to be safe. This meant that the risk aspect of the risk-benefit equation was arguably negligible in both human and environmental contexts. The benefit side of the equation was considerable because glyphosate was and is a very effective herbicide. Thus, from an approvals-for-marketing perspective, glyphosate was close to ideal.

Use of glyphosate

When glyphosate was approved in the 1970s, it was indicated for general herbicide use. It was used by farmers to prepare land for crop planting without the necessity for tilling, which minimised soil erosion. Its use changed significantly after 1995 with the introduction of Roundup Ready® crops (Benbrook, 2016). Roundup Ready® crops (e.g., canola) are genetically modified to express a form of EPSP synthase from *Agrobacterium* strain CP4 that is resistant to glyphosate inhibition. This allows the herbicide to be used to kill weeds in a field of the Roundup Ready® growing crop. This has no direct relevance to New Zealand because genetically modified crops are not permitted in New Zealand. However, it might contribute to glyphosate food residues in imported products.

Glyphosate is also used as a crop desiccant to speed up the drying of near harvest crops (e.g., wheat) and facilitating an evenly dry, storable product (e.g., in Canada; Darwent *et al.*, 1994). This is common practice in New Zealand (FAR, 2017) and will lead to crop (and likely food) residues.

In recent years, glyphosate has been used to kill off pastures to facilitate their re-seeding or for follow-on planting with forage crops (e.g., brassicas). In order not to waste the dying pasture, stock are often grazed on the glyphosate-treated pasture (this will be discussed later). This might also lead to food residues.

Mammalian metabolism of glyphosate

The main human exposure route to glyphosate is via food. Farm workers might also be exposed dermally and via inhalation during mixing and applying sprays in an agricultural setting. Similarly, council workers and contractors might be exposed during spraying to control, for example, roadside weeds. Indeed, in a study of 48 farm families in the USA, glyphosate was detected (<1-233 ng/mL) in 60% of the urine samples analysed (Acquavella *et al.*, 2004).

Oral exposure leads to the ingested glyphosate being exposed to the gut microbiome, and this is very likely to lead to significant breakdown by a pathway akin to that for soil bacteria (Fig. 5). Based on animal studies, which appear to be reflected in humans, only approx. 30% of an oral glyphosate dose is absorbed from the gastrointestinal tract (GIT), peak plasma concentration is at approx. 1-2 h, and blood levels decline quickly due to urinary excretion rather than metabolism (Brewster *et al*., 1991; Bradberry *et al*., 2004). AMPA has been found in the blood of human glyphosate poisoning cases; this likely arose from gut microbial rather than human metabolism (Bradberry *et al*., 2004).

A study in glyphosate-exposed farm workers in the USA (Acquavella *et al*., 2004) clearly showed the presence of glyphosate in their urine. Urinary glyphosate in the exposed farm workers might be due to oral, dermal and/or inhalation absorption. Oral absorption is unlikely to result in significant blood concentrations (and thus urinary concentrations) of glyphosate (as discussed above). Studies in *in vitro* human skin preparations showed that <2% is absorbed, and dermal absorption studies in rhesus monkeys was approx. 0.8% of the applied dose (Wester *et al*., 1991). A study to assess the individual contributions of inhalation and dermal absorption following glyphosate exposure in humans showed that dermal absorption is greater than inhalation absorption (Pierce *et al*., 2020) and thus likely contributes more to blood (and urine) glyphosate concentration following workplace or bystander exposure (i.e., via sprays). It is, therefore, likely that the urinary glyphosate in the farmworker study was predominantly from dermal absorption of either aerosols (e.g., from spray) or direct skin contact (e.g., when diluting concentrate).

Glyphosate's $t_{½}$ in humans (using urinary excretion data) is in the range 5.5-10 h depending on the calculation method (Connolly *et al*., 2019). This means that, if a worker is repeat spraying over several days, there might be a build-up of glyphosate body burden because with a $t_{½}$ of 10 h only approx. two half-lives (i.e., 25% of body burden remaining) would have lapsed between exposures. However, as soon as exposure stops, it would take 6 half-lives (180 h, 7.5 d) to reduce the glyphosate body burden to approx. 1.2% of its peak value (likely of little or no toxicological significance for 'normal' agricultural exposures).

Glyphosate's human toxicity profile

Acute toxicity

The acute toxicity (i.e., following a single dose) of glyphosate is likely negligible in humans because it is not well absorbed (particularly from the GIT) and relatively quickly cleared ($t_{½}$ = 5.5-10 h). Use of appropriate personal protective equipped (PPE) will reduce the risk of acute effects significantly – particularly wearing impervious gloves to minimise dermal absorption (Acquavella *et al*., 2004). Cases of acute human poisoning have been recorded, but the doses involved are very large. In addition, commercial formulations of herbicides (e.g., Roundup®) containing glyphosate also contain excipients, including surfactants (e.g., polyethoxylated tallow amine - POEA) to aid absorption by plants and therefore it is often difficult to separate excipient toxicity from glyphosate toxicity *per se* (Bradberry *et al*., 2004) or to take account of the excipient's effects on glyphosate's toxicity (e.g., POEA might increase human absorption of glyphosate from the GIT or dermally). Also, the glyphosate salt used differs between glyphosate-containing commercial herbicides (e.g., in Roundup® glyphosate isopropylamine salt is used). All of these factors affect toxicity of the product. However, it has been shown that following suicide attempts, oral ingestion of 85 mL of Roundup® concentrate causes significant toxicity (but not necessarily death) in adults (Bradberry *et al*., 2004). Roundup® concentrate contains 41% w/v glyphosate isopropylamine salt (Fig. 6; molar mass = 346.4 g/mol), which means that 85 mL of Roundup® concentrate contains approx. 35 g glyphosate isopropylamine salt, which equates to approx. 17 g glyphosate (molar mass = 169.1 g/mol); therefore, a glyphosate oral dose of approx. 300 mg/kg body weight (using standard human weight = 60 kg) is near fatal in humans. The estimated lethal dose of aspirin in humans is 5-15 g (Clarke, 1978; ~ 83-250 mg/kg body weight), which means that glyphosate is of the same order of acute oral toxicity as aspirin.

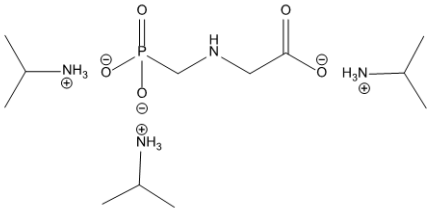

Figure 6. Depiction of glyphosate isopropylamine salt

The symptoms of acute Roundup® toxicity in humans are increased saliva production, burns in the mouth and throat, nausea, vomiting and/or diarrhoea. It is difficult to assign these signs of toxicity to glyphosate *per se* because some (e.g., burns in the mouth and throat) might be caused by POEA. The salivation response is, however, interesting because it is associated with organophosphate (OP) intoxication due to OPs inhibition of acetylcholinesterase (AChE). Glyphosate is also a simple OP and thus might exhibit AChE inhibitory properties – in this context, it is interesting that glyphosate has been shown to inhibit AChE in carp (*Cyprinus carpio*) (Gholami-Seyedkolaei *et al*., 2013).

Chronic toxicity

On 20 March 2014, the World Health Organization's International Agency for Research on Cancer (IARC) classified glyphosate as Carcinogen 2A (probably carcinogenic to humans). This led to action by several countries (e.g., The Netherlands banned glyphosate in 2015; In Habitat, 2014) and a recent partial ban by France (Euro Coop, 2021). There followed significant international debate and conjecture at government level about the validity of the IARC's carcinogen classification, and whether the significant economic benefit of glyphosate outweighed its risks (especially as some jurisdictions disputed the IARC's deliberations and conclusion). The New Zealand Government commissioned a report to assess the IARC's findings (Temple, 2016) which dismissed the IARC's carcinogenicity data as inconsistent and showing a lack of association, in part due to the possibility that subjects in the studies might have been exposed to other pesticides. Temple (2016), however, missed a key mechanistic possibility in his report: that glyphosate might be a non-genotoxic carcinogen; this is relevant in the context of glyphosate's estrogen mimicry (see later).

The evidence that the IARC presented in support of their categorisation of glyphosate as Carcinogen 2A included three key

studies: (1) A study in farmworkers which showed an association between farmworkers' glyphosate exposure and non-Hodgkin's lymphoma and multiple myeloma (Acquavella *et al*., 2004); (2) A chronic exposure study in mice which showed a dose-related relationship with skin cancers (George *et al*., 2010); (3) A study in cultured human estrogen receptor (ER)-expressing breast cancer cells which showed a glyphosate dose-related increase in proliferation that was inhibited by the potent ER antagonist, fulvestrant (Thongprakaisang *et al*., 2013). The last-named study is particularly important because it initiated the thinking that glyphosate might be an estrogen mimic (see later).

A later expert panel review of the carcinogenicity data used in support of the IARC's ruling pointed out limitations in some of the studies used by the IARC. In particular, they found that the association between non-Hodgkin's lymphoma and glyphosate exposure might have been confounded by multiple pesticide exposures (as pointed out by Temple (2016)) and so was unreliable (Acquavella *et al*., 2016). Several years' later, a meta-analysis of glyphosate exposure-linked non-Hodgkin's lymphoma concluded confidently that there is indeed a link (Zhang *et al*., 2019). Further to this, a recent and extensive meta-analysis of human (occupational) exposure to glyphosate and the incidence of non-Hodgkin's lymphoma and multiple myeloma showed no increased risk except possibly at very high glyphosate exposure (Donato *et al*., 2020). This conjecture makes it difficult to conclude whether or not glyphosate is carcinogenic.

The molecular structure of glyphosate (Fig. 1) has no features that would point to genotoxic carcinogenicity (e.g., reactive moieties that might alkylate DNA leading to mutations) and therefore Temple's (2016) conclusion that it is not a genotoxic carcinogen is justified on structure activity grounds alone. However, mounting evidence that glyphosate interacts agonistically with ERs is good evidence for a non-genotoxic mechanism of carcinogenesis. Non-genotoxic carcinogens affect cells in such a way that they induce proliferation (e.g., an inflammatory response) that increases the chance of a transcriptional defect which leads to a carcinogenic event (Shaw & Jones, 1994). In addition, receptor-mediated tumours (e.g., ER+ breast cancer) proliferate in response to their receptor (e.g., ER) agonist (e.g., the estrogen, 17β-estradiol (E2)) and therefore natural receptor ligand mimics (e.g., estrogen mimics) might act as non-genotoxic carcinogens via this mechanism (Ye *et al*., 2018). It is interesting to note that in animal models, administration of E2 causes proliferation of ER-expressing lymphoid and myeloid lineage bone marrow cells (Issa *et al*., 1996) – this has a possible link to non-Hodgkin's lymphoma via a non-genotoxic hormone-mediated mechanism. If glyphosate is an estrogen mimic it might initiate this response.

Is glyphosate estrogenic?

There has been considerable conjecture about whether or not glyphosate is estrogenic, since the first experiments in MCF-7 cells showed that proliferation stimulated in a dose-dependent manner by glyphosate was inhibited by the potent ER antagonist, fulvestrant (Thongprakaisang *et al*., 2013). This conjecture was largely because the molecular structure of glyphosate has no apparent structural relationship to ER's natural ligand, E2, whereas most estrogen mimics have significant molecular analogies with E2 that allow them to interact with key amino acid residues in the ER and thus initiate an estrogen-like response (Fig. 7).

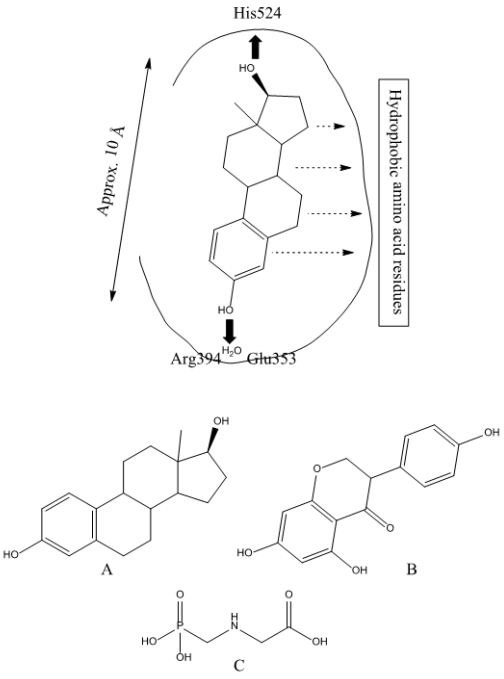

Figure 7. Top: Schematic representation of ERα with E2 *in situ* showing its key interactions with amino acid residues that lead to biological activity.

Bottom: E2 (A), the known estrogen mimic, genistein (B), and glyphosate (C), are also shown to illustrate glyphosate's lack of molecular analogy with E2.

To initiate an estrogen response a ligand must ideally possess two hydroxyl groups – one aromatic and one aliphatic – separated by approximately 10 Å of hydrophobicity. The hydroxyls form hydrogen bonds with the Arg394/Glu353/water triumvirate at one end of the ligand binding cleft (LBC) and with His524 at the other (in ERα, for ERβ the amino acid residue interactions are the same, but because of differences in the amino acid sequence the residue numbers are different; i.e., Glu306/Arg346/water and His475 (Pike *et al*., 1999)). There are a significant number of hydrophobic interactions with a cluster of hydrophobic amino acid residues aligned with the steroid skeleton of E2 *in situ*. Estrogen mimics (e.g., genistein – an isoflavone from soybeans) have the appropriate structural attributes, albeit often not ideal (e.g., genistein has two aromatic hydroxyl groups) to interact with the amino acid residues in the LBC. These interactions lead to a receptor conformational change, which in turn leads to the formation of a receptor/ligand dimer with increased affinity for an estrogen responsive element (ERE) on DNA. When bound to the ERE the ER-ligand dimer upregulates key genes which result in the biological response (Ye *et al*., 2018). The fact that glyphosate does not have the key ER-binding molecular attributes makes it unlikely on structure activity relationship (SAR) grounds to be estrogenic by a 'conventional' estrogen mimicry mechanism.

Despite the controversy following Thongprakaisang *et al.* (2013)'s findings, Mesnage *et al.* (2017) showed that glyphosate was indeed estrogenic, but by a ligand-independent activation mechanism leading to ERE-luc expression via protein kinase A (PKA) signalling. This brought the spotlight back onto glyphosate and the importance of its estrogenicity in both a human exposure and environmental context. Further work in ERα-expressing cholangiocarcinoma cells showed that a non-genomic ER pathway via extracellular signal-regulated kinases (ERK) 1 and 2 might explain glyphosate's estrogenic response (Sritana *et al.*, 2018). Interestingly, ERK1 and ERK2 stimulate cell proliferation which might also link glyphosate to a non-genotoxic mechanism of carcinogenesis as speculated above. It is therefore likely that glyphosate is estrogenic, perhaps by a non-genomic mechanism that does not rely on E2/glyphosate SARs and ER interactions.

Environmental toxicology

Glyphosate has an apparently short (<1 week) to long (years) soil $t_{½}$, which is due to an interplay between its binding to soil particles (sequestration) and its conversion (e.g., by soil bacteria) to AMPA (then CO_2, NH_3 and H_20) (Carlisle & Trevors, 1987). Sequestration is possibly reversible when environmental conditions change, and it is possible that, while bound to soil particles, glyphosate is still bioavailable in some circumstances. Glass (1987) reported that glyphosate binds to clay soils, possibly by an ion exchange mechanism. He noted that binding was 'stronger' in the presence of divalent metal ions (M^{2+}; e.g., Mg^{2+}, Ca^{2+}) which supports an ion exchange mechanism involving binding of M^{2+} to the negatively charged clay particles giving an overall +1 charge to which negatively charged glyphosate (Fig. 1) can bind. An increase in soil pH (i.e., greater OH^- concentration) could displace glyphosate from its clay-bound complex because OH^- would likely compete with glyphosate for positively charged regions of the clay-M^{2+} complex. These possibilities are rarely discussed in a regulatory context (Mertens *et al.*, 2018). It is, however, clear that a short soil $t_{½}$ that was originally interpreted as short-lived environmental impact is not necessarily the case. Interestingly, as far back as 1976, it was noted that the soil $t_{½}$ of glyphosate was composed of two phases; an initial rapid soil bacteria-mediated biotransformation of free glyphosate and a slower biodegradation of soil-bound glyphosate (Hance, 1976).

Studies in the planktonic crustacean, *Daphnia magna* have shown that exposure to glyphosate-Cu^{2+} complexes alter the *Daphnia*'s behaviour, indicating that glyphosate might mediate metal toxicity in ecosystems (Hansen & Roslev, 2016) – this introduces a new mechanism of glyphosate's impact on ecosystems that does not rely on its direct toxicity, but rather mediated toxicity. This could extend beyond Cu^{2+} to other divalent metal ions (e.g., Pb^{2+}). Similarly, glyphosate's chelating properties can affect metal sorption to soils: Morillo *et al.* (2002) studied Cu^{2+} adsorption in three soil types and found that sorption differed with soil type due to the equilibrium between soil-Cu^{2+} and glyphosate-Cu^{2+}. This means that the presence of glyphosate in terrestrial (and perhaps aquatic silt) systems might alter the balance between adsorbed and aqueous metals, and change the bioavailability of these metals to organisms in the ecosystem. The bioavailability of toxic metals and 'nutrient' metals is important in ecosystem health. If glyphosate upsets this balance it will perturb ecosystem health (Mertens *et al.*, 2018).

There has been a great deal of work exploring the ecotoxicity of glyphosate and its formulations (including Roundup®) that shows a plethora of impacts on individual test species (Carlisle & Trevors, 1988). It is important to note that it is often difficult to separate the effects of glyphosate *per se* and other constituents (e.g., surfactants) of glyphosate herbicide formulations (Carlisle & Trevors, 1988) and that in some cases the excipients are more toxic than glyphosate *per se* (Peréz *et al.*, 2011). Obviously, glyphosate is very toxic to plants (approx. 18μM (approx. 3 mg/L) glyphosate inhibits growth of the green microalga, *Chlorella sorokiniana*, by 50% (Christy *et al.*, 1981)). Similarly, growth of the single-celled alga, *Euglena gracilis*, a mixotroph (capable of living photosynthetically or by phagocytosis), is impacted by 1.3 mM (approx. 219 mg/L) glyphosate (Richardson *et al.*, 1979). From this very cursory foray into the literature, it is clear that low environmental concentrations of glyphosate have been known to impact non-target plants from the time that glyphosate was first introduced to the market in the 1970s, but this is hardly surprising for a herbicide!

Glyphosate's impact on animals is quite a different matter. Since glyphosate was thought to be ostensibly non-toxic to animals because of its plant-focused mechanism of toxicity, little work appears to have been conducted on animals in an ecological context until the 1980s. An early study of Roundup® in *D. magna* gave an EC_{50} (48 h, immobilisation) of 3.0 mg/L and EC_{50} (48 h, mortality) in the amphipod crustacean, *Gammarus pseudolimnaeus* of 62.0 mg/L (Folmar *et al.*, 1979). Since these studies used glyphosate in its Roundup® formulation, it is important to consider the toxicity of its major surfactant constituent, POEA. A study of the toxicity of POEA in *D. pulex* gave an EC_{50} (96 h, immobilisation) of 2.0 mg/L, while Roundup® gave an EC_{50} (96 h, immobilisation) of 8.5 mg/L (Servizi *et al.*, 1987). This suggests that POEA is largely responsible for Roundup®'s toxicity in this *Daphnia* study. This is a very important consideration when assessing the environmental toxicity of Roundup®. However, it is equally important to consider the fate and behaviour of POEA in environmental systems and the differential exposures of creatures to glyphosate and/or POEA following the use of Roundup® in an agricultural setting, but this is beyond the scope of this paper.

Pérez *et al.* (2011) extensively reviewed the invertebrate toxicity of glyphosate-containing commercial formulations (including Roundup®). They collated EC_{50}s for variable endpoints (e.g., immobilisation, mortality) and exposure times (48 h or 96 h) and found EC_{50}s in the range 3.0 mg/L (*D. magna*, 48 h, immobilisation) – 415.0 mg/L (*Ceriodaphnia dubia*, 48 h, mortality). This gives an idea of the level of environmental toxicity of commercial glyphosate products, but of course, includes the toxicity of excipients such as POEA. It is also important to note that invertebrates do not express ERs (Brennan *et al.*, 2006) and therefore are not susceptible to glyphosate's estrogenicity.

In general, the toxicity of glyphosate *per se* to higher animals (e.g., fish) is not greater than its toxicity to invertebrates (Table 1) which suggests that its estrogenicity is not a major determinant of toxicity in the short term (96 h). Interestingly, glyphosate commercial formulations (mean 96 h LC_{50} = 15.9 mg/L) are often very much more toxic than glyphosate *per se* (mean 96 h LC_{50} = 246.8 mg/L) (Table 1); this makes the point that the formulation excipients are a major determinant of toxicity. Indeed, fish POEA toxicity studies show this clearly (e.g., *O. mykiss* 96 h LC_{50} for POEA = 2.0 mg/L (Folmar *et al.*, 1979)).

Table 1. Toxicity (96 h) of glyphosate and Roundup® to fish.

Species	LC_{50} mg/L	Reference
Glyphosate *per se*		
Rainbow trout (*Oncorhynchus mykiss*)	140.0	Folmar *et al*., 1979
Fathead minnow (*Pimpehales promelas*)	97.0	Folmar *et al*., 1979
Channel catfish (*Ictalurus punctatus*)	130.0	Folmar *et al*., 1979
Common carp (*Cyprinus carpio*)	620.0	Neškovic *et al*., 1996
Mean	246.8	
Roundup®		
Oncorhynchus mykiss	8.3	Folmar *et al*., 1979
	52.0	Hildebrand *et al*., 1982
	8.5	Servizi *et al*., 1987
Pimpehales promelas	2.3	Folmar *et al*., 1979
Ictalurus punctatus	13.0	Folmar *et al*., 1979
	14.5	Abdelghani *et al*., 1997
Bluegill sunfish (*Lepomis macrochirus*)	13.0	Abdelghani *et al*., 1997
Mean	15.9	

Therefore, even in fish, glyphosate is less toxic than at least one of its excipients (POEA).

Glyphosate's chronic toxicity is less well documented in fish. However, Salbego *et al.* (2010) reported decreased weight and AChE activity in *Leporinus obtusidens* exposed to 1 mg/L Roundup® for 90 d. In addition, a study in which *Platichthys flesus* was exposed to Roundup® + AMPA for 62 d showed liver damage at a glyphosate exposure concentration of 0.16 µg/L (Evrard *et al*., 2010). These studies suffer from the problem of excipient toxicity, but since glyphosate is an OP, it likely caused the decreased AChE activity.

Studies in amphibians show similar, if not more extreme, toxicity differentials between Roundup® and glyphosate *per se* (Pérez *et al*., 2011). For example, the 48 h LC_{50} for glyphosate in *Lymnodynastes dorsalis* is >400 mg/L, while the corresponding value for Roundup® is 3.0 mg/L, and for *Heleioporus eyrie* the corresponding values are >373 mg/L and 6.3 mg/L respectively (Mann & Bidwell, 1999). Clearly, not only are Roundup®'s excipients more toxic than glyphosate *per se*, they might even ameliorate glyphosate's toxicity. A single long-term (42 d) study in *Rana cascadae* shows the first inkling of a possible hormone effect – earlier metamorphosis at an exposure concentration of 1.94 mg/L (Cauble & Wagner, 2005), but this is far from conclusive.

Therefore, glyphosate *per se* is far less toxic to vertebrates than Roundup®. This is likely due to POEA as evidenced by its 96 h LC_{50} in *Xenopus laevis* of 2.7 mg/L (Perkins *et al*., 2000).

A long-term study in *Carassius auratus* exposed to 0.2 mmol/L glyphosate as a commercial formulation (Nongteshi®) for 90 d showed significant metabolomic changes, including raised aspartate aminotransferase (AST) – a marker of liver and muscle damage, lactate dehydrogenase (LDH) – a marker of general cell damage, and alanine aminotransferase (ALT) – a marker of liver damage (Li *et al*., 2017). This study indicates a wide-ranging generalised impact on the fish biochemistry. Unfortunately, no hormone-related parameters were measured. As with so many commercial formulation studies, it is impossible to determine whether the effects were due to glyphosate or excipients; indeed, POEA could be responsible for all of the observed effects. These naïve study designs give very little information of values when attempting to assess environmental impact because, even though commercial formulations are used in agriculture, it is impossible to determine the fate, behaviour and impact of the individual components, especially when (in the laboratory) the formulation is applied to a closed environment test system which does not allow for differential distribution of the formulation's components.

In order to fully assess the environmental impact of glyphosate, we need good, reliable long-term studies because it is clear that from an acute toxicity standpoint, glyphosate is of little concern (i.e., the risk is low). It has been noted that there is a dearth of long-term glyphosate environmental toxicity data (Howe *et al*., 2004); this situation remains unchanged today (2021). The need for long-term toxicity data is because our knowledge of glyphosate's effects or potential effects in animals points firmly to long-term impact. For example, glyphosate's estrogenicity would not manifest in the short term, but possibly in a multigenerational growth and development context. Interestingly, one of the few long-term studies (in frogs) shows a multitude of effects, most of which might be attributable to POEA. However, there is one very interesting finding: glyphosate causes an increase in the female:male ratio and this effect is not seen for POEA alone. The sex ratio change was also seen following Transorb® exposure. Transorb® is a commercial glyphosate formulation containing POEA and glyphosate as its potassium salt (Roundup® contains glyphosate isopropylamine salt; Fig. 6). This needs further scrutiny because exposure to estrogenic compounds suppresses male development in some species (e.g., fish; Jobling *et al*., 1998).

Glyphosate concentration in the environment

In order to assess the potential environmental impact of glyphosate's use, environmental glyphosate concentrations are compared with toxicological effect parameters (e.g., EC_{50}, LC_{50}). I could find no published data on glyphosate concentrations in New Zealand soils or waterways. However, studies in Argentina found 35 – 1502 µg/kg soil (mean of samples with measurable concentrations = 340 µg/kg); the low value in the range was in a soil sample taken 40 d after spraying, and the high value 1 d after spraying – the difference reflects glyphosate's soil $t_{½}$; they also found silt-bound glyphosate in approx. 15% of water samples analysed, with 'free' water concentrations in the range 0.4 – 7.6 µg/L (mean of samples with measurable concentrations = 2 µg/L) (Aparicio *et al*., 2013). It is interesting that silt-bound glyphosate was found in waterways because this suggests that transfer from land can be via silt (Aparicio *et al*., 2013). Environmental concentrations in New Zealand would be expected to be lower than in Argentina because Roundup Ready® crops are not permitted here, but are used extensively in Argentina.

Using the mean values for soil and water glyphosate concentrations from the Argentinian study, the mean water concentration of 2 µg/L is far below all of the *Daphnia* short-term (48 – 96 h) exposure, immobilisation endpoint EC_{50} values collated by Pérez *et al*. (2011): range 3.0 – 66.2 mg/L; even the top value of 7.6 µg/L is only approx. 0.25% of the most sensitive test result. This suggests that, in conventional toxicological terms, the agricultural use of glyphosate would have little or no short-term environmental impact, even following intensive use in a Roundup Ready® agricultural setting. Assessing the impact of terrestrial animal exposure via soil is difficult because I could find only one published study on terrestrial invertebrate toxicity. This study was carried out in earthworms (*Eisenia foetida*) at very high exposure concentrations (10 - 1,000 mg/kg soil) (Correia & Moreira, 2010) and so is irrelevant to predicted glyphosate soil

Figure 8. A glyphosate-treated field with grazing cattle in the Canterbury region.
(Photograph by the author.)

concentrations following glyphosate-containing herbicide use. LD_{50} studies in terrestrial vertebrates show extremely low oral toxicity; e.g., LD_{50} in deer mouse (*Peromyscus maniculatus*) >6,000 mg/kg body weight. The corresponding value for the rough-skinned newt (*Taricha granulosa*) is >2,600 mg/kg body weight (McComb *et al.* 2008). It is difficult to equate gavage LD_{50} study doses with terrestrial environmental exposure; therefore, I have used data from two silt-living aquatic/marshland species, the black worm (*Lumbriculus variegatus*) and the buzzer midge (*Chironomus plumosus*) larva in lieu of bona fide terrestrial invertebrates for this assessment. The 48 h EC_{50} for glyphosate per se in *C. plumosus* (immobilisation endpoint) is 55 mg/L and the corresponding value for *L. variegatus* (glutathione S-transferase activity (GST)) is 0.05 mg/L (Pérez *et al.* 2011). These values are both far below the equivalent terrestrial glyphosate concentrations from the Argentinian study, even with the very sensitive GST toxicity endpoint in the *L. variegatus* study. Thus, the likely short-term impact of glyphosate on the terrestrial environment is negligible.

This clean bill of health for glyphosate following its agricultural use is, however, misleading because it reflects only short-term impact. There are no data on long-term effects – glyphosate's estrogenicity will only manifest after long-term, multigenerational exposure.

Glyphosate residues in food

There is growing interest in residues of glyphosate in food (perhaps following the IARC's categorisation of glyphosate as Carcinogen 2A). The widespread use of glyphosate in agriculture means that residues in food are inevitable. In New Zealand, genetically modified crops are not permitted and therefore direct application of glyphosate to growing crops does not occur, except for its use as a desiccant to aid the even ripening and drying of, for example, wheat – this reduces the risk of widespread high crop residue levels that might occur overseas. However, glyphosate is used extensively in non-till regimes to prepare land for new crops, including livestock forage and grass. In New Zealand it is common to see livestock grazing recently sprayed pastures to maximise the efficient use of 'carbon' (Fig. 8). There is no Roundup® withholding period for stock grazing in New Zealand (unless Roundup® is being used to kill toxic plants; e.g., ragwort (*Jacobaea vulgaris*)); therefore, there is no safeguard to reduce residues in meat and milk following livestock's consumption of glyphosate-treated pasture. Since glyphosate is rapidly metabolised and excreted in mammals, meat residues are unlikely to be a problem; however, if glyphosate is secreted into milk there are no metabolic enzymes present, and so the residues might remain.

The New Zealand Ministry of Primary Industries (MPI) carried out surveys on glyphosate residues in honey in 2017/2018 and 2018/2019 and released a report bringing together the results in response to industry concerns in January 2020 (MPI, 2020a). In the 2017/2018 survey, 1.7% were above the default maximum residues limit (MRL) of 0.1 mg/kg, and 20.7% had measurable (i.e., above the limit of determination of the analytical method) glyphosate residues. In the 2018/2019 survey, 18.3% of samples had measurable glyphosate, but none exceeded the default MRL (MPI, 2020b). From a food safety perspective, the glyphosate residues found in New Zealand honey are of little concern, even considering glyphosate's estrogenicity because honey is not a 'major' food and so the glyphosate dietary intake from honey would be low. The question is, where did the glyphosate come from? Clover, pasture and multifloral honeys had the greatest proportion of residues in both surveys. This suggests that bees were accessing glyphosate-sprayed pastures prior to the flowers dying. A related problem occurred in the UK in the 1990s where treatment of rape with insecticides was associated with bee deaths – advising farmers not to spray flowering rape solved the problem (personal information; the author was chairman of the UK Pesticide Residues Committee 1992–2000). An additional possible source of New Zealand's honey glyphosate contamination might be the use of Roundup® to kill herbage around beehives.

Glyphosate residues in honey have been found in many other countries, with concentrations as high as 160 mg/kg in USA honey (Rubio *et al.*, 2014) – this might reflect the use of Roundup Ready® flowering crops which, even though highly contaminated with glyphosate, are not killed and so continue to attract and contaminate bees.

In addition, glyphosate residues in other foods have been reported from around the world. For example, residues in 9/28 (32%) samples of soy sauce in a USA survey had glyphosate residues >100 mg/L (one sample exceeded 500 mg/L) (Rubio *et al.*, 2014). The health risks associated with this are likely to be minimal via western diets, but would be greater for some Asian communities. A Swiss study found that glyphosate residues were very often found in fruits, wine and honey, but that pasta was the most important source of glyphosate residues in a dietary intake context, but they found no MRL exceedances (Zoller *et al.*, 2017).

It is clear that the extensive use of glyphosate in agriculture is reflected in its food residues spectrum. While the health risk from glyphosate intake from individual commodities might

be low, since residues are widely distributed it is important to consider total dietary intakes. Zoller *et al.* (2017) assessed dietary intake and found that neither the ADI nor the acute reference dose (ARfD) for glyphosate were exceeded. They concluded that glyphosate residues are of no concern from a human health perspective.

It is important to note that all of the toxicological endpoints used to determine the ADI and ARfD are unlikely to have included endpoints (e.g., testicular atrophy, endometrial thickening) that would have indicated estrogenicity. They were almost certainly based on acute toxicology. This is important because glyphosate has a 'clean' acute toxicity profile which might be misleading in the context of new evidence regarding its estrogenicity and long-term risk.

In the context of food residues, Low *et al.* (2005) demonstrated that *Saccharomyces cerevisiae* (the yeast used in breadmaking) can metabolise glyphosate, thus reducing potential residues in fermented food products (e.g., bread) made from, for example, flour with glyphosate residues. This is less important in New Zealand where Roundup Ready® wheat is not grown, but is an interesting concept that should be borne in mind when predicting food residues and their health risks.

Continuing the idea that glyphosate is estrogenic and that this might have implications in a long-term risk context, it is important to consider multigenerational effects, in particular growth and development effects on the embryo and fetus (Shaw *et al.*, 2009). Aris & Leblanc (2011) measured glyphosate in serum from, non-pregnant and pregnant women and their fetuses (fetal cord blood). They did not find glyphosate or AMPA in serum from pregnant women (n = 30) or fetal cord blood (n = 30), but did find glyphosate in serum (mean SD = 73.6 28.2 ng/mL) of 2/39 (5%) of the non-pregnant women (n = 39) studied. Surprisingly, AMPA was not found in the two glyphosate-positive non-pregnant women's samples; the authors suggest that this might be for technical reasons or because AMPA is rapidly cleared.

A very misleading study in rats showed that glyphosate is teratogenic at huge maternal doses (500 – 1,000 mg/kg/d) (Dallegrave *et al.*, 2003); this is likely to be due to maternal toxicity causing retardation of fetal growth, and so has little or no relevance to potential effects of low-level glyphosate exposure to pregnant women. A follow-on rat study by Dallegrave *et al.* (2007) using lower (but still irrelevant to human exposure) maternal oral glyphosate doses showed that at doses ≥50 mg/kg body weight there was a dose-related reduction in sperm number in offspring 65 d *post-partum*, but that the values returned to normal by 140 days *post-partum*. In addition, serum testosterone decreased in a dose-dependent manner at glyphosate doses ≥50 mg/kg body weight on day 65 *post-partum*, but this had only partly recovered on day 140 *post-partum*. Even though the doses used are irrelevant to human exposure, the effects on sperm number and serum testosterone point to an estrogenic response. Unfortunately, Dallegrave and her colleagues used Roundup® throughout their studies (Dallegrave *et al.*, 2003; Dallegrave *et al.*, 2007) and so POEA was co-administered to the rats. However, Mesnage *et al.* (2017) convincingly proved that POEA is not estrogenic and so the Dallegrave *et al.* (2007) findings are very likely due to glyphosate.

Current use of Roundup® in New Zealand and its impacts on human health, livestock, and ecosystems

Our knowledge about the chemistry and toxicology of glyphosate has increased considerably since it was licensed 47-years ago. We now know that its short soil $t_{½}$ might be partly due to its binding to charged soil components and that it might be released at a later time. In addition, there is mounting evidence that glyphosate is estrogenic, albeit with low relative (to E2) estrogenicity. The implications of this to environmental impact and human health have hardly been discussed because of the conjecture about the initial studies indicating estrogenicity. One thing is certain, glyphosate as an environmental estrogen will have effects at exposure concentrations far below those associated with its conventional toxicology.

Our understanding of glyphosate's short-term toxicity to humans, animals and ecosystems has not changed; glyphosate remains of little toxicological concern following acute exposure, except at very high doses. It is rapidly broken down by soil bacteria and metabolised and excreted in animals (including humans), which means that short-term environmental contamination or human exposure is unlikely to lead to long-term adverse effects.

Glyphosate's long-term impact is very much less well understood. Its estrogenic effects are likely only to be manifest following long-term exposure. As its use patterns have changed over the years, and its use has increased considerably on account of its importance in agriculture, it is appearing as residues in crops and food. In most cases, residue levels are low, but the effects of multiple exposures to multiple residues in multiple foods on human health have not been adequately assessed. This has particular resonance in the context of estrogenicity, which can cause subtle changes in growth and development following very long-term, very low-level exposures.

An issue that appears not to have been considered is the effect of stock grazing glyphosate-sprayed paddocks. It is possible that this route of exposure results in high glyphosate doses because there is no statutory grazing withdrawal time. High doses of estrogenic compounds can have effects on reproductive health, which might affect ovulation, sperm health and thus fecundity. This could have economic impacts in an agricultural setting. Similarly wild animals (e.g., birds) foraging in recently glyphosate sprayed will be exposed to high levels of the chemical which could affect their ovulation cycles and thus fecundity.

It is important that we do not simply accept the longstanding dogma that glyphosate is safe; we must question this, consider our increasing understanding of glyphosate's interactions with biological systems, explore its long-term effects, and modify our use of this vitally important agrochemical accordingly. As Jacob Bronowski noted in the *Ascent of Man*, They [We] are here not to worship what is known, but to question it… (Bronowski, 1973).

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