

WASHINGTON REPORT

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Federal Government Funded on CR Until Dec. 9.

A day before FY 2016 government funding expired on September 29, Congress passed a continuing resolution (CR), H.R. 5325 that extends government funding FY 2016 levels until Dec. 9, when lawmakers are expected to be in Washington for a lame-duck session after the election. The CR was cleared by the House on a 342-85 vote and earlier in the day was passed by the Senate, 72-26. The legislation includes \$1.1 billion in Zika response funding, \$500 million for flood relief in Louisiana and other states and fiscal 2017 appropriations for military construction and veterans. The initial conference report back in July contained language that would have provided mosquito sprayers, including vector control districts, a 180 day waiver from NPDES permit requirements for applying FIFRA approved insecticides near waters of the United States. Unfortunately, that language was eventually removed due to the objections from a handful of Senators and Representatives who were loaded up with misinformation and fear-mongering from environmental extremist groups.

MIT Researchers Find New Way to Make Pesticides Stick to Leaves 10 Times Better?

Massachusetts Institute of Technology. "Making pesticide droplets less bouncy could cut agricultural runoff: Researchers find a way to make pesticides stick to leaves instead of bouncing off." ScienceDaily, 30 Aug 2016. www.sciencedaily.com/releases/2016/08/160830121724.htm

By using a clever combination of two inexpensive polymer additives, called polyelectrolytes, MIT researchers found they could drastically cut down on the amount of liquid that bounces off plants. The new approach uses two different kinds of additives. The spray is divided into two portions, each receiving a different polymer substance. One gives the solution a negative electric charge; the other causes a positive charge. When two of the oppositely-charged droplets meet on a leaf surface, they form a hydrophilic (water attracting) "defect" that sticks to the surface and increases the retention of further droplets.

Based on the laboratory tests, the team estimates that the new system could allow farmers to get the same effects by using only 1/10 as much of the pesticide or other spray. And the polymer additives themselves are natural and biodegradable, so will not contribute to the runoff pollution.

Journal Reference: Maher Damak, Seyed Reza Mahmoudi, Md Nasim Hyder, Kripa K. Varanasi. **Enhancing droplet deposition through in-situ precipitation.** *Nature Communications*, 2016; 7: 12560 DOI: [10.1038/ncomms12560](https://doi.org/10.1038/ncomms12560)

Abstract: Retention of agricultural sprays on plant surfaces is an important challenge. Bouncing of sprayed pesticide droplets from leaves is a major source of soil and groundwater pollution and pesticide overuse. Here we report a method to increase droplet deposition through in-situ formation of hydrophilic surface defects that can arrest droplets during impact. Defects are created by simultaneously spraying oppositely charged polyelectrolytes that induce surface precipitation when two droplets come into contact. Using high-speed imaging, we study the coupled dynamics of drop impact and surface precipitate formation. We develop a physical model to estimate the energy dissipation by the defects and predict the transition from bouncing to sticking. We demonstrate macroscopic enhancements in spray retention and surface coverage

for natural and synthetic non-wetting surfaces and provide insights into designing effective agricultural sprays.

EPA says Glyphosate “Not Likely to be Carcinogenic to Humans”

In September, EPA officially released its cancer review assessment and background paper on glyphosate along with more than 100 other documents that will be the focus of a FIFRA Scientific Advisory Panel (SAP) review that is scheduled conducted from Oct. 18-21. Based on their extensive and comprehensive review, EPA has concluded for the third time that glyphosate is not likely carcinogenic to humans.

Because glyphosate is so widely used in both agricultural and non-agricultural settings and so many weed scientists have been getting questions about the erroneous and misleading IARC classification, I have decided to include the full four page executive summary from the EPA’s Cancer Assessment Review Committee (CARC). The CARC executive summary, while lengthy and technical, does an excellent job of laying out their assessment while describing the shortcomings of the IARC findings.

GLYPHOSATE: Report of the Cancer Assessment Review Committee

<https://www.regulations.gov/document?D=EPA-HQ-OPP-2016-0385-0014>

EXECUTIVE SUMMARY

Glyphosate is a nonselective herbicide that is currently registered for pre- and post-emergence application to a variety of fruit, vegetable, and field crops.

In 1985, the agency, in accordance with the Proposed Guidelines for Carcinogen Risk Assessment, classified glyphosate as a Group C chemical (Possible Human Carcinogen) based on the presence of kidney tumors in male mice. There was no evidence for carcinogenicity in male or female rats. Furthermore, there were no mutagenicity concerns (TXR No. 0052067).

In 1986, the agency requested the FIFRA Scientific Advisory Panel (SAP) to evaluate the carcinogenic potential of glyphosate. On February 24, 1986, the SAP recommended that glyphosate should be categorized as a Group D chemical: **Not Classifiable as to Human Carcinogenicity**. The panel determined that the data on renal tumors in male mice were equivocal: they were only adenomas, and the increase did not reach statistical significance. The panel also advised the agency to issue a data call-in notice for further studies in rats and/or mice to clarify unresolved questions (SAP Report, 02/24/1986). This review is available at http://www.epa.gov/pesticides/chem_search/cleared_reviews/csr_PC-103601_24-Feb-86_209.pdf

In 1991, the Carcinogenicity Peer Review Committee (CPRC) of the Health Effects Division (HED), of the Office of Pesticide Programs (OPP), of the U.S. Environmental Protection Agency (USEPA) evaluated the carcinogenic potential of glyphosate. In accordance with the agency’s 1986 *Draft Guidelines for Carcinogen Risk Assessment*, the CPRC classified glyphosate as a Group E Chemical: **“Evidence of Non-Carcinogenicity for Humans”** based upon lack of evidence for carcinogenicity in mice and rats and the lack of concern for mutagenicity (TXR# 0008897).

Earlier this year (March 2015), the International Agency for Research on Cancer (IARC), Lyon, France, assessed the carcinogenic potential of glyphosate. The IARC reviewed the available epidemiological studies and carcinogenicity studies for glyphosate in experimental animals. The IARC concluded that there is *limited evidence* in humans for the carcinogenicity of glyphosate based on a positive association for non-Hodgkin lymphoma (NHL). The IARC also concluded that there is *sufficient evidence* in experimental animals based on significant positive trends for kidney tumors in one study and for hemangiosarcomas in another study in male mice. IARC determined that there is strong evidence for genotoxicity. Overall, IARC classified glyphosate as “*probably carcinogenic to humans (Group 2A)*” (IARC, 2015).

IARC’s conclusion was based on epidemiologic studies available in the open literature and carcinogenicity studies in rats (4 studies) and mice (2 studies) by dietary administration. Of these six studies reviewed by IARC, two studies in rats and one study in mice were previously not available to OPP. The conclusion by IARC and the additional studies not available to OPP, prompted the agency to re-evaluate the carcinogenic potential of glyphosate.

On September 16, 2015, HED’s Cancer Assessment Review Committee (CARC) evaluated all available epidemiological studies published in the open literature that examined the association between glyphosate exposure and one or more cancer outcomes. This included one cohort study, seven nested case-control studies based on the cohort study population, and 25 case-control studies. The CARC also evaluated 11 chronic toxicity/carcinogenicity studies in rats (7) and mice (4) following dietary administration for up to two years. Six of the studies (4 rat and 2 mouse) were submitted to OPP to support registration/re-registration requirements, including two studies in rats and one study in mice which were not previously available to OPP (but reviewed by IARC). Data for review of the other five studies (3 rat and 2 mouse) were obtained from a review article and its supplement published in the open literature (Greim *et al.*, 2015) that also had not been previously reviewed by the agency (IARC did not evaluate the five studies cited in the Greim *et al.* 2015 review article). The CARC also evaluated the mutagenicity/genotoxicity studies submitted to OPP as well as studies summarized in two review articles (Williams *et al.*, 2000, and Kier and Kirkland, 2013) published in the open literature.

The CARC concluded that the epidemiological studies in humans showed no association between glyphosate exposure and cancer of the following: oral cavity, esophagus, stomach, colon, rectum, colorectum, lung, pancreas, kidney, bladder, prostate, brain (gliomas), soft-tissue sarcoma, leukemia, or multiple myelomas.

The CARC concluded that there is conflicting evidence for the association between glyphosate exposure and NHL. No association between glyphosate exposure and NHL was found in population-based case-control studies in the United States, Canada or France. Additionally, the large prospective Agricultural Health Study (AHS) with 54,315 licensed pesticide applicators in Iowa and North Carolina did not show a significantly increased risk of NHL. A population-based case-control study from Sweden suggested an association between glyphosate exposure and NHL; however, this finding was based on only 4 glyphosate-exposed cases and 3 controls.

When data from two case-control studies in Sweden (one on NHL and the other on hairy cell leukemia) were pooled, a univariate analysis showed an increased risk (odds ratio (OR) = 3.04;

95% confidence interval (CI) = 1.08–8.52); however, when study site, vital status, and exposure to other pesticides were taken into account in a multivariate analysis, the risk was attenuated (OR=1.85; 95% CI=0.55–6.20). In another case-control study in Sweden, among the 29 glyphosate-exposed cases, a multivariate analysis showed an increased risk for NHL (OR=1.51; 95% CI=0.77–2.94) and B-cell lymphoma (OR=1.87; 95% CI=0.998–3.51). A meta-analysis of the six separate studies showed an association between glyphosate exposure and NHL with a meta-risk ratio of 1.5 (95% CI=1.1–2.0) (Schinasi and Leon, 2014). The CARC noted that most of the studies in the database were underpowered, suffered from small sample size of cancer cases with glyphosate exposure, and had risk/odds ratios with large confidence intervals. Additionally, some of the studies had biases associated with recall and missing data.

In an attempt to address the noted power/sample size issues across studies, IARC used adjusted weighting estimates of the two Swedish studies (Hardell *et al.* 2002 and Eriksson *et al.* 2008) and reported an lower odds ratio in a second meta-analysis of the same data (OR=1.3; 95% CI=1.03–1.65). Given the limitations of the studies used and uncertainty in the analytical methods, the CARC concluded that a different weighting scheme could have resulted in a different meta risk ratio. Thus, while epidemiologic literature to date does not support a direct causal association, the CARC recommends that the literature should continue to be monitored for studies related to glyphosate and risk of NHL.

Overall, the CARC concluded that there was no evidence of carcinogenicity in the eleven carcinogenicity studies conducted in Sprague Dawley or Wistar rats and CD-1 mice. There were no treatment-related increases in the occurrence of any tumor type in either sex of either species.

By contrast, the IARC concluded that there is *sufficient evidence* in experimental animals based on a positive trend in the incidence of a relatively rare tumor type, renal tubular carcinoma and renal tubule adenoma or carcinoma (combined) in CD-1 males in one feeding study. A second study reported a positive trend for hemangiosarcomas in male CD-1 mice. The CARC did not consider these tumors to be treatment-related since the observed tumors did not exhibit a clear dose-response relationship, were not supported non-neoplastic changes, were not statistically significant on pairwise analysis with concurrent control groups, and/or were within the range of the historical control data. If the kidney tumors and the hemangiosarcomas are really treatment-related, it is unlikely that the same tumors would not have been detected at higher incidences in the studies in the other studies of CD-1 mice when tested at similar or higher doses (1000–4000 mg/kg/day). Moreover, in 4 of the 11 studies (3 rat and 1 mouse) evaluated by CARC, there was no biologically or statistically significant increases in the occurrence of any tumor type in either species. The other observed differences in incidence did not show a dose response relationship, and were within the range of the background/historical control range. The four studies which were negative for carcinogenicity were reported in the review article by Greim *et al.* (2015) but were not included in the IARC evaluation. This omission of the negative findings from reliable studies may have had a significant bearing on the conclusion drawn for evidence of carcinogenicity in animals.

The CARC evaluated a total of 54 mutagenicity/genotoxicity studies which included studies submitted to the agency, as well as studies reported in the two review articles (Williams *et al.*, 2000, and Kier and Kirkland, 2013). A number of studies reported in the review article by Kier

and Kirkland (2013) were not considered by IARC. The CARC, based on a weight-of-evidence of the *in vitro* and *in vivo* studies, concluded that there is no concern for genotoxicity or mutagenicity. Glyphosate was not mutagenic in bacterial reversion (Ames) assays or *in vitro* mammalian gene mutation assays. There is no convincing evidence that glyphosate induces micronuclei formation or chromosomal aberrations *in vitro* or *in vivo*.

By contrast, IARC's conclusion that glyphosate is genotoxic based on positive results that included studies that tested glyphosate-formulated products as well as studies where the test material was not well-characterized (*i.e.*, no purity information was provided). The IARC analysis also focused on DNA damage as an endpoint (*e.g.*, comet assay). DNA damage is often reversible and can result from events that are secondary to toxicity (cytotoxicity), as opposed to permanent DNA changes which are detected in tests for mutations and chromosomal damage (*e.g.* chromosomal aberrations or micronuclei induction). The studies that IARC cited as positive findings for chromosomal damage had deficiencies in the design and/or conduct of the studies confounding the interpretation of the results. In addition these positive findings were not reproduced in other guideline or guideline-like studies evaluating the same endpoints. Furthermore, IARC's evaluation did not include a number of negative results from studies that were reported in the review article by Kier and Kirkland (2013). The inclusion of the positive findings from studies with known limitations, the lack of reproducible positive findings and the omission of the negative findings from reliable studies may have had a significant bearing on IARC's conclusion on the genotoxic potential of glyphosate.

In accordance with the 2005 Guidelines for Carcinogen Risk Assessment, based on the weight-of-evidence, glyphosate is classified as **"Not Likely to be Carcinogenic to Humans"**. This classification is based on the following weight-of-evidence considerations:

- The epidemiological evidence at this time does not support a causal relationship between glyphosate exposure and solid tumors. There is also no evidence to support a causal relationship between glyphosate exposure and the following non-solid tumors: leukemia, multiple myeloma, or Hodgkin lymphoma. The epidemiological evidence at this time is inconclusive for a causal or clear associative relationship between glyphosate and NHL. Multiple case-control studies and one prospective cohort study found no association; whereas, results from a small number of case-control studies (mostly in Sweden) did suggest an association. Limitations for most of these studies include small sample size, limited power, risk/odd ratios with large confidence intervals, and recall bias as well as missing data. The literature will continue to be monitored for studies related to glyphosate and risk of NHL.
- **In experimental animals, there is no evidence for carcinogenicity.** Dietary administration of glyphosate at doses ranging from 3.0 to 1500 mg/kg/day for up to two years produced no evidence of carcinogenic response to treatment in seven separate studies with male or female Sprague-Dawley or Wistar rats. Similarly, dietary administration of glyphosate at doses ranging from 85 to 4945 mg/kg/day for up to two years produced no evidence of carcinogenic response to treatment in four separate studies with male or female CD-1 mice. The CARC did not consider any of the observed tumors in 11 carcinogenicity studies in rats and mice to be treatment-related since the observed tumors did not exhibit a clear dose-response relationship, were not supported pre-neoplastic changes (*e.g.*, foci, hypertrophy, and

hyperplasia), were not statistically significant on pairwise statistical analysis with concurrent control groups, and/or were within the range of the historical control data.

- Based on a weight of evidence approach from a wide range of assays both *in vitro* and *in vivo* including endpoints for gene mutation, chromosomal damage, DNA damage and repair, **there is no *in vivo* genotoxic or mutagenic concern for glyphosate.**

EPA Manual Available on How to Comply with the Revised Worker Protection Standards

The EPA in conjunction with the [Pesticide Educational Resources Collaborative \(PERC\)](#) has made available a guide to help users of agricultural pesticides comply with the requirements of the 2015 revised federal Worker Protection Standard (WPS). You should read this manual if you employ agricultural workers or handlers, are involved in the production of agricultural plants as an owner/manager of an agricultural establishment or a commercial (for-hire) pesticide handling establishment, or work as a crop advisor. The “How to Comply” manual includes:

- details to help you determine if the WPS requirements apply to you;
- information on how to comply with the WPS requirements, including exceptions, restrictions, exemptions, options, and examples;
- “Quick Reference Guide”- a list of the basic requirements (excluding exemptions, exceptions, etc.);
- new or revised definitions that may affect your WPS responsibilities; and explanations to help you better understand the WPS requirements and how they may apply to you.

The revised EPA Pesticide Worker Protection Standard “How to Comply” Manual is available at: <https://www.epa.gov/pesticide-worker-safety/pesticide-worker-protection-standard-how-comply-manual>

NISC Adopts New Management Plan

The National Invasive Species Council (NISC) announced the release of their 2016-2018 Management Plan. The plan sets forth high priority, interdepartmental actions for the Federal government and its partners to take to prevent, eradicate, and control invasive species, as well as restore ecosystems and other assets adversely impacted by invasive species. The thirteen Federal Departments and Agencies whose senior officials comprise NISC will:

- Provide Federal leadership on invasive species issues by establishing the structures, policy, and planning priorities necessary to enable Federal agencies to effectively prevent, eradicate, and/or control invasive species, as well as restore impacted ecosystems and other assets;
- Limit the spread and impact of invasive species through high-level policy and planning by strengthening coordination between the United States and other governments, across the Federal government, and between the Federal government and non-governmental stakeholders;
- Raise awareness of the invasive species issue and mobilize the policies, programs, and financial resources necessary to minimize the spread and impact of invasive species;
- Remove institutional and policy barriers to the Federal actions needed to prevent, eradicate, and control invasive species, as well as restore ecosystems and other assets;
- Conduct assessments of Federal capacities to meet the duties set forth in Executive Order 13112, as well as other high-level policy priorities, and build Federal capacities, as needed;
- Foster the scientific, technical, and programmatic innovation necessary to enable Federal

agencies and their partners to prevent and mitigate the impacts of invasive species in a timely and cost-effective manner with negligible impacts to human and environmental health.

The 2016-2018 NISC Management Plan is available at:

<https://www.doi.gov/sites/doi.gov/files/uploads/2016-2018-nisc-management-plan.pdf>

FHWA Updates Roadside Revegetation Handbook with Emphasis on Pollinators

In its first major update since 2007, the Federal Highways Administration (FHWA) has expanded their roadside revegetation manual to include a major emphasis on pollinators. The handbook is now titled “Roadside Revegetation: An Integrated Approach to Establishing Native Plants and Pollinator Habitat”. With at least 17 million acres of roadsides in the U.S., roadside vegetation can serve as much needed habitat for pollinators, offering food, breeding, or nesting opportunities and connectivity that can aid pollinator dispersal. Roadside vegetation management influences how pollinators use roadsides, and even influences the number of pollinators killed by vehicles. For example, butterfly vehicle mortality rates increase with more frequent mowing and decrease with high plant diversity in roadside vegetation.

The publication is written specifically for the “designer,” those individuals or members of a road design team who will be directly involved in planning, implementing, monitoring, or maintaining a revegetation project. The first draft was released in September 2016 and is available at:

http://www.nativerevegetation.org/pdf/RoadsideReveg_PollinatorHabitat_DRAFTv1-1_sept2016.pdf

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