BEYOND PESTICIDES
701 E Street, SE ■ Washington DC 20003
202-543-5450 phone ■ 202-543-4791 fax
info@beyondpesticides.org ■ www.beyondpesticides.org

Statement of Beyond Pesticides in Support of H.326 with Amendments
Vermont House Committee on Agriculture, Food Resiliency, and Forestry
May 9, 2025

Honorable Chair Durfee, Vice Chair Surprenant, Ranking Member Morgan, and members of House Committee on Agriculture, Food Resiliency, and Forestry. We appreciate the opportunity to testify on H.326, and the importance of adopting legislation to protect people and local ecosystems from toxic rodenticides. Beyond Pesticides is a national, grassroots, membership organization that represents community-based organizations and a range of people seeking to improve protections from pesticides and promote alternative pest management strategies that reduce or eliminate a reliance on toxic pesticides. Our membership spans the 50 states, the District of Columbia, and groups around the world. We are providing this testimony on behalf of our members and supporters in the state of Vermont.

We urge the Vermont legislature, including the House Agriculture Committee, to vote in favor of H.326 with amendments. While this proposed legislation recognizes a problem, we urge the Committee to place the burden of responsibility on regulatory agencies to consider a broader approach in response to the biodiversity and public health threats referenced in the bill and ensure a more robust response to regulatory failures that exacerbate risks to nontarget organisms from rodenticides, as defined in a large body of peer-reviewed scientific findings.[1]

There are several provisions of the bill that undermine the protections needed and additional issues that must be addressed to affect a meaningful response to pollinator decline and adverse ecosystem effects associated with the use of first- and second-generation rodenticides and related compounds.

The following amendments should be made, as all pesticides, including rodenticides, in commerce are regulated by the U.S. Environmental Protection Agency (EPA) and are said to not cause “unreasonable adverse effects” under federal and state of Vermont law. Therefore, under this language in the bill, all first- and second-generation anticoagulants have already met this standard. The purpose and intent of this legislation, as we understand it, however, is to create a higher standard of environmental protection. The need for improved protection is supported by this testimony, the scientific literature, and findings of EPA deficiencies cited herein.

1. To this end the following text in italics in the same section should be stricken: "…*or when no other pest control method would be effective*."

2. Lines 14 and 15 should be replaced with the following language:

   (D): "no other pest management practice, including organic management practice with delineated allowable substances, will be effective to address such environmental emergency."

3. The following new section should be added to define "delineated allowable substances:"

   a. **Natural, organic or "non-synthetic."** A substance that is derived from mineral, plant, or animal matter and does not undergo a "synthetic" process as defined in the Organic Foods Production Act, 7 U.S.C. § 6502(21), as the same may be amended from time to time.
   b. **Pesticides determined to be "minimum risk pesticides"** pursuant to the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA) and listed in 40 C.F.R. § 152.25(f)(1) or (2), as may be amended from time to time.

It is important that the proposed legislation prioritize ecological pest management practices, best defined in federal law as "organic," as the alternative that must be assessed relative to the use of rodenticides and related compounds because of the numerous deficiencies in the EPA pesticide registration process on which the State of Vermont relies for determinations of safety. With a proper assessment of the need for these highly toxic chemicals to be dispersed in an already vulnerable environment, the state can find that management strategies are available that utilize mechanical, biological, and cultural (operational) practices that prevent the need for toxic pesticides that escalate the destruction of biodiversity and ecosystem services. Legislation that protects ecosystem services preserves the important role that soil organisms, bats, birds, goats, and other animals/forms of wildlife play in preventing pest populations that exceed damage thresholds.

The continued dependence on pesticides, as the current bill language inadvertently allows, fails to respond to the pesticide treadmill effect that elevates pest populations by depressing ecological balance while increasing pest resistance to pesticide applications and reducing plant resiliency to pest populations.

Integrated Rodent Management (IRM)

The first step in pest management is pest prevention. IRM is an Integrated Pest Management (IPM) [2] approach that places strong emphasis on sanitation, pest exclusion (which includes addressing human behavior and structural pest proofing), education and training. To address human behavior, one must focus on food, water, and harborage (FWAH) for rodents.[3] These three factors can help regulators determine the extent of rodent infestation and the causes. Since rodents can reproduce quickly, the problem must be addressed completely, especially

since many of the FWAH factors contributing to the presence of rodents may extend beyond an infested area.

Regular assessments are essential to integrated rodent management. Assessments include performing systematic, regular, and repeat FWAH inspections of all properties, which identify opportunities for exclusion techniques, or rodent proofing homes and buildings, such as repairing holes in a foundation, installing a pest proof door sweep, and keeping residential and commercial doors closed to prevent rodent entry. Landscape planning is also part of preventing rodent infestations, which may include: removal of ivy along the base of buildings, since rodents like to burrow in ivy and similar ground cover; small mesh heavy gauge wire fencing and trenches to act as a barrier to prevent rodent access; planting repellents like gopher purge (*Euphorbia lathyrus*), castor bean (*Ricinus communis*), and garlic, and; use of repellent sprays, including cayenne pepper and peppermint soap to deter rodents. Ecological rodent management may include introducing or encouraging natural predators like gopher snakes, corn snakes, rat snakes, owls, hawks, great blue herons, weasels, bobcats, coyotes, and domestic dogs and cats. For example, the installation of owl boxes, can provide a very effective way to address rodent populations on farms and in large landscaped areas.[4] Lastly, dry ice placed in rodent burrows, carbon dioxide-controlled traps, and electric traps provide an instant and humane death without harming humans and others.[5]

IRM is a successful approach that municipalities can use instead of rodenticides to eliminate rodent infestations. IRM provides a more comprehensive and preventive approach to eliminating the conditions that lead to pest infestation—as opposed to rodenticides, which put a band-aid on the problem, rather than tackling the pest-conducive conditions at the source.

Rodenticide Risks

For baits, the rodenticide must be eaten by pests and uses feeding stations. Tracking powders must also be eaten—they stick to fur and are ingested via grooming. Risk includes bioaccumulation of rodenticides in pests and potential secondary poisoning. Pests can also develop resistance to rodenticides, allowing them to ingest and tolerate higher concentrations of the product. In turn, nontarget organisms who consume the pest ingest a higher concentration of rodenticides. Lastly, fumigants involve releasing poisonous gases into inhabited areas (e.g., burrows, holes, etc.) However, risk includes drifting to nontarget organisms since the applied toxic substance does not remain in the treated area.

First-Generation Anticoagulants

A persistent issue among first-generation rodenticides is resistance, with evidence dating as far back as the 1970s.[6] Rats developing resistance to anticoagulant rodenticides pose a risk to the food chain, as presence and uptake of rodenticides results in higher concentrations to those higher on the food chain. For example, levels found in wild nontarget species range from trace amounts to lethal levels and occur in up to 81% of barn owl carcasses surveyed.[7]

Toxicity for all generations of rodenticides is high due to their mode of action and directly impacts the clotting ability of blood. An antivitamin K rodenticide[8], chlorophacinone is one of

the only ones registered for field use in wildlife.[9] Long-term exposure to diphacinone causes nerve, heart, liver, and kidney damage as well as damage to skeletal muscles. Like all other first-generation rodenticides, warfarin inhibits the synthesis of vitamin K-dependent clotting factors. Symptoms of poisoning do not appear suddenly and will culminate in death in rodents within about 5–7 days of initial ingestion. Therefore, the risk of secondary poisoning increases during that period.

Second-Generation Anticoagulants

Just like first-generation rodenticides, pests are developing resistance to second-generation rodenticides. Rodents developing resistance to anticoagulant rodenticides pose a risk to the food chain, so presence and uptake of rodenticides results in higher concentrations entering those higher on the food chain. Additionally, controlled exposure studies have demonstrated that second-generation anticoagulant rodenticides are more toxic than first-generation compounds, however the difference in potency is diminished when first-generation compounds are administered on multiple days.

Although there are no residential uses, substantial mortality was noted in secondary exposure studies in mammals ingesting prey or tissue diets containing either second-generation or non-anticoagulant compounds.[10] One unintended consequence of restricting these rodenticides is the real possibility that industry will replace them with equally toxic active ingredients like non-anticoagulants. To avoid this problem, the agency must consider the full effects of the potential shift in the market and the resulting exposure, poisoning, and contamination.

The California Department of Pesticide Regulation quotes the Fish and Wildlife Service: "Secondary exposure to SGARs [second generation anticoagulant rodenticides] is particularly problematic due to the high toxicity of the compounds and their long persistence in body tissues. For example, brodifacoum, a common SGAR, is persistent in tissue, bioaccumulates, and appears to impair reproduction[…]. Even in cases where the proximate cause of death has been identified as automobile strike, predation, or disease, toxicologists and pathologists have attained sufficient toxicological evidence to conclude that rodenticide-induced blood loss increased animal vulnerability to the proximate cause of death."[11]

A study in *Science of The Total Environment* finds evidence of SGARs in frog species. Brodifacoum was found in four of the six frog species analyzed by the researchers, and they share, "This is the first report of anticoagulant rodenticide detected in wild amphibians, raising concerns about potential impacts on frogs and extending the list of taxa shown to accumulate rodenticides."[12] Prior research, also published in *Science of The Total Environment*, designates SGARs as "(very) persistent, (very) bioaccumulative, and toxic."[13] While new research is continuing to emerge regarding rodenticides, the authors highlight the previous lack of focus on aquatic species: "So far, worldwide monitoring of AR residues mainly focused on terrestrial and avian non-target species and their routes of exposure… AR residue screening in aquatic compartments is challenging, and accordingly little is known about direct and indirect exposure routes as well as anticoagulants' distribution and fate in the aquatic environment."

The researchers also share: “Further research should investigate the potential risks and hazards of ARs in the aquatic environment in order to pave the way for scientific-based, targeted, and effective regulatory decisions. Until then, the ecological implications for aquatic organisms as well as fish-eating predators remain largely unknown.” This highlights the many data gaps that call in to question EPA’s ability to declare risks to aquatic organisms as “not reasonably certain to occur.”

All vertebrates that eat bait or prey poisoned with brodifacoum are at risk of second-generation poisoning. Compounds are slowly eliminated from the liver and therefore accumulate in vertebrates. Species most at risk from secondary poisoning are predatory and scavenging birds.[14] The threat of secondary poisoning has led the state of California to ban the use of brodifacoum for almost all uses as the risks are extremely high.[15] Additionally, with the increase in marijuana products for commercial use on state levels, an issue with the “outbreak of life-threatening bleeding following inhalation of synthetic cannabinoids has been attributed to contamination with the long-acting anticoagulant rodenticide (LAAR) brodifacoum, a second-generation, highly potent, long-acting derivative of the commonly used blood thinner warfarin.”[16,17] Although there are claims for use to maximize crop production from illegal synthetic cannabis producing facilities, unknowing consumers inhale these toxic chemicals.[18]

Bromadiolone, as known as “Super-warfarin,” has a long half-life that poses a greater risk for bioaccumulation and further contamination in wildlife. Being one of the only rodenticides registered for field use in wildlife, [19]bromadiolone exposure can cause excessive or inappropriate bleeding of skin mucosa, digestive tract and urinary tract; possible intracerebral haematoma.[20] Because of the lower acute toxicity threshold for second-generation rodenticides, it may only take a single, rather than multiple, exposure to induce toxic symptoms. Therefore, primary poisoning poses a greater risk to nontarget species that may consume contaminated substances. For instance, difenacoum is often involved in primary poisonings of domestic animals and secondary poisonings of wildlife that feed on contaminated rodents.[21] Additionally, secondary poisonings are a very high risk among bird and mammals exposed to difethialone.

Non-Anticoagulants

As resistance to traditional anticoagulants grows, regulators may turn to non-anticoagulants for rodent management. Since these compounds can vary in modes of action, symptoms of animals suffering from exposure to non-anticoagulant rodenticides suffer from the following list of immediate toxic effects: rapid onset of seizures, muscle tremors, limb weakness, ataxia, neurologic signs, respiratory paralysis, anorexia, nausea, vomiting, diarrhea, and lethargy.[22] Unlike anticoagulants, non-anticoagulants have no antidote, and the treatment is spotty, as symptoms often return.

We acknowledge the effort of this legislature to specifically address Bromethalin-based rodenticides in H.326. Bromethalin is highly neurotoxic and works by disrupting adenosine triphosphate (ATP) production which impairs cellular ability to control osmosis. As the cells swell with water, so too does the brain and spinal cord fill with cerebrospinal fluid, putting pressure

on the central nervous system. This damage can cause paralysis, convulsions, and death.[23] In 2022, a case of bromadiolone poisoning was recorded in a patient who had digestive tract, abdominal hemorrhage, as well as secondary paralytic ileus.[24] Cholecalciferol is toxic to all vertebrates and exposure can result in vitamin D_3 accumulation. The main consequences of excess vitamin D or vitamin D toxicity are buildup of calcium in the blood leading to calcium stone formation, bone pain, nausea, vomiting, weakness, and frequent urination via kidney problems.[25] A health-based case study in 2020 finds the use of non-anticoagulants as a replacement for second-generation anticoagulants still poses a risk to human health, especially for intentional poisonings. For instance, "A 56-year woman was admitted to our hospital following a 3-week history of nausea, vomiting, and muscle weakness. The patient had been assuming a very high dose of cholecalciferol for 20 months (cumulative 78,000,000UI, mean daily 130,000UI), as indicated by a non-conventional protocol for multiple sclerosis."[26]

Strychnine is extremely toxic, having specific uses on mammalian pests (e.g., coyotes, foxes). With a rapid onset of symptoms (within 15-20 mins of ingestion), [27]exposure can cause violent convulsions through the central nervous system, chiefly the spinal cord. Death is caused by convulsive-induced muscle spasms of the diaphragm and thoracic intercostals, resulting in impaired respiration. However, increased use of strychnine has resulted in changes in mammalian communities, such as size. For instance, larger coyotes may be able to withstand multiple exposure to non-anticoagulants compared to smaller coyotes, developing resistance over time. As these smaller coyotes die off, the larger ones will reproduce and have more access to food, water, and harborage. Additionally, ingestion of zinc phosphide rodenticide baits poses an immediate primary poisoning risk. Stomach acid causes the zinc phosphide to release phosphine. Phosphine distributes throughout the body, especially the liver, kidney, and central nervous system causing nausea and vomiting, agitation, chills, chest tightness, dyspnea, and cough may progress to pulmonary edema. Despite the body's ability to break down the phosphine into less toxic compounds, the process is extremely slow, increasing accumulation risk.

Unreasonable Adverse Effects from Exposure

Rodenticide exposure has links to various health and environmental effects highlighted in the scientific literature. Exposed humans can suffer internal bleeding, coma, anemia, nosebleeds, bleeding gums, bloody urine and bloody stools. Because rodents are mammals, many mammalian species or species that eat mammals can experience the same death as target pests.

Children

Children's bodies are smaller and can experience greater toxic burdens to the body than adults. Considering that children can ingest a rodenticide without an adult knowing, it may be too late to save a child's life once symptoms progress. Additionally, the onset of symptoms may quicken. EPA concedes that the number of exposure incidents resulting in symptomatic diagnoses and/or requiring treatment is unacceptably high. Data from the New York City Department of Health and Mental Hygiene also indicates that between 2000 and 2010 of a total 4,250 unintentional exposures to rodenticides, 79% were children less than six years old. Exposure to these poisons

have been shown to cause paralysis due to cerebral hemorrhage and is teratogenic (causes birth defects).[28]

Additionally, children in low-income families are disproportionately exposed, especially in urban areas.[29] Because the city officials determine how to manage rodents in urban areas, they may turn to traditional toxic rodenticides as a quick and cheap option. However, the Integrated Rodent Management section above highlights that municipalities can use nonchemical techniques instead of rodenticides to eliminate rodent infestations. IRM provides a more comprehensive approach to reducing resources leading to pest infestation, rather than rodenticides, which put a band-aid on the issue, rather than tackling the issue at the source

Pets and Wildlife

Beyond the known health risks at home, there is strong evidence that pets and wildlife are being poisoned due to secondary exposure to rodenticide baits. Rodents can feed on poisoned bait multiple times before death, and as a result their carcasses contain residues that may be many times the lethal dose. Poisoning occurs when predators or scavengers feed on these poisoned rodents. Studies have deemed anticoagulant rodenticides "super-predators" in ecosystems because of the widespread damage that can result from their use. This is because rodents that eat these chemicals, often contained in toxic baits, do not die immediately. The anticoagulant nature of these rodenticides means that they stop an animal's blood from clotting, resulting in a slow, painful death. The animal becomes confused and slow, blood vessels are ruptured, hair and skin loss begin to occur, and nosebleeds and bleeding gums will present prior to succumbing to the poison. While a rodent is likely to die from this poison, ingesting it also turns it into a sort of poison trojan horse for any predator that may take advantage of its slow decline. For instance, predator birds and scavenging animals can eat a poisoned rodent at the edge of death will be the next to succumb to the anticoagulant effects of the chemical. If not killed outright, a poisoning event can weaken a predator's immune system and make the animal more susceptible to disease.[30]

As documented by Lohr, M. *et al*., "Anticoagulant rodenticides (ARs) have been detected in non-target wildlife species worldwide… Second generation anticoagulant rodenticides (SGARs) pose a particular threat to scavengers and top-order carnivores because their long half-lives allow for biomagnification and bioaccumulation beyond their intended rodent targets." In analyzing liver tissues from carnivorous and scavenging mammals, 50% tested positive for the presence of ARs. Multiple samples showed more than one AR compound as well.[31]

"This study is the first to document widespread and pervasive AR exposure in native Australian marsupial carnivores, including those in remote locations away from towns," the researchers share. They continue: "The frequency and severity of exposure, sometimes from multiple ARs, suggest potential population-level impacts on these threatened species. These findings provide further evidence that ARs should be listed as a key threatening process under state and federal legislation."

A similar study, in *Environmental Chemistry Letters*, reports: "We analyzed residues of eight anticoagulant rodenticides in liver samples of 96 great cormorants, 29 common mergansers, various fish species, and coypu, in different German regions. Results show that hepatic residues of anticoagulant rodenticides were found in almost half of the investigated cormorants and mergansers due to the uptake of contaminated fish from effluent-receiving surface waters."[32] This highlights the presence of ARs in aquatic organisms that are then transferred through the aquatic food web to predators and adds to the concern about ARs' propensity for biomagnification and bioaccumulation.

The authors conclude that: "Our biomonitoring study demonstrated that piscivorous avian predators in anthropogenically influenced landscapes are exposed to second-generation anticoagulant rodenticides via their fish prey. Transfer of second-generation active ingredients along the aquatic food chain was thus confirmed. Without doubt, future improvements of regulatory measures concerning biocides will be required to mitigate the yet unknown consequences for aquatic wildlife from the nowadays almost exclusive application of second-generation anticoagulant rodenticides during chemical rodent control."

Also documenting secondary exposure to ARs, a study in *The Journal of Wildlife Management* shows how anticoagulant rodenticides cause "the death of mammalian predators and scavengers directly and indirectly through sublethal effects that reduce fitness." In quantifying AR exposure in carcasses of 365 urban and suburban coyotes in southern California, the researchers report, "Nearly all urban coyotes (98.1%) were exposed to at least 1 AR, compared to 41.7% of rural coyotes, and most individuals had residues of both first-generation (FGAR) and the more potent second-generation (SGAR) compounds, often at concentrations exceeding thresholds considered lethal in other mammals."[33]

The authors also share that the "adults tended to have residues of more compounds and at higher concentrations than juveniles, suggesting repeated and chronic exposure." They continue, "[S]ome coyotes showed evidence of internal bleeding consistent with AR toxicosis and were in poorer body condition," raising additional concerns for mechanisms of toxicity.

U.S. Fish and Wildlife Service 2006 Comments to EPA on anticoagulants say:
"Widespread nontarget exposure to anticoagulants cannot be disputed. Based on a study of carcasses collected from 1998-2001 in New York State, including samples asymptomatic of anticoagulant exposure submitted for West Nile Virus surveillance, Ward Stone, Wildlife Pathologist for New York State Department of Environmental Conservation, concluded that anticoagulants were present in the majority of great homed owls, about half of the red-tailed hawks, and in a substantial fraction of other raptors in New York State (Stone et al., 2003). Detection of more than one rodenticide in a number of these carcasses indicates that a percentage of these birds are acquiring these residues through multiple exposures."

Endangered Species
Due in large part to the use of rodenticides in the cultivation of illegal marijuana grow operations, earlier this month the United States Fish and Wildlife Service (FWS) announced a

proposal to list fishers, medium sized carnivores of the weasel family, as threatened under the Endangered Species Act.[34]

Conclusion

While we support the elimination of toxic rodenticides, it must be noted that these chemicals are merely the "poster children" for broader problems associated with EPA's evaluation and registration of pesticides. At a time of cascading and intersecting public health, biodiversity, and climate crises, we must stop the use of chemical classes causing immense harm; yet, we must also move toward an approach that incentivizes sustainable practices that do not necessitate these chemicals in the first place.

In addition, no human health or environmental safety findings associated with the Endocrine Disruptor Screening Program (EDSP) were made in the registration process for various rodenticide products. EPA must examine all ingredients in these products, including so-called "inert" or "other" ingredients for endocrine disrupting properties. An Endocrine Disruptor Screening Program FFDCA § 408(p) determination is required for registration. It is simply unacceptable to continue to register new pesticides without EDSP findings, thus creating an even greater backlog, while evaluating chemicals presented good affinities *in silico* for proteins associated with breast cancer, oxidative stress and metabolism of xenobiotic compounds.[35]

Rodenticides disproportionate use patterns in urban areas highlight the potential adverse impacts and disproportionate risk to people of color communities (including, but not limited to essential workers) and the need to consider environmental justice when developing best pest management practices. An adequate assessment must include an evaluation of the potential adverse effects cited in this document in relationship to underlying conditions and comorbidities that exist in these communities, with specific attention to each potential health outcome and its potential disproportionate effect on people of color.

According to "Anticoagulant Rodenticides and Wildlife" in the journal *Emerging Topics in Ecotoxicology,* ecological risk assessments of anticoagulant rodenticides can improve with additional data. A more complete understanding of the toxicity of anticoagulant rodenticides in nontarget wildlife would enable regulators and natural resource managers to better predict and protect against harm."[36]

In summation, we urge passage of H.326 with the considerations proposed in our statement. With the adoption of these changes to H.326, we urge the Vermont legislature to take action in the context of eliminating damaging pesticides that can be replaced by practices and materials compatible with the environment and public safety.

We would be happy to work with the legislative sponsors to achieve these broader health and sustainability goals going forward. Vermont has the opportunity to reverse adverse ecosystem impacts exacerbated by rodenticides, while concurrently increasing protections for public health and the wider environment.

Thank you for your consideration of our comments.

Jay Feldman, Executive Director
Sara Grantham, Science, Regulatory, and Advocacy Manager
Max Sano, Senior Policy & Coalitions Associate
Beyond Pesticides

Endnotes

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