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December 6, 2024

Office of Pesticide Programs
Environmental Protection Agency, (28221T)
1200 Pennsylvania Ave., NW
Washington, DC 20460-0001

Re: Pesticide Product Registration: Applications for New Uses (Bifenthrin) [EPA-HQ-OPP-2023-0555]

Dear Madam/Sir,

These comments are submitted on behalf of Beyond Pesticides. Founded in 1981 as a national, grassroots, membership organization that represents community-based organizations and a range of people seeking to bridge the interests of consumers, farmers, and farmworkers. Beyond Pesticides advances improved protections from pesticides and alternative pest management strategies that eliminate a reliance on pesticides. Our membership and network span the 50 states and the world.

In the Environmental Protection Agency (EPA) approving additional uses of bifenthrin on crops, the pesticide treadmill is perpetuated and further contributions to the existential crises of biodiversity collapse, health threats, and the climate emergency occur. Bifenthrin is a pyrethroid insecticide that has reported hazards of organ damage and suspected carcinogenicity with human exposure, as well as endocrine disruption and neurological, dermal, respiratory, ocular, cardiovascular, and gastrointestinal effects. [1,2] The National Institutes of Health reports, "Exposure to bifenthrin, even at 'acceptable' limits, can increase the risk for and frequency of inflammatory responses and diseases such as asthma."[1] Studies in the last two years associate bifenthrin exposure to increased incidence of liver disease and prostate cancer as well.[3,4]

Bifenthrin is also linked to high toxicity in aquatic organisms and pollinators on an acute and chronic basis.[1,2] Studies find bifenthrin in water samples that threaten aquatic habitats.[5] One study, in *Aquatic Toxicology*, of pesticide contaminated algae finds that the disruption of algal communities has a devastating effect on the health of the aquatic food web. The study findings show that contact with pesticides can result in changes to "algal physiology, causing tissue injury, developmental delay, genotoxicity, procreative disruption, and tissue biomagnification" that alters the dominance of algae species in the environment. This in turn "can impact higher trophic levels and have a domino effect on the aquatic food web. It is possible for biodiversity to disappear, reducing ecosystem stability and resistance to environmental alterations," the authors state.[6]

Effects to Children

The concern about bifenthrin extends to the entire synthetic pyrethroid family and the common mechanism of toxicity for all chemicals within the family.[7] We are particularly concerned about the risks that bifenthrin and other synthetic pyrethroids pose to children. Published data finds that children are increasingly exposed to synthetic pyrethroids as restrictions around the home use of organophosphate insecticides have been adopted.[8]

Links to developmental problems are most notable in scientific literature. Living near a field where pyrethroids were applied during a woman's third trimester corresponded with an 87% increased risk of having a child with autism.[9] Children whose mothers were highly exposed to synthetic pyrethroids during pregnancy are three times more likely to have mental delay compared to children whose mothers experienced lower levels of exposure.[10] Exposure during pregnancy, and high levels of the pyrethroid metabolite cis-DCCA is associated with internalizing disorders in children up to six years old, while high levels of a different synthetic pyrethroid metabolite, 3-PBA, is associated with externalizing disorders, such as behavioral and impulse-control issues.[11] Pregnancy exposures and higher concentrations of pyrethroid breakdown products in maternal urine samples correspond with a 98% increase in the odds of their children having ADHD scores in the 90th percentile at ages 2-4, a strong predictor for an ADHD diagnosis.[12] Similar findings have been documented for older children, aged 6-11. For every tenfold increase in urinary levels of pyrethroid metabolites, children are twice as likely to score high on parent-reported behavioral problems, such as inattention and hyperactivity.[13]

Endocrine Disruption

On endocrine disruption cited above, there is evidence that pyrethroid metabolites have more endocrine disrupting activity than their parent compounds, dependent on the optical isomer present.[14] Research has demonstrated that the estrogenic effects of certain pyrethroids increase the levels of estrogen in breast cancer cells.[15] Because increased cell division enhances the chances for the formation of a malignant tumor in the breast, artificial hormones, like those found in pyrethroids, may increase breast cancer risk. Moreover, pyrethroid insecticides exhibit the highest affinities for multiple breast cancer proteins.[16] There is evidence that ambient pesticide exposure to those living or working near agricultural pyrethroid pesticide applications can induce chronic neurological issues, like Alzheimer's disease, and psychological disorders, in addition to disruption of the central nervous system that induces various cancers.[17] Environmental exposures to pyrethroids insecticides are associated with an increased risk of mortality from all causes and specific causes, including cardiovascular disease and cancer.[18] Some pyrethroids are classified as human carcinogens by EPA.

To the extent that EPA has not fully evaluated the endocrine disrupting effects of pesticides that have been extensively captured in the independent scientific literature, the agency should not be expanding uses of bifenthrin and synthetic pyrethroids until such time as they are fully evaluated for endocrine disruption. This would be an unreasonable exposure in light of current science.

Pyrethroids and the Environment

While the development of synthetic pyrethroids was heralded with claims of selective toxicity to insects, both pyrethroids and pyrethrins are extremely toxic to aquatic organisms, including fish such as the bluegill and lake trout, with LC50 (lethal air concentration for 50% of the test population) values less than 1.0 parts per billion. These levels are similar to those for mosquito, blackfly, and tsetse fly larvae, often the actual targets of a pyrethroid application. Lobster, shrimp, mayfly nymphs and zooplankton are the most susceptible nontarget aquatic organisms.[19] The nonlethal effects of pyrethroids on fish include damage to the gills and behavioral changes. Since aquatic organisms lack enzymes that can hydrolyze (breakdown) pyrethroids, oxidative stress is the primary mechanism of toxicity among fish and aquatic organisms. Many pyrethroids induce reactive oxygen species (ROS) production in the gills, liver, and muscle of fish, which leads to histological changes, such as lipid peroxidation and alterations in the expression and activity of antioxidant enzymes.[20] Pyrethroids are moderately toxic to birds, with most LD50 values greater than 1000 mg/ kg. Birds can also be indirectly affected by pyrethroids, because of the threat to their food supply. Waterfowl and small insectivorous birds are the most susceptible. Moreover, wild birds can encounter pyrethroids mainly in urban areas, increasing the number of unhatched eggs.[21] Both beneficial insects and "pests" are affected by pyrethroid applications. Many wild bees who forage on flowers in pyrethroid treated areas have seen reductions in colony size, and other beneficial insects (e.g., beetles, grubs, worms) encountering pyrethroid residues in soil have a high mortality rate.[22] In some cases, predator insects may be susceptible to a lower dose than the target insect, disrupting the predator-prey relationship. Synthetic pyrethroid use can induce trophic cascades in aquatic ecosystems. By killing off larval aquatic macroinvertebrates, like mayflies, stoneflies, and caddisflies who eat periphyton (attached algae), their use can result in algae blooms. The endocrine-disrupting properties of many synthetic pyrethroids can also cause aquatic organisms to speed up their metamorphosis, emerging smaller and earlier than usual, resulting in less food for amphibians, reptiles, and birds who rely on these insects as a food source.[23]

Conclusion

The threats to humans and wildlife, as well as the environmental implications of bifenthrin use, are exorbitant. Although pesticides are by definition harmful, what makes these adverse effects "unreasonable" is the existence of an alternative—an organic production system—that does not harm human health, other species, or ecosystems and, in addition, helps to mitigate climate change.

In all its decisions, EPA must use organic production as a yardstick, denying any toxic chemical for which organic production is successful. EPA is required to consider these alternative management practices and materials that are available, such as those used in organic agriculture, to conduct an accurate assessment, compliant with the unreasonable adverse effects standard of the *Federal Insecticide, Fungicide, and Rodenticide Act* (FIFRA),[24] of the hazards associated with continued and expanded pesticide use. A failure to evaluate alternative practices results in a decision that lacks scientific integrity.

We urge the agency to deny any further uses of bifenthrin, as well as to consider revoking the registration of this harmful compound, due to findings of high risk and demonstrated adverse impacts on health and the environment.

Thank you for your consideration of our comments.

Respectfully,

Sara Grantham
Science, Regulatory, and Advocacy Manager

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