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An Open Letter to EPA

Environmental Protection Agency
Docket Center (EPA/DC), (28221T)
1200 Pennsylvania Ave. NW
Washington, DC 20460-0001

Re: Clothianidin; Pesticide Tolerances for Emergency Exemptions
Docket No: EPA-HQ-OPP-2014-0253

We urge the U.S. Environmental Protection Agency (EPA) to open for public comment the time-limited tolerance for clothianidin granted to Florida citrus growers on an emergency basis, allowing use for 2½ years, under the Federal Insecticide Fungicide and Rodenticide Act (FIFRA) and Federal, Food, Drug, and Cosmetic Act (FFDCA). The Florida Department of Agriculture and Consumer Services requested an emergency "crisis" exemption for the specific use of clothianidin as a soil drench on immature citrus trees to control transmission of Huanglongbing (HLB) disease caused by the Asian Citrus Psyllid (ACP).

According to the state of Florida, clothianidin is needed due to the "lack of effective available alternatives for season long control practices," and "significant economic losses will occur if this urgent, non-routine disease is not controlled." However, as evidenced by the 6,000 acres of organic growers operating under threat of ACP, and without significant economic losses as a result of HLB, effective season long control practices exist and are currently in practice by organic Florida citrus growers. EPA should not, without public comment, approve the emergency exemption for clothianidin, a member of the nitroguanidine subgroup of neonicotinoids, a neurotoxic class of persistent and systemic insecticides implicated in adverse biological and ecological impacts to non-target invertebrates throughout the world.[1]

Further, EPA has not identified the lack of efficacy with other chemicals currently on the market, weakening claims that this need arises from an "urgent, non-routine disease" and "lack of effective available alternatives for season long control practices." Because of the toxic, persistent and systemic nature of this chemical, the agency has a duty to provide the public with information concerning the registered products that have proved ineffective.

Rather than expand the uses of this toxic chemical and further imperil pollinators and other non-target invertebrates, EPA should act to suspend the use of clothianidin, and further

[1] L.W. Pisa et al. 2014. Effects of neonicotinoids and fipronil on non-target invertebrates. *Environmental Science and Pollution Research.* http://link.springer.com/article/10.1007/s11356-014-3471-x

develop biological controls, like predator *tamarixia* wasps, to stop the spread of the citrus psyllid. Although the immediate chemical needs of chemical-intensive agriculture for increasingly toxic and persistent chemicals is understood, EPA must play a role in helping to stop the treadmill, lest it allow irreversible harm to the environment, biodiversity, and human health.

In the absence of a full suspension on the use of clothianidin, the agency must require that growers who intend to use clothianidin through an emergency application create a management plan for the adoption of alternative methods. Such a plan will require growers to adopt integrated and organic pest management systems that will ultimately decrease long-term reliance on toxic chemicals, such as clothianidin or other neonicointoids, which may be used, and prevent future emergencies which involve the toxic pesticide uses that are not registered.

Honey bees in Florida, researchers say, contribute to the productivity of several groups of citrus fruit, including many orange and grapefruit varieties. Through their pollination services and foraging in citrus fruit, bees and other pollinators will be exposed to the contaminated pollen and nectar in the trees' flowers, as the systemic clothianidin translocates throughout the treated trees. Bees are exceedingly common in citrus groves, from which they produce a high quality honey crop.

According to the University of Florida, there are approximately 6,000 acres of certified organic citrus in Florida. Farm operations that are USDA certified organic avoid the use of toxic chemicals by implementing holistic management systems plans.

<u>Federal agencies have begun to eliminate the use of neonicotinoids in light of non-target harms to pollinator species:</u>

- The Council on Environmental Quality and the General Services Administration addendum to their Sustainable Practices for Designed Landscapes guidance states that, "Chemical controls that can adversely affect pollinators should not be applied in pollinator habitats. This includes herbicides, broad spectrum contact and systemic insecticides, and some fungicides." [2]

- The U.S. Fish and Wildlife Service (FWS) in its decision to phase out neonicotinoid use has stated that neonicotinoids "can be effective against targeted pests, but may also adversely impact many non-target insects," and that "the prophylactic use of neonicotinoids and the potential broad-spectrum adverse effects to non-target species do not meet the intent of IPM principles or the Service's Biological Integrity, Diversity, and Environmental Health (BIDEH) policy."[3]

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[2] Council on Environmental Quality and the General Services Administration. 2014. Supporting the Health of honey Bees and Other Pollinators.
http://www.whitehouse.gov/sites/default/files/docs/supporting_the_health_of_honey_bees_and_other_pollinators.pdf

[3] US Department of Interior. US Fish and Wildlife Service. 2014. Memorandum: Guidelines regarding the interim use and phase out of neonicotinoid insecticides to grow agricultural crops for wildlife on NWRs in the Pacific

Clothianidin Poses a Risk To Honey Bees and Other Pollinators, and Its Registration Should Be Cancelled

Clothianidin belongs to the nitroguanidine subgroup of neonicotinoids, a neurotoxic class of insecticides. Neonicotinoids affect the nervous system of insects, causing irreversible blockage of the postsynaptic nicotinergic acetylcholine receptors (via a selective agonistic mechanism).[4] Clothianidin is a potential risk to non-target organisms including terrestrial and aquatic invertebrates, birds, and mammals. For the honey bee, toxicity has been observed at LD_{50} 22 ng/bee for clothianidin (30 ng/bee for thiamethoxam),[5] levels lower than those noted in EPA's clothianidin registration factsheet (LD_{50}> 43.9 ng/bee). In fact, clothianidin and thiamethoxam are only second in toxicity to imidacloprid (LD_{50} 17.9 ng/bee) for this class of chemicals. Clothianidin has also been observed to be highly toxic to other wild bee species like the common eastern bumble bees (*Bombus impatiens* (Cresson), alfalfa leafcutting bees (*Megachile rotundata* (F.)) and the blue orchard bee (*Osmia lignaria* Cresson).[6]

Neonicotinoid insecticides like clothianidin have sublethal effects in honeybees, which include disruptions in mobility, navigation, and feeding behavior.[7] Lethal and sublethal exposures have been shown to decrease foraging activity, along with olfactory learning performance and decrease hive activity.[8] In a study looking at the acute effects of sublethal doses of clothianidin under field-like conditions at 0.05 -2 ng/bee, a significant reduction of foraging activity and longer foraging flights at doses of ≥0.5 ng/bee during the first three hours after treatment were recorded.[9] Bees are exposed to these pesticides via foliar and systemic treatments when they pollinate flowering crops, or pesticide drift from surrounding areas. Bees living and foraging near agricultural fields are exposed through multiple mechanisms throughout the spring and summer.

Researchers are beginning to discover that in addition to residues found in pollen and nectar,[10,11] neonicotinoid pesticides can be detected in other areas. Guttated water of seed-treated plants, which provides a source of water for bees, can also be a source of

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Region. Available at: http://www.centerforfoodsafety.org/files/guidelines-for-interim-use-and-phase-out-of-neonicotinoid-insecticides-in-refuge-farming-for-wildlife-programs-signed-kf-7914_67415.pdf

[4] USEPA. 2011. BEAD Chemical Profile for Registration Review: Clothianidin (044309). Federal Register Docket Id. No.: EPA–HQ–OPP–2011– 0865

[5] Iwasa, T el al. 2004. Mechanism for the differential toxicity of neonicotinoid insecticides in the honey bee, *Apis mellifera. Crop Protection*. 23(5): 371–378.

[6] Scott-Dupree CD, Conroy L, Harris CR. 2009. Impact of currently used or potentially useful insecticides for canola agroecosystems on Bombus impatiens (Hymenoptera: Apidae), Megachile rotundata (Hymentoptera: Megachilidae), and Osmia lignaria (Hymenoptera: Megachilidae). *J Econ Entomol.* 102(1):177-82.

[7] Desneaux, N. et al., 2007. Sublethal Effects of Pesticides on Benefical Anthropods. Annual Review of Entomology, 52:81-106

[8] Decourtye, A. et al., 2004. Effects of imidacloprid and deltamethrin on associative learning in honeybees under semi-field and laboratory conditions. Ecotoxicology and Environmental Safety.57: 410-419

[9] Schneider CW, Tautz J, Grünewald B, and Fuchs S. 2012. RFID Tracking of Sublethal Effects of Two Neonicotinoid Insecticides on the Foraging Behavior of Apis mellifera. *PLoS One:*7(1):e30023.

[10] Bernal J, et al. 2010. Overview of pesticide residues in stored pollen and their potential effect on bee colony (Apis mellifera) losses in Spain. J Econ Entomol;103(6):1964-71.

[11] Krupke CH, Hunt GJ, Eitzer BD, Andino G, Given K. 2012. Multiple Routes of Pesticide Exposure for Honey Bees Living Near Agricultural Fields. *PLoS ONE* 7(1): e29268. doi:10.1371/journal.pone.0029268.

contamination and exposure.[12] Reetz et al. found that corn seeds treated with clothianidin resulted in neonicotinoid concentrations up to 8,000 ng/ mL in the guttated fluid.[13] This concentration, while decreasing rapidly, remained detectable over several weeks.

The rapid disappearance of the honey bees, also dubbed "Colony Collapse Disorder" or CCD, has been observed in the U.S. since 2006. Even though researchers have indicated that there may be several variables associated with CCD, clothianidin, and other chemicals in its class, cannot be ruled out as a major contributor and this must be factored into the agency's assessment. Honey bees intercept, and are contaminated by, particles on crops and suspended in the air, and retain them in their hair-like setae and/or accumulate them in their bodies and hives. Mitigation techniques (e.g. product label restrictions) to prevent honeybees from coming into contact with this highly toxic pesticide once it is used in the environment are highly infeasible. The only way to protect important pollinators is to remove these toxic neonicotinoids from the environment.

Neonicotinoids Affect Long-Term Ecosystem and Biodiversity

Neonicotinoid residues have been found in pollen, nectar and droplets of water exuded by the plant due to the systemic nature of the chemicals. This feature makes the plant effectively toxic to insects that could potentially cause plant damage, resulting in protection for the plant. However, this also results in increasing their exposure potential to pollinators.

- The International Union for the Conservation of Nature, known as the Task Force on Systemic Pesticides (IUCN Task Force) reviewed approximately 800 peer-reviewed articles on the neonicotinoids, plus fipronil. Their report, entitled the "Worldwide Integrated Assessment on Systemic Pesticides" key findings include: [14]

  --Neonicotinoids are present in the environment "at levels that are known to cause lethal and sublethal effects on a wide range of terrestrial (including soil) and aquatic microorganisms, invertebrates and vertebrates."
  --The active ingredients persist, particularly in soils, with half-lives of months and, in some cases, years, and they accumulate. This increases their toxicity by increasing the duration of exposure of non-target species.
  --The metabolites of neonicotinoids are often as or more toxic than the active ingredients.
  --Typical measurements used to assess the toxicity of a pesticide (short-term lab toxicity results) are not useful for systemic pesticides and conceal the true impacts. They measure direct acute effects rather than chronic effects via multiple routes of exposure. The weight of the published evidence is very strong that the acute and chronic effects

[12] *Hoffmann, E and Castle, S. 2012. Imidacloprid in Melon Guttation Fluid: A Potential Mode of Exposure for Pest and Beneficial Organisms. J Economic Entomology 105(1):67-71.*
[13] Reetz, J. et al. 2011. Neonicotinoid insecticides translocated in guttated droplets of seed-treated maize and wheat: a threat to honeybees? *Apidologie* 42(5): 596-606.
[14] Van der Sluijs JP, et al. 2014. Conclusions of the Worldwide Integrated Assessment on the risks of neonicotinoids and fipronil to biodiversity and ecosystem functioning. Environ Sci Pollut Res. doi:10.1007/s11356-014-3229-5

pose a serious risk of harm to colonies/populations of honey bees, bumblebees and other pollinators.
--The most affected group of species include soil invertebrates and insect pollinators, with high exposure through air and plants and medium exposure through water. Invertebrates exposed to contaminated pollen, nectar and fluids are harmed at "field-realistic" concentrations.

- One study, "Macro-Invertebrate Decline in Surface Water Polluted with Imidacloprid".10 This comprehensive study concludes, "While a large amount of evidence exists from laboratory single species and mesocosm experiments, our study is the first large scale research based on multiple years of actual field monitoring data that shows that neonicotinoid insecticide pollution occurring in surface water has a strong negative effect on aquatic invertebrate life, with potentially far-reaching consequences for the food chain and ecosystem functions."[15]

- A Hallman et al. study, "Declines in Insectivorous Birds are Associated with High Neonicotinoid Concentrations," published in Nature, found that commonly-detected levels of imidacloprid in the Netherlands's surface water is correlated with 3.5% annual decline in bird populations. They assessed other likely contributing factors and found imidacloprid contamination was the key to the declines.[16]

- A recent review by Morrissey et al., which builds on previous work, finds that neonicotinoid concentrations detected in aquatic environments pose risks to aquatic invertebrates and the ecosystems they support. [17]

- A review by Goulson also notes that the prophylactic use of broad-spectrum pesticides goes against the long-established principles of integrated pest management (IPM), leading to environmental concerns, and that reported levels in soils, waterways, field margin plants and floral resources overlap substantially with concentrations that are sufficient to control pests in crops, and commonly exceed the LC_{50} (the concentration which kills 50% of individuals) for beneficial organisms. Concentrations in nectar and pollen in crops are sufficient to impact substantially on colony reproduction in bumblebees.[18]

- New research by the U.S. Geological Survey (USGS), also documents similar risks from neonicotinoids in the rivers and streams of the Midwest.[19]

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[15] Van Dijk TC, et al. 2013. Macro-invertebrate decline in surface water polluted with imidacloprid. PLoS ONE 8(5): e62374. doi:10.1371/journal.pone.0062374

[16] Hallmann CA, et al. 2014. Declines in insectivorous birds are associated with high neonicotinoid concentrations. Nature doi:10.1038/nature13531

[17] Morrissey, C. et al. 2015. Neonicotinoid contamination of global surface waters and associated risk to aquatic invertebrates: A review. Environment International. doi:10.1016/j.envint.2014.10.024.)

[18] Goulson, D. 2013. REVIEW: An overview of the environmental risks posed by neonicotinoid insecticides. Journal of Applied Ecology. 50: 977–987. doi: 10.1111/1365-2664.12111.)

[19] Hladik ML, et al. 2014. Widespread occurrence of neonicotinoid insecticides in streams in a high corn and soybean producing region, USA. Env. Poll. 193:189-196

Potential Human Health Impacts

- In 2013, the European Food Safety Authority (EFSA) published an opinion report linking two neonicotinoids to adverse effects on the developing human nervous system. This agency reviewed the study by Kimura-Kuroda et al., which found that neonics exert excitatory effects on mammalian nAChRs at concentrations greater than 1 µM. EFSA's panel of experts concluded that "acetamiprid and imidacloprid may adversely affect the development of neurons and brain structures associated with functions such as learning and memory." According to the report, data suggests that the neonicotinoid chemicals under review are responsible for the excitation or desensitisation or both of nicotinic acetylcholine receptors (nAChRs), which may affect the developing mammalian nervous system, as is known to occur with nicotine. Specifically, the report states,[20]

  "Evidence of effects on offspring from a developmental neurotoxicity study for imidacloprid in rats, submitted within the EU and the US-EPA assessment frameworks, reported decreased pup body weights, reduced motor activity level and changes in dimensions of brain structures (reduction in the thickness of corpus callosum and a decreased width of caudate/putamen). The [developmental neurotoxicity] DNT study for acetamiprid, carried out within the US-EPA assessment framework, showed decreased pup body weights, reduced pup pre-weaning survival and decreased maximum auditory startle response. The Panel therefore concludes that the two neonicotinoid compounds may affect neuronal development and function."

- Other preliminary studies have found that gestational exposure to a single large, nonlethal, dose of the neonic, imidacloprid, can produce significant neurobehavioral deficits, and may even impact sperm fertilization ability, as well as embryonic development, and general suppression of testicular function.[21]

In light of harm to humans and non-target species, EPA should immediate open this decision to public comment and review, so that the agency may fully understand whether there is "scientific data on, or other relevant information on this pesticide indicate that the residues are not safe."

The public has a right to weigh in on decisions of this magnitude and EPA has a responsibility to consider the full range of experience among the agricultural community –including those who have adopted practices not reliant on toxic pesticides– and the science associated with the hazards of clothianidin. The protection of pollinators (bees, butterflies, and birds), biodiversity, and people requires EPA to consider the full range of public input.

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[20] EFSA Panel on Plant Protection Products and their Residues (PPR). Scientific Opinion on the developmental neurotoxicity potential of acetamiprid and imidacloprid. EFSA Journal 2013;11(12):3471. doi:10.2903/j.efsa.2013.3471; and Kimura-Kuroda J, Komuta Y, Kuroda Y, Hayashi M, Kawano H (2012) Nicotine-Like Effects of the Neonicotinoid Insecticides Acetamiprid and Imidacloprid on Cerebellar Neurons from Neonatal Rats. PLoS ONE 7(2): e32432. doi:10.1371/journal.pone.0032432

[21] Abou-Donia, Larry B. Goldstein, Sarah Bullman, et al. 2008. Imidacloprid Induces Neurobehavioral Deficits and Increases Expression of Glial Fibrillary Acidic Protein in the Motor Cortex and Hippocampus in Offspring Rats Following in Utero Exposure. Journal of Toxicology and Environmental Health, Part A: Current Issues . 71(2): 119-130; and Gu YH, Li Y, Huang XF, et al. 2013. Reproductive effects of two neonicotinoid insecticides on mouse sperm function and early embryonic development in vitro. *PLoS One.* 8(7):e70112