DEPARTMENT OF ZOOLOGY

☐ ZOOLOGY RESEARCH BUILDING
1117 W. JOHNSON ST.

☐ BIRGE HALL
430 LINCOLN DR.

☐ LOWELL E. NOLAND HALL
250 N. MILLS ST.

UNIVERSITY OF WISCONSIN - MADISON

December 9, 2015

Stephen Brown
Assistant Director, Plant Health and Pest Preventions Services, Room 220
1220 N St.
Sacramento, CA 95814

Re: Public Comment for Japanese Beetle Scientific Advisory Panel – Please distribute this letter to all SAP panelists

Dear Mr. Brown,

I, Warren P. Porter, am a Professor of Zoology and a Professor of Environmental Toxicology at the University of Wisconsin, Madison. I received a B.S. in Zoology from the University of Wisconsin, Madison; an M.A. in Ichthyology from the University of California, Los Angeles; and a Ph.D. in Physiological Ecology from University of California, Los Angeles.

I have conducted scientific research and published peer reviewed articles on pesticide mixtures at low concentrations for more than 25 years. I regularly lecture and teach at the University about the real biological consequences of pesticide exposures and how they affect neurological, endocrine, immune, developmental, and genetic processes in animals and humans at very low concentrations in the parts per billion to parts per trillion concentration range, which is where concentrations of natural hormones operate in fetal and adult organisms. I have personal knowledge of the facts stated herein and could and would competently testify to them.

Repeated and heavy spraying of pesticides in the environment can play a role in promoting inflammatory diseases [1], and have devastating effects on ecological systems, harming a wide-range of organisms from microscopic communities to macro flora and fauna such as birds, mammals, and aquatic organisms [2-15]. Adverse impacts on these organisms are associated with exposure to the chemicals currently being used by the California Department of Food and Agriculture (CDFA): imidacloprid, carbaryl, and cyfluthrin. Additionally, the benefits of pesticide use, in relation to their risks, in controlling pests has been questioned [16]. Ignoring these impacts puts both the environment and human health at risk.

It is important to understand the neuroendocrine immune connection present in vertebrates and invertebrates and the impact that pesticides such as the three listed above can have not only on neurological, but endocrine, immune, epigenetic and developmental, physiological, ecological, and ecosystem processes, as well as the fundamental energetics that permit survival, growth and reproduction[2]. As described by Demas et al. and others, the neurological, immune, and epigenetic effects of these chemicals impact human and animal health and development.

MADISON, WI 53706 TELEPHONE (608) 262-1051 FAX (608) 262-9083

We can expect that beneficial insects, worms and caterpillars, and the urban bird population are experiencing changes in their cellular maintenance processes, their growth and development processes, their reproduction and immune function processes, and their normal behavior and foraging capabilities. Since these processes also apply to mammals including humans, especially children, there are ample reasons to suspend the spray operations currently in process.

The destruction of natural predators and ecosystem functions because of exposure to the three chemicals employed in the spray program should also create vacancies in the ecosystem structure that would make the local ecosystems vulnerable to invasion by a variety of invasive species. There should be monitoring for environmental impacts and human impacts, especially neurological, endocrine and immune functions. Urine samples should be collected from children to determine whether these pesticides are entering their bodies, which is likely, given the surfactants and nonionic solvents typically present in pesticide mixtures.

Chemical information

Imidacloprid

Imidacloprid belongs to the nicotinoid chemical family, a family of chemicals similar to the tobacco chemical, nicotine. It works by interfering with the transmission of stimuli in the insect nervous system causing irreversible blockage of acetylcholine receptors, which are found broadly throughout the animal kingdom. These receptors are rendered incapable of receiving acetylcholine molecules (an important neurotransmitter) and an accumulation on of acetylcholine occurs, resulting in the insect's paralysis and eventual death. It is effective on contact and via stomach action.

In soil, imidacloprid has the ability to readily leach due to its high water solubility and its inability to adhere to soil particles. Imidacloprid persists for long periods of time in soil. Several soil half-lives have been reported for imidacloprid under various soil conditions ranging from 27-229 day [17]. Imidacloprid is also persistent in groundwater. Groundwater tests in the state of New York have detected imidacloprid at concentrations ranging from less than 0.1 ppb to 1.0 ppb. The State of California has placed imidacloprid on the Ground Water Protection List due to its potential to contaminate groundwater [17].

The effects of imidacloprid on biodiversity have been well-documented. Imidacloprid applied to the soil around trees has been found to cause adverse effects on litter dwelling earthworms [18]. Fallen leaves from imidacloprid-treated trees were found to result in reductions in feeding rates by insects and earthworms, inhibition of aquatic and terrestrial microbial decomposition activity, and a subsequent decrease in leaf decomposition [19]. There is a rapidly growing body of science that links the bee decline to exposure to imidacloprid and other neonicotinoids. Field-realistic exposure to imidacloprid has been found to result in a reduction in bumblebees' ability to gather food [20]. Similarly, exposure to imidacloprid can lead to both acute and chronically impaired pollen foraging performance, resulting in effects that include a decrease in pollen foraging efficiency and a change in flower preference and/or impairment in ability to find flowers in exposed

bumblebees. These findings contrasted starkly with unexposed colonies, which improved their pollen foraging performance [21]. In a 2014 study published in the journal *Nature*, bird populations were found to be negatively correlated with surface water concentrations of imidacloprid [22].

Carbaryl

Carbaryl, a carbamate, is a contact nerve toxin that inhibits the enzyme acetylcholinesterase, with a resulting disruption of nerve impulse transmissions. Although carbaryl has low volatility due to its low vapor pressure, it may become airborne from sorption to particulates or as a spray drift immediately following application. Carbaryl is also moderately soluble in water, and concentrations have been detected in surface waters near both agricultural and urban areas in 42 states. Carbaryl has also been found in the groundwater of several states [23].

Carbaryl causes an array of serious neurotoxic effects in animals, including irreversible neurological damage and behavioral disturbances. Carbaryl and its formulation are highly toxic to bees [24-25]. In a 2009 study, carbaryl was found to contaminate bee pollen at 1 ppm levels [26]. Under realistic conditions of increased exposure times and low concentrations, predator-induced stress makes carbaryl more toxic, becoming 2 to 4 times more lethal, to gray tree frog tadpoles [27]. It is highly toxic to aquatic invertebrates, such as Daphnia (the common water flea) [28-29]. Carbaryl is moderately toxic to fish, and may also bioaccumulate [30].

Cyfluthrin

Cyfluthrin, a broad-spectrum synthetic pyrethroid insecticide, structurally resembles the organochlorine DDT more than other pyrethroids. Like DDT, cyfluthrin rapidly accumulates in fatty tissues, including the central nervous system and persists in the environment. In addition, cyfluthrin causes reproductive problems. The DDT-like pesticide causes repetitive discharge and strong excitatory action on the central nervous system, peripheral nerves and skeletal muscle fibers, by interfering with axonal sodium and potassium channels, as does DDT [31-33]. According to EPA, cyfluthrin is moderately persistent and, therefore, available for runoff through erosion as well as spray drift [34].

Cyfluthrin is clearly highly toxic in acute animal studies. EPA states that cyfluthrin and *beta*-cyfluthrin are moderately toxic to mammals and highly toxic to terrestrial invertebrates on an acute basis. Furthermore, according to EPA, both chemicals are classified as very highly toxic to aquatic organisms [34]. It caused changes in a wide variety of organs such as submaxillary gland, liver, adrenal, spleen, and ovary in rats [35]. Hazardous effects on the testes of Swiss albino rats have also been recorded [36].

Conclusion

There is a litany of adverse effects of imidacloprid, carbaryl, and cyfluthrin on the environment. These chemicals can seriously alter and harm the environment in which they are used. As illustrated above, these impacts are diverse and broad ranging encompassing molecular, epigenetic, neuroendocrine, immune, neurological,

developmental, physiological and ecological and ecosystem processes. These effects are common to invertebrate and vertebrate lifeforms as illustrated in the article by Demas et al., (2011) and other published research cited here. There is ample information in the current peer-reviewed literature to seriously question the use of these chemicals in the environment in terms of both their safety and efficacy.

Warren Porter
Prof. of Zoology and former chair
Prof. of Environmental Toxicology
Invited Affiliate Faculty Member, Engineering Physics
http://www.zoology.wisc.edu/faculty/Por/Por.html
University of Wisconsin, Madison

cc: Karen Ross, Secretary of CDFA
Matthew Rodriquez, Secretary for Environmental Protection
Gina Solomon, Deputy Secretary for Science and Health, CalEPA
Brian R. Leahy, Director of Pesticide Regulation
Rick Kreutzer, Chief, Division of Environmental and Occupational Disease Control

Works Cited

1. Straub, R.H., et al., Energy regulation and neuroendocrine–immune control in chronic inflammatory diseases. *Journal of Internal Medicine*, 2010. 267(6): p. 543-560.
2. Demas, G.E., S.A. Adamo, and S.S. French, Neuroendocrine- immune crosstalk in vertebrates and invertebrates: implications for host defence. *Functional Ecology*, 2011. 25(1): p. 29-39.
3. Eskandari, F., J.I. Webster, and E.M. Sternberg, Neural immune pathways and their connection to inflammatory diseases. *Arthritis Research and Therapy*, 2003. 5(6): p. 251-265.
4. Taub, D.D., Novel connections between the neuroendocrine and immune systems: the ghrelin immunoregulatory network. *Vitamins & Hormones*, 2007. 77: p. 325-346.
5. Alaux, C., et al., Interactions between Nosema microspores and a neonicotinoid weaken honeybees (Apis mellifera). *Environmental Microbiology*, 2010. 12(3): p. 774-782.
6. Balani, T., S. Agrawal, and A. Thaker, Effects of imidacloprid a neonicotinoid insecticide on the immune system of white leghorn cockerels. *Journal of Veterinary Pharmacology and Therapeutics*, 2008. 7: p. 27-30.
7. Gawade, L., et al., A detailed study of developmental immunotoxicity of imidacloprid in Wistar rats. *Food and Chemical Toxicology*, 2013. 51: p. 61-70.
8. James, R. and J. Xu, Mechanisms by which pesticides affect insect immunity. *Journal of Invertebrate Pathology*, 2012. 109(2): p. 175-182.
9. Mason, R., et al., Immune suppression by neonicotinoid insecticides at the root of global wildlife declines. *Journal of Environmental Immunology and Toxicology*, 2013. 1(1): p. 3-12.
10. Rondeau, G., et al., Delayed and time-cumulative toxicity of imidacloprid in bees, ants and termites. *Scientific reports*, 2014. 4.

11. Bal, R., et al., Insecticide imidacloprid induces morphological and DNA damage through oxidative toxicity on the reproductive organs of developing male rats. *Cell Biochemistry and Function*, 2012. 30(6): p. 492-499.
12. Bhaskar, R. and B. Mohanty, Pesticides in mixture disrupt metabolic regulation: In silico and in vivo analysis of cumulative toxicity of mancozeb and imidacloprid on body weight of mice. *General and Comparative Endocrinology*, 2014. 205: p. 226-234.
13. Ibrahim, K.A., et al., Imidacloprid and/or Esfenvalerate Induce Apoptosis and Disrupt Thyroid Hormones in Neonatal Rats. *Global Journal of Biotechnology & Biochemistry*, 2015. 10(3): p. 106-112.
14. Kapoor, U., M. Srivastava, and L. Srivastava, Toxicological impact of technical imidacloprid on ovarian morphology, hormones and antioxidant enzymes in female rats. *Food and Chemical Toxicology*, 2011. 49(12): p. 3086-3089.
15. Vohra, P., K.S. Khera, and G.K. Sangha, Physiological, biochemical and histological alterations induced by administration of imidacloprid in female albino rats. *Pesticide Biochemistry and Physiology*, 2014. 110: p. 50-56.
16. Pimental, D., Amounts of pesticides reaching target pests: Environmental impacts and ethics. *Journal of Agricultural and Environmental Ethics*, 1995. 8(1): p.17-29.
17. Fossen, M., Environmental Fate of Imidacloprid. California Department of Pesticide Regulation, 2006.
18. Kreutzweiser, D.P., et al., Effects of litter-dwelling earthworms and microbial decomposition of soil-applied imidacloprid for control of wood-boring insects. *Pest Management Science*, 2007. 34(2): p. 112-118.
19. Kreutzweiser, D.P., et al., Are Leaves that Fall from Imidacloprid-Treated Maples Trees to Control Asian Longhorned Beetles Toxic to Non-target Decomposer Organisms? *Journal of Environmental Quality*, 2008. 37(2): p. 639-646.
20. Feltham, H., K. Park, and D. Goulson, Field realistic doses of pesticide imidacloprid reduce bumblebee pollen foraging efficiency. *Ecotoxicology*, 2014. 23(3): p. 317-323.
21. Gill, R.J. and N.E. Raine, Chronic impairment of bumblebee natural foraging behavior induced by sublethal pesticide exposure. *Functional Ecology*, 2014. 28(6): p. 1459-1471.
22. Hallmann C.A., et al., Declines in insectivorous birds are associated with high neonicotinoid concentrations. *Nature*, 2014. 511(7509): p. 341-343.
23. Gunasekara, A.S., et al., Environmental Fate and Toxicology of Carbaryl. *Reviews of Environmental Contamination and Toxicology*, 2008. 196: p. 95-121.
24. Johansen, C.A. and C. Brown, Toxicity of Carbaryl-Contaminated Pollen Collected by Honey Bees. *Environmental Entomology*, 1972. 1(3): p. 385-386.
25. Akca, I., et al., Residual Toxicity of 8 Different Insecticides on Honey Bee (*Apis mellifera* Hymenoptera: Apidae). *Journal of Animal and Veterinary Advances*, 2009. 8(3):436-440.
26. Mullin, C.A., et al., High Levels of Miticides and Agrochemicals in North American Apiaries: Implications for Honey Bee Health. *PLoS ONE*, 2010. 5(3).
27. Relyea, R.A. and N. Mills. Predator-induced stress makes the pesticide carbaryl more deadly to gray treefrog tadpoles (*Hyla versicolor*). *Proceedings of the National Academy of Sciences*, 2001. 98(5): p. 2491-2496.

DEPARTMENT OF ZOOLOGY

☐ ZOOLOGY RESEARCH BUILDING
1117 W. JOHNSON ST.

☐ BIRGE HALL
430 LINCOLN DR.

☐ LOWELL E. NOLAND HALL
250 N. MILLS ST.

UNIVERSITY OF WISCONSIN – MADISON

28. Cerbin, S., et al., Combined and single effects of pesticide carbaryl and toxic *Microcystis aeruginosa* on the life history of *Daphnia pulicaria*. *Hydrobiologia*, 2010. 643(1): p. 129-138.
29. Hanazato, T., Effects of long- and short-term exposure to carbaryl on survival, growth and reproduction of *Daphnia ambigua*. *Environmental Pollution*, 1991. 74(2): p. 139-148.
30. Todd, N.E. and M. Van Leeuwen, Effects of Sevin (Carbaryl Insecticide) on Early Life Stages of Zebrafish (*Danio rerio*). *Ecotoxicology and Environmental Safety*, 2002. 53(2): p. 267-272.
31. Aldridge, W.N., An assessment of the toxicological properties of pyrethroids and their neurotoxicity. *Critical Reviews in Toxicology*, 1990. 21(2): p. 89-104.
32. Vijverberg, H.P.M., J.M.V. Der Zalm, and J.V.D. Bercken, Similar mode of action of pyrethroids and DDT on sodium channel gating in myelinated nerves. *Nature,* 1982. 295: p. 601-603.
33. Soderlund, D.M. and J.R Bloomquist, Neurotoxic actions of pyrethroid insecticides. *Annual Review of Entomology*, 1989. 34: p. 77-96.
34. U.S. Environmental Protection Agency, Cyfluthrins Summary Document Registration Review: Initial Docket. 2010.
35. Doherty, J.D., Memorandum to Gardner, T.A. Cyfluthrin (Baythroid). Request for tolerances for residues of cyfluthrin in/on cottonseed, cottonseed oil, cottonseed hulls, meat and milk. Request for registration of Baythroid 2 formulated product. U.S Environmental Protection Agency, Office of Pesticide Programs, 1985.
36. Rajawat, N.K., et al., Cyfluthrin-induced toxicity on testes of Swiss albino mice. *International Journal of Current Microbiology and Applied Sciences*, 2014. 3(3): p. 334-343.

Most sincerely,

Warren Porter
Prof. of Zoology and former chair
Prof. of Environmental Toxicology
Invited Affiliate Faculty Member, Engineering Physics
http://www.zoology.wisc.edu/faculty/Por/Por.html

MADISON, WI 53706 TELEPHONE (608) 262-1051 FAX (608) 262-9083