THE CENTER FOR FOOD SAFETY

August 28, 2012

FIFRA Scientific Advisory Panel
Environmental Protection Agency
Washington, DC
Submitted online at: www.regulations.gov

Re: Docket EPA-HQ-OPP-2012-0543, Pollinator Risk Assessment Framework.

Dear Science Advisory Panel Members,

The Center for Food Safety (CFS) is pleased to submit these comments on the "White Paper in Support of the Proposed Risk Assessment Process for Bees" (hereinafter, the "Proposed RA Process). CFS is a nonprofit organization located in Washington, DC, and San Francisco California. CFS has over 200,000 members across the country. We seek to protect human health and the environment by advocating for thorough, science-based, safety testing of new agricultural crops and products prior to any marketing of them in order to minimize negative impacts, such as increased use of pesticides and evolution of resistant pests and weeds.

We request that this comment be submitted to all of the FIFRA Science Advisory Panel (SAP) members and we request the opportunity to provide oral comments at the meeting.

General Comments

The description of the Proposed RA Process is unnecessarily verbose and numbingly redundant in places, which deters comprehension. In short, it needs dramatic editing and tightening to be more accessible and capable of use in the future.

It largely relies on complex conceptual modeling based on a large number of assumptions some of which seriously downplay the significance of various routes by which bees can be exposed to very high levels of neonicotinoid pesticides when used as seed treatment, e.g., planting dust, contaminated talc and guttation fluid. The model refers to a good deal of unpublished EPA analysis that appears rooted in studies that may no longer be current. In just the last year, a far-reaching international scientific debate has occurred over what constitutes excessively risky levels of neonicotinoid insecticides in pollen, nectar, dust and other routes of exposure. The

continuing danger is that incorrect conceptual modeling may result in decisions later shown after the fact to expose bees to excessive risk. Given the known risks to bees already occurring particularly from persistent, systemic insecticides such as the neonicotinoids, the SAP should place **much greater emphasis on reliance on broadly representative, multi-year, controlled field tests**, to provide realistic data points from which to judge an insecticide's impacts on both honey bees and a broadly representative suite of native bees before an insecticide is approved for sale across the country.

The additional field testing called for in a revised EPA RA Process should be national in scope, across a wide range of agricultural settings, using common combinations of crops, machinery, spray adjuvants and other factors. Actual risks to bees from applied insecticides will be influenced by the make-up of the crops involved, which may include genetically engineered insecticidal, fungicidal and other properties that may synergize with the applied insecticides. Ignoring the potential added jeopardy to bees in real world agronomic settings is too risky. Broader field testing will tease out risk factors as well as help to either validate or invalidate the conceptual models used.

The SAP should note EPA's approach to date has been the opposite of precautionary, by which thiamethoxam, clothianidin and other neonicotinoid pesticides have been conditionally registered without adequate pollinator field studies to show they would not have unreasonable adverse effects to bees. In a vast and extremely risky experiment EPA has allowed neonicotinoids to be used on close to 200 million acres and dozens of different crops with no adequate assessment framework in place. This fact is to a large extent driving the current FIFRA SAP process and the SAP must take the existing baseline risk into account as it advises the EPA.

In 2012, independent researchers, such as Henry et al., have conducted the sort of in-depth, controlled pollinator field tests that EPA has failed to obtain earlier from the neonicotinoid product registrants themselves.[1] Sublethal exposure of honey bees to thiamethoxam at field-realistic levels was shown, in this seminal French study that received world-wide attention, to cause high mortality due to homing failure at levels that could put a colony at risk of collapse. Despite severe questioning by Bayer CropScience and others, both independent and European Food Safety Agency (EFSA) reviews have confirmed that the thiamethoxam levels used in the Henry et al. study were field-relevant. That study, as well as other new science developments and bee kill incidents, led the Agriculture Ministry in France to suspend its prior approval of

[1] Henry, M., Beguin, M., Requier, F., Rollin, O., Odoux, J.-F., Aupinel, P., Aptel, J., Tchamitchian, S. and Decourtye, A. (2012). A common pesticide decreases foraging success and survival in honey bees. *Sciencexpress* 1215039

thiamethoxam products as seed treatments on oilseed crops, on June 24 of this year.[2] The Ministry did this notwithstanding detailed submissions by Syngenta arguing against the suspension.

The Henry et al. results above are not substantively addressed in the Proposed RA Process, with no explanation for their omission. The SAP should correct that.

Specific Comments on the Proposed RA Process Document

p. 45 – 2.2.1 – Management Goals and Assessment Endpoints

This section of the document includes this assertion when addressing studies reported in the open literature that described "a broad array of endpoints":

> *These include alterations to biochemical pathways (molecular changes), effects on feeding behavior (e.g., proboscis extension reflex), and alterations in immune response of individual bees; however, no information may be available from these studies on subsequent effects on survival, growth or reproduction and* ***frequently assumptions are made regarding potential relationships.*** (emphasis added).

This is a key assertion that serves to downplay numerous studies – some very recent, such as Henry et al. – that described sub-lethal effects of neonicotinoid insecticides on honey bees and bumble bees. The RA Process document does not cite the studies for which it asserts "no information may be available on subsequent effects" and "frequently assumptions are made". If the RA document authors have disregarded particular studies – which clearly they have - it would be useful if they cited to the studies rather than making blanket assertions about them.

For example, the RA Process ignores a key recent study showing that neonicotinoid exposures make bees more vulnerable to highly-damaging introduced parasites, such as the genus of gut parasites, *Nosema* spp. The study, Pettis *et al.,* led by the USDA Agricultural Research Station in Beltsville, Maryland, demonstrates a link between these two bee hazards.[3] The abstract, states (emphasis added):

---

[2] Official Gazette No 0172 of 26 July 2012, p. 12 246, Order of 24 July 2012 on the prohibition of use and placing on the market for use in the national territory of seeds of oilseed crucifers treated with plant protection products containing thiamethoxam NOR: AGRG1230159A . Online at: www.legifrance.gouv.fr/affichTexte.do;jsessionid=B6BA3FD207F4511CFC5DE479AD94C239.tpdjo08v_2?cidTexte=JORFTEXT000026223233&dateTexte=20120814.

[3] Jeffery S. Pettis et al., Pesticide exposure in honey bees results in increased levels of the gut pathogen *Nosema*, *NATURWISSENSCHAFTEN* (2012) DOI: 10.1007/s00114-011-0881-1; see also Cyril Vidau et al., Exposure to sublethal

> *[W]e exposed honey bee colonies during three brood generations to sub-lethal doses of a widely used pesticide, imidacloprid, and then subsequently challenged newly emerged bees with the gut parasite, Nosema spp. The pesticide dosages used were below levels demonstrated to cause effects on longevity or foraging in adult honey bees.* ***Nosema infections increased significantly in the bees from pesticide-treated hives when compared to bees from control hives demonstrating an indirect effect of pesticides on pathogen growth in honey bees****. We clearly demonstrate an increase in pathogen growth within individual bees reared in colonies exposed to one of the most widely used pesticides worldwide, imidacloprid, at below levels considered harmful to bees. The finding that individual bees with undetectable levels of the target pesticide, after being reared in a sub-lethal pesticide environment within the colony, had higher Nosema is significant.* ***Interactions between pesticides and pathogens could be a major contributor to increased mortality of honey bee colonies, including colony collapse disorder, and other pollinator declines worldwide.***

The SAP must ensure consideration of these interactions is incorporated into the RA Process.

pp. 47- 49 – Foliar Spray Applications and p. 57 - Characterization of Exposure

For sprayed pesticides in all sections of the document, there is no mention of this recent paper and its key findings, "Learning impairment in honey bees caused by agricultural spray adjuvants," which states (emphasis added):[4]

> ***Background***
> *Spray adjuvants are often applied to crops in conjunction with agricultural pesticides in order to boost the efficacy of the active ingredient(s). The adjuvants themselves are largely assumed to be biologically inert and are therefore subject to minimal scrutiny and toxicological testing by regulatory agencies. Honey bees are exposed to a wide array of pesticides as they conduct normal foraging operations, meaning that they are likely exposed to spray adjuvants as well. It was previously unknown whether these agrochemicals have any deleterious effects on honey bee behavior.*
>
> ***Methodology/Principal Findings***
> *An improved, automated version of the proboscis extension reflex (PER) assay with a high degree of trial-to-trial reproducibility was used to measure the*

doses of fipronil and thiacloprid highly increases mortality of honeybees previously infected by *Nosema ceranae*, 6 *PLoS ONE* e21550.

[4] Ciarlo TJ, Mullin CA, Frazier JL, Schmehl DR (2012) Learning impairment in honey bees caused by agricultural spray adjuvants. *PLoS ONE* 7(7): e40848. doi:10.1371/journal.pone.0040848

olfactory learning ability of honey bees treated orally with sublethal doses of the most widely used spray adjuvants on almonds in the Central Valley of California. Three different adjuvant classes (nonionic surfactants, crop oil concentrates, and organosilicone surfactants) were investigated in this study. Learning was impaired after ingestion of 20 µg organosilicone surfactant, indicating harmful effects on honey bees caused by agrochemicals previously believed to be innocuous. Organosilicones were more active than the nonionic adjuvants, while the crop oil concentrates were inactive. Ingestion was required for the tested adjuvant to have an effect on learning, as exposure via antennal contact only induced no level of impairment.

Conclusions/Significance

A decrease in percent conditioned response after ingestion of organosilicone surfactants has been demonstrated here for the first time. Olfactory learning is important for foraging honey bees because it allows them to exploit the most productive floral resources in an area at any given time. Impairment of this learning ability may have serious implications for foraging efficiency at the colony level, as well as potentially many social interactions. ***Organosilicone spray adjuvants may therefore contribute to the ongoing global decline in honey bee health.***

The SAP should take these findings into account as they are particularly relevant when conducting field studies associated with sprayed pesticides, as well as in accurate risk modeling. The current RA Process document does not mention adjuvants.

pp. 98 – 99 – Contact with Dust

The significance of planting dust and talc contaminated via neonicotinoid seed treatments is downplayed without explanation. The document gives short shrift to quantitative modeling of their impacts. This is despite the vast number of bee kill incidents attributable to this exposure route. Regulatory agencies in Germany, Italy, France and Slovenia have prohibited various seed treatment uses largely due to this pathway and the typically hundreds or even thousands of bee colony deaths associated with it.

If modeling of this route is not feasible, then the answer is to do extensive additional field studies to determine its significance. If the SAP does not recommend that, then it is commonly accepted that in doing a risk assessment with an element involving high uncertainty, the conservative approach (which the RA Process document repeatedly purports to follow) is to consider a reasonable "worst case" description of that element. **The answer is definitely not to take the evasive, optimistic and self-serving tack that EPA has taken to this issue and to avoid**

further mention of Tier II or III field studies on bee poisoning via contaminated dust/talc, as appears to be the aim of the document.

At p. 99, the Proposed RA document refers to various claimed possible technical fixes as justification for the fact that the current risk modeling does not account for this route of neonicotinoid exposure. EPA seeks to steer the focus toward undefined "future assessment methods of this route". EPA claims it is:

> *….working with pesticide registrants, seed treatment companies and seeding equipment manufacturers to better ensure the development and use of appropriate sticking agents, seeding equipment lubricating agents, and more effective venting/filtering options for such equipment to minimize the evolution of dust at the time of planting so that exposure to honey bees through contaminated dust is minimized.*

However, the findings of Tapparo et al. demonstrate the equipment modifications only have a "limited effect".[5] Krupke et al., who were very clear that they followed the EPA's label directions for seed treatment use, confirm Tapparo et al. on the point that the toxic dust from planting coated seeds will escape in any event.[6] This is regardless of the current label warnings, directions for use or other farming techniques that EPA may urge. It is disingenuous for EPA to imply it can minimize this exposure route – with no stated time frame - as it is in no position to mandate or realistically enforce any "venting/filtering" modifications to the tens of thousands of seed planters across the United States that would have to be modified in order for this route to be addressed, in as these coated seeds now are virtually ubiquitous. Further, it is common knowledge that actual field enforcement of EPA's label restrictions and directions for use is extremely rare. **No basis exists for the SAP to defer to EPA's unrealistic optimism.**

p. 100 – Consumption of Contaminated Drinking Water

In dismissing further consideration of contaminated guttation fluid, the document states (emphasis added):

> *If bees drink a substantial amount of water from guttation fluid or dew, conservative exposures may be similar to or even exceed pesticide exposures through the diet or direct spray. Because this indicates a potential concern for assessing exposures of honey bees to pesticides, potential exposures through drinking dew and guttation fluid were investigated further. This investigation*

[5] Tapparo,A, Marton,D et al. 2012. Assessment of the environmental exposure of honeybees to particulate matter containing neonicotinoid insecticides coming from corn coated seeds**.** *Environmental Science & Technology* 46(5): 2592-2599.

[6] 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.

> *concluded that pesticide exposures through dew and guttation fluid are not expected to be as significant when compared to diet because of two primary reasons. First, although the importance of dew and guttation fluid to bees as a source of drinking water is unknown, dew and* ***guttation fluid are only expected to be present during a portion of the morning which would prevent bees from drinking a substantial amount of water from these sources****. Second, for many worker bees, pesticide doses through consumption of dew and guttation fluid may be much less due to lower or non-existent drinking water consumption rates (because of higher amounts of water consumed through food). Therefore, pesticide exposure through drinking water is not included in the proposed Tier I exposure route for bees.*

Similar to the way the RA Process appears to address, but actually evades, analysis of the toxic dust route of exposure, the above "investigation" is a sham. It ignores this observation at p. 214 in Appendix 2:

> *……pesticide exposures through drinking guttation fluid of crops that received treatments of pesticides may be substantial for bees.*

The text on p. 100 literally leaps to the conclusion that because guttation fluid may only be present "during a portion of the morning" it will not cause a significant exposure. This is contradicted elsewhere in the document by the authors' summary of Girolami et al.[7], on p. 249 in Appendix 4, "Summaries of empirical studies from the scientific literature that were used to evaluate Tier I methods for estimating pesticide exposure" (emphasis added):

> *Girolami et al. 2009 quantified imidacloprid, clothianidin, thiamethoxam, and fipronil residues in guttation fluid. Guttation fluid was collected from corn seedlings grown in the field from seeds coated with imidacloprid (Gaucho® 0.5 mg a.i./seed), clothianidin (Poncho® 1.25 mg a.i./seed), thiamethoxam (Cruiser® 1.0 mg a.i./seed), and fipronil (Regent® 1.0 mg a.i./seed). Guttation fluid was collected in the field from 8-9 a.m. daily until a volume of 5 mL was reached, beginning at seedling emergence and continuing for the first 3 weeks after emergence. Concentrations of chemicals in guttation fluid were measured using high-performance liquid chromatography (HPLC).* ***In corn, it was observed that guttation fluid can flow down the plant into the crown cup and persist throughout the day.***

In addition to being incorrect on the "morning only" availability of guttation fluid, the text on p. 100 ignores whether the portion of water that bees do get from this source can be so contaminated with a neonicotinoid that it is toxic, even if it is only a part of the bee's daily

[7] Girolami, V., L. Mazzon, A. Squartini, N. Mori, M. Marzaro, A. Di Bernardo, M. Greatti, C. Giorio, and A. Tapparo. 2009. Translocation of neonicotinoid insecticides from coated seeds to seedling guttation drops: a novel way of intoxication for bees. *Journal of Economic Entomology* 102(5): 1808-1815.

water consumption. The document text is inconsistent with EPA and Bayer's prior treatment of this topic as stated in an internal EPA Memorandum concerning "Clothianidin Registration of New Products Sepresto [etc.]" dated Sept. 15, 2009.[8] The Memo refers to a Bayer-reported incident (MRID 477987-01) conducted in Austria, in which bees were exposed to guttation fluid from clothianidin seed-treated corn seedlings. The EPA summary of the incident recounts guttation fluid as a potentially deadly exposure route:

> *Clothianidin residue levels in bees were greater than the level of quantification up to 14 days after seedling emergence; in bees without alternative water supply,* [that is, other than the guttation fluid] *honeybee mortality appeared to be correlated with clothianidin residues.*

The SAP should insist on real analysis of this route rather than conveniently dismissing its risk via a pseudo-investigation. Again, if the information is lacking about the full extent of bee mortality associated with it, more semi-field or field studies must be called for comparable to that reported by Bayer from Austria, above. In the interim, a conservative modeling approach is to include a reasonable "worst case" description of this element. The answer is not to avoid further mention of Tier II or III field studies on poisoning via guttation fluid, as appears to be the aim of the document.

p. 100 – Tier II Exposure Assessment for Honey Bees

As indicated in the toxic dust and dew/guttation water exposure routes, a conservative and reasonable approach to address uncertainties is not disregarding them, rather it is to include them as elements in the field studies called for under Tiers II and III in the RA process. The discussion at the bottom of p. 100 ignores these exposure routes, but this sentence at the top of p. 101 clearly should be seen by the SAP applicable to them:

> *The strength of basing the Tier II exposure approach on empirical data from field studies is that some of the uncertainties associated with the Tier I exposure method are reduced or eliminated.*

pp. 113-116 – 4.1.2.1.2 Sublethal Effects

This is an extremely disappointing and didactic section of the document. It contains at least quadrupally repeated statements on how difficult it is to connect some sublethal effects with assessment endpoints, with no discussion of which, if any, sublethal effects <u>do</u> relate to assessment endpoints based on the extensive science developed on this question over the last

[8] EPA OPP Memorandum from Jose L. Menendez, Chemist, et al. to Kable Davis, Risk Manager Reviewer, et al., dated 9/15/09.

several years. It is not a useful up-to-date assessment of the science in this area, despite the agencies taking years to get to this point and the hundreds of pages in the document.

It also avoids the obvious conclusion that if it is difficult to connect sublethal effects observed in laboratories to "field relevant" situations, then the answer is to require more detailed Tier II and III field studies that tease out the relevance of different effects, e.g., Whitehorn et al., Henry et al., and so on, before approving pesticides.

The key findings of another more recent study also are not substantively addressed in the Proposed RA Process. "Effects of imidacloprid, a neonicotinoid pesticide, on reproduction in worker bumble bees (*Bombus terrestris*)":[9]

> *….The key result emerging from our work is that ingestion of imidacloprid at environmentally realistic levels substantively reduced the fecundity of worker bumble bees. This finding is consistent with those of previous studies, which have shown that exposure of B. terrestris workers to dietary imidacloprid at 10 ppb in feeder syrup reduced larval production by 43% (Tasei et al. 2000) and drone production by between 41 and 62% (Tasei et al. 2000; Mommaerts et al. 2010). However, wild bees are probably exposed to imidacloprid residues lower than 10 ppb when they consume the nectar and pollen of treated crops (Bonmatin et al. 2003, 2005; Chauzat et al. 2006). We have now demonstrated that dietary trace residues of imidacloprid in the range of 1 ppb can reduce worker fecundity by at least one third…..*
>
> *Our findings raise further concern about the impact of systemic neonicotinoids on wild bumble bee populations. A recent review summarising 15 years of research on the hazards of neonicotinoids to bees highlighted the sub-lethal effects of exposure in the laboratory to neonicotinoids > or = 6 ppb on reproduction and behaviour in bumble bees (Blacquie`re et al. 2012). We have now shown that dietary neonicotinoids in the range < 6 ppb can cause substantive sub-lethal effects on bumble bee reproduction.*

The SAP needs to take these findings into account, addressing serious sublethal effects of exposure levels often claimed in the past to be safe.

pp. 157-160 - 5.3 Consideration of Non-*Apis* Bees

While much of the Proposed RA is too long-winded, this section has the opposite problem. Its cursory treatment of the risks of insecticides to the ~4,000 species of native North American

[9] Laycock I, Lenthall K, Barratt AT, Cresswell JE (2012). Effects of imidacloprid, a neonicotinoid pesticide, on reproduction in worker bumble bees (*Bombus terrestris*) *Ecotoxicology* DOI 10.1007/s10646-012-0927-y

bees is unconvincing, a major failure given the stakes. Several of these species face severe declines.[10] These bees lack the carefully-bred adaptability and the resilient social structures of *Apis mellifera* and of course many have entirely different life cycles and vulnerabilities. *A. mellifera* will always survive in large numbers as a key commodity producer and agricultural "tool," whereas native species are at far higher risk.

Readers are left with no confidence that the conceptual modeling and tiered assessment approach for *Apis* will aid the conservation of the native species, many of which are essential pollinators and several of which are in jeopardy of foreseeable extinction.

This section of the Proposed RA needs dramatic bolstering. It refers in several places to possible "modifications" but it does not provide those modifications. If the regulatory agencies proceed with the current framework it appears likely that native bees, including rare and endangered species, will face continuing unassessed jeopardy from insecticides. Given that many of these native species have small, localized native ranges, the assessment process should consider the need to restrict or limit the use of the pesticides in those locations, a common-sense consideration lacking in the document. Otherwise, such exposure routes as foliar spraying and toxic dust and talc could conceivably effectively eliminate large portions of remaining populations of highly local native bees.

The section contains no reference to the key new Whitehorn et al. paper, "Neonicotinoid pesticide reduces bumble bee colony growth and queen production," which provides:[11]

> *Abstract: Growing evidence for declines in bee populations has caused great concern due to the valuable ecosystem services they provide. Neonicotinoid insecticides have been implicated in these declines as they occur at trace levels in the nectar and pollen of crop plants. We exposed colonies of the bumble bee Bombus terrestris in the lab to field-realistic levels of the neonicotinoid imidacloprid, then allowed them to develop naturally under field conditions. Treated colonies had a significantly reduced growth rate and suffered an 85 percent reduction in production of new queens compared to control colonies. Given the scale of use of neonicotinoids, we suggest that they may be having a considerable negative impact on wild bumble bee populations across the developed world.*

[10] See, for example, Elaine Evans et al., *Status Review of Three Formerly Common Species of Bumble Bee in the Subgenus* Bombus, Xerces Society (2009), *available at* www.xerces.org/wp-content/uploads/2009/03/xerces_2008_bombus_status_review.pdf .

[11] Authors: Penelope R. Whitehorn, Stephanie O'Connor, Felix L. Wackers, Dave Goulson
http://www.sciencemag.org/content/early/2012/03/28/science.1215025.abstract Science
DOI:10.1126/science.1215025

The SAP needs to ensure the well-supported findings of this study are incorporated in the RA Process.

p. 170 – Epilogue

Strangely, the final conclusions of the document refer to Figures 12 and 13 as illustrating certain concerns about interpreting field studies involving treated seeds. However, nowhere is a description or a citation given for the study the figures refer to nor is the pesticide identified. This information is needed; there is no reason apparent from the figures or discussion as to why the concerns may be generalizable.

In sum, CFS urges revision of the Risk Assessment Process according to the comments above. Finally, we note that in the past, EPA has tended to point to the multi-factor nature of bee declines and the difficulty of precisely stating the significance of each factor as excuses for not taking prompt action to reduce the harm posed by pesticides. If that continues to be EPA's approach we should expect continued broad declines of bees in the future.

Pesticides are the one factor that can be immediately changed tomorrow by an EPA decision to suspend, cancel, or deny a use that is deemed excessively harmful to bees. The benefits of precautionary decisions can be experienced by bees and beekeepers nationally, promptly and across the board, as was clearly the case in Italy after the suspension of neonicotinoid seed treatments. On June 26 of this year, Italy's Ministry of Health announced it would continue its suspension that it originally imposed in 2009 in response to bee kills that clearly resulted from the use thiamethoxam and clothianidin on corn seeds.[12] On June 27, the EFSA issued a report noting that Italy's suspension had been extremely effective in reducing bee kill incidents.[13]

Just for the year 2012 to date, the EPA has admitted it has received more than 130 bee kill incident reports (unconfirmed but under investigation) attributed to neonicotinoid pesticide

[12] Confermata La Sospensione Dei Neonicotinoidi Per La Concia Del Mais. Order of Minister of Health No. 145, dated 26 June, 2012. Online at www.mieliditalia.it/images/stories/sito/documenti/rassegna_stampa/20120626_cs_ministero_salute_145.pdf

[13] EFSA, "Assessment of the scientific information from the Italian project "APENET" investigating effects on honeybees of coated maize seeds with some neonicotinoids and fipronil". EFSA Journal 2012;10:2792. Available online at: www.efsa.europa.eu/it/efsajournal/pub/2792.htm

dust poisoning from the U.S. and Canada combined.[14] Media accounts and other information indicate that likely hundreds more bee kill incidents actually occurred this year that were not reported to EPA. The SAP should consider Italy's experience, as well as that in other countries in Europe with comparable experiences, to the high-risk situation in North America where the neonicotinoid treatment regimens continue unabated.

Please contact the undersigned if you have questions on any of these comments. We would like to provide oral comments at the SAP meeting.

Sincerely,

/s/_____

Peter T. Jenkins, Attorney for Petitioners
Center for Food Safety and International Center for Technology Assessment
660 Pennsylvania Ave., SE Suite 302
Washington, DC 20003
Phone: (202) 547-9359
Email: PJenkins@icta.org

[14] Steven P. Bradbury, EPA Office of Pesticide Programs, Letter to Peter T. Jenkins, Center for Food Safety and International Center for Technology Assessment, "Clothianidin Emergency Citizen Petition dated March 20, 2012," July 17, 2012, online at: www.epa.gov/opp00001/about/intheworks/epa-respns-to-clothianidin-petition-17july12.pdf.