

1  
2 SAINATH SURYANARAYANAN  
3 DANIEL LEE KLEINMAN  
4 **Disappearing Bees and Reluctant Regulators**  
5

6 Imagine this: You're a commercial beekeeper, who relies entirely on keeping  
7 honeybees for making a living. You head out one morning to examine your bees and find  
8 that thousands of your previously healthy hives have "collapsed" mysteriously, after your  
9 bees pollinated crops in the fields of one of the farmers with whom you contract. Your  
10 bees have abandoned their hives, and they've not returned.

11 Beginning in the winter of 2004–2005, many U.S. beekeepers, especially  
12 commercial ones, saw this happening. Several commercial beekeeping operations lost  
13 between 30 and 90% of their hives, a figure significantly higher than the roughly 15%  
14 that is common when hives are afflicted with parasitic mites or common diseases or when  
15 bees suffer from poor nutrition. Half a decade later, losses have remained troublingly  
16 high, hovering around 30% in each subsequent year.

17 Bee researchers dubbed this new phenomenon colony collapse disorder (CCD),  
18 and more than a half decade after beekeepers first saw their bees ravaged by it,  
19 controversy and uncertainty remain about what causes it. The field observations of  
20 commercial beekeepers suggest a causal role for systemic agricultural insecticides such  
21 as imidacloprid. However, the Environmental Protection Agency's (EPA's) "sound  
22 science" approach to regulation does not permit the use of informal observational data  
23 such as that gathered by beekeepers in federal rulemaking. **And traditional scientific**  
24 **research consistent with the EPA's Good Laboratory Practice policy has thus far not**  
25 **established a definitive role for imidacloprid in causing CCD.** Accordingly, the EPA  
26 has refused to take imidacloprid and other similar agrochemicals off the market.  
27 Importantly, the laboratory research on which the EPA based its determination is  
28 premised on a preference for type II (false negative) over type I (false positive) errors. A  
29 false negative result incorrectly labels as safe a substance that is dangerous; a false  
30 positive incorrectly labels as dangerous a substance that is safe. We suggest that given the  
31 commercial stakes for beekeepers and the health impacts on bees, the regulatory  
32 preference for false negative over false positive results is misguided, and serious  
33 consideration should be given to precautionary regulatory policy.

34 The term CCD was coined by bee researchers to refer to a phenomenon in which  
35 managed honeybees abandoned their colonies en masse, leaving behind the queen, young  
36 bees, and large stores of honey and pollen. CCD threatens the viability of over 90  
37 different U.S. fruit, nut, and vegetable crops, whose quantity and quality of production  
38 depend on the pollination services provided by managed honeybees. Emerging scientific  
39 investigations of CCD suggest that microbial pathogens such as viruses are causally  
40 involved. However, the fact that different studies identify different sets of associated  
41 microbial pathogens has led CCD researchers to surmise that the discovered pathogens  
42 are secondary infections. The identity of the primary causal factor(s) that render  
43 honeybees susceptible to such secondary infections is a flashpoint within and between  
44 groups of beekeepers, researchers, agrochemical representatives, regulatory officials, and  
45 environmentalists.

46 CCD was first discovered by commercial beekeepers, who travel around the  
47 country renting out their colonies for pollination purposes to farmers. Several beekeepers  
48 observed CCD unfolding in the fields of the commercial growers with whom they  
49 contract. They consistently noted connections between the occurrence of CCD and the  
50 proximity of their hives to fields treated with relatively new systemic insecticides such as  
51 the neonicotinoid imidacloprid. Affected beekeepers reported that CCD occurred in  
52 colonies several months after initial exposure to neonicotinyl insecticides. This suggested  
53 to the beekeepers that foraging bees, instead of dying immediately (as experienced in bee  
54 kills resulting from exposure to more traditional pesticides), were bringing back pollen  
55 and nectar contaminated with low levels of the systemic insecticide to the colony. This,  
56 the beekeepers surmised, had long-term progressive effects on developing bees that were  
57 chronically exposed to accumulating insecticidal stores. To date, U.S. regulators have  
58 dismissed beekeepers' on-the-ground evidence. Government officials view beekeeper  
59 evidence as anecdotal, and they will not consider it in promulgating regulations, since  
60 beekeepers do not isolate causal variables in the way done in formal laboratory and field  
61 experiments. From the perspective of many commercial beekeepers, however, with high  
62 stakes in maintaining strong and healthy colonies, their hypothesis provides sufficient  
63 justification for developing regulations that lead to limiting bee exposure to imidacloprid  
64 while more-conclusive evidence is sought. Theirs is a precautionary approach predicated  
65 on a false positive error norm.

66

#### 67 **Lab and field studies**

68 Some ecotoxicological laboratory studies of the influence of the newer systemic  
69 insecticides on honeybees have shown adverse effects that can potentially culminate in  
70 CCD. Chronic feeding of neonicotinyl insecticides to honeybees at sublethal doses  
71 comparable to levels found in pollen and nectar of treated field crops had deleterious  
72 effects on learning, memory, behavior, and longevity. Lab studies also suggest that  
73 synergistic interactions between the newer systemic insecticides and other environmental  
74 toxins and pathogens could enhance the toxicity to honey bees.

75 EPA officials recognize that these data on the ecological effects of the newer  
76 systemic toxins is a cause for some concern but maintain that it is too inconsistent to  
77 restrict the use of these toxins. And although regulatory officials point to the agency's  
78 own risk assessments conducted during the registration process in order to support the  
79 claim that these insecticides pose minimal risks to honeybees, they also acknowledge that  
80 their current risk assessments do not systematically consider the effects of either short-  
81 term or chronic exposure to sublethal doses of these insecticides on honeybees. Neither  
82 do they assess the effects of multiple interactions between insecticidal toxins and other  
83 environmental variables on honeybees. Insecticidal effects on younger honeybee brood  
84 are not part of the EPA's evaluation scheme either. In effect, the EPA's sound science  
85 approach permits the release of the newer systemic insecticides based on experimental  
86 practices that tend to ignore the findings highlighted by some laboratory- and many  
87 beekeeper-initiated studies. EPA officials note that indirect laboratory findings on  
88 individual bees do not necessarily translate to what is actually occurring to whole  
89 colonies in the field. The agency persists in demanding more direct causal experimental  
90 evidence from field studies on colonies. The direct causal experimental evidence  
91 available to date is inconclusive.

92 Experimental field studies typically impose conditions whereby one set of  
93 colonies receives no pesticide while other sets receive known doses, with other variables  
94 of interest ideally controlled. But the actual environmental settings in which commercial  
95 beekeepers work expose honeybees throughout their life cycle to a multitude of local  
96 environmental variables such as nutrition, other toxins, pathogens, and parasites, many of  
97 which are known to interact with the newer systemic insecticides. Contemporary field  
98 study designs, which tend to focus on only one or two toxins, do not test real-life  
99 scenarios in which low levels of the toxins by themselves may not cause CCD but may do  
100 so through intricate interactions with multiple other environmental variables across the  
101 life cycle. Additionally, the statistical norm for accepting field experiment findings (95%  
102 confidence that a result is not a product of chance) is an academic convention with no  
103 intrinsic justification. It is predicated on a preference for false negative conclusions, and  
104 this in turn reflects a predilection to overlook potentially valuable findings rather than  
105 suffer the embarrassment of having to withdraw results later determined to be incorrect.  
106 These are matters of social history, not nature.

107 Following this logic, field experiments tend toward finding no significant  
108 difference between pesticide-treated and untreated colonies, when in fact there might be.  
109 These historically established biases in field studies are further compounded by the fact  
110 that the EPA gives greater weight to studies that comply with the regulatory standards of  
111 good laboratory practice (GLP) than those that do not. GLP standards specify how a  
112 study should be constituted, performed, recorded, and interpreted, and by whom. In order  
113 to be GLP-compliant, an investigation has to be validated by regulatory bodies composed  
114 of academic and agrochemical company researchers. GLP requires traditional standards  
115 of isolating causal factors and establishing experimental controls. As a result, cutting-  
116 edge studies on the effects of sublethal chronic doses of the newer systemic insecticides  
117 on honeybee adults and brood, which are academically sound but have yet to be validated  
118 as GLP, are typically not considered in federal rulemaking. Moreover, the exorbitant  
119 expenditure required to meet GLP standards means that public researchers and  
120 beekeepers will have difficulty undertaking investigations that are GLP-compliant.

121 Although ecotoxicological field study designs may appear sound from the  
122 standpoint of established regulatory standards, they bear little resemblance to the reality  
123 that beekeepers and honeybees face. Consequently, we should not take their policy  
124 relevance for granted. It is time for the EPA to take seriously innovative ecotoxicological  
125 practices that push at the very limits of what is seen as experimentally feasible. Of  
126 course, because such studies will probably not be able to sharply isolate and control for  
127 the effects of the myriad factors plausibly at play in CCD, these kinds of investigation are  
128 likely to produce only suggestive results. Virtually inevitably, they will not provide the  
129 kind of unambiguous proof that the EPA's regulators demand as part of their sound  
130 science approach. Instead of dismissing such studies, however, we suggest that the CCD  
131 epidemic should prompt us to revisit the bases for pesticide regulation.  
132

### 133 **The precautionary approach**

134 Instead of a sound science approach to pesticide regulation, we advocate a broadly  
135 precautionary orientation. This entails a regulatory preference for false positives over  
136 false negatives. Regulators must accept suggestive data when all uncertainties are not  
137 resolved. Government decisionmakers would need to seriously value a much broader

138 array of knowledge forms, practices, and actors, both certified and noncertified, in  
139 discussions that frame research questions, study designs, data interpretations, and policy  
140 decisions regarding pesticides than the EPA currently considers. This approach shifts the  
141 onus of showing no harm from at-risk groups, such as commercial beekeepers, to those  
142 who produce or deploy the technology of concern, which in this case would be the  
143 manufacturers of systemic agricultural insecticides such as imidacloprid.

144 In 1999, the French government set the precautionary precedent for the regulation  
145 of newer systemic insecticides in the case of honeybee exposure. French policymakers  
146 decided to limit the use of Gaucho (imidacloprid) and Regent TS (fipronil) in the face of  
147 uncertainty surrounding the risks they pose to honeybee health. They drew on a  
148 preponderance of indirect evidence from observations in actual crop settings by French  
149 beekeepers and followup studies by researchers affiliated with the government. This  
150 research suggested that sublethal levels of the systemic insecticides were available in the  
151 pollen and nectar of treated crop plants and were retained in soils over multiple years and  
152 reentered crops during subsequent cultivations. These studies also provided evidence that  
153 chronic exposure to systemic insecticides in laboratory and semi-field settings  
154 significantly impaired honeybee foraging, learning, and longevity.

155 Advocates for the established sound science approach to pesticide regulation tout  
156 it as unbiased. In fact, all research requires choices and thus has biases. There is nothing  
157 inherently superior about type II (false negative) over type I (false positive) errors. There  
158 is nothing intrinsically better about the preference for higher levels of certainty on more  
159 narrowly construed problems as against greater uncertainty in understanding more  
160 complex relationships. These matters are value-laden, political, and in the case of CCD,  
161 they affect different stakeholders differently. The current approach to sound science–  
162 based regulation benefits the short-term interests of agrochemical producers by treating  
163 the absence of conclusive evidence of pesticide harm as justification for allowing a given  
164 chemical to remain on the market. A precautionary approach in the case of CCD, in  
165 contrast, could hurt agrochemical companies, because indirect evidence of the sublethal  
166 effects might justify removing certain systemic insecticides from the market or, more  
167 likely, restricting their use in some fashion. For commercial beekeepers, on the other  
168 hand, sound science regulatory policy in the case of CCD offers no immediate advantage.  
169 If certain agricultural systemic insecticides contribute to CCD, then beekeepers are  
170 helped by restricting bee exposure to these chemicals. If it turns out that the toxins of  
171 concern are not involved in CCD, beekeepers will be harmed less by the move to remove  
172 it from use than they would be if it transpired that they contributed to CCD, but exposure  
173 had not been restricted.

174 There are those who express fears that removing or limiting the use of the newer  
175 systemic insecticides, which are categorized by the EPA as reduced risk, would force  
176 growers to revert to older pesticides considered more harmful to human and  
177 environmental health. These fears are not entirely unreasonable, given the current  
178 structure of U.S. agriculture, with its predilection for large monoculture crops, which  
179 depend heavily on pesticides and herbicides in order to survive. Consequently, any  
180 significant reduction in the use of these insecticides will not ultimately be effective  
181 without a broader shift toward more sustainable forms of agriculture, including an  
182 increase in smaller-scale farm production, polycultures, and ecological strategies of pest  
183 management. Perhaps the case of CCD can serve as an opportunity to prompt broad

184 dialogue about the future of U.S. agriculture and lead to experiments on the advantages  
185 and drawbacks of a wide array of alternative agricultural practices.

186 At a minimum, the complicated knowledge landscape surrounding CCD should  
187 lead the EPA to consider supporting methodologically innovative research that would  
188 improve our understanding of CCD and the multitude of factors that may interact in  
189 complex ways to cause it. The decision to seek an understanding of real-world  
190 environmental complexity and not to base regulation on artificially reductive  
191 experimental designs requires different standards of statistical rigor and experimental  
192 control than those that are currently practiced. This research would monitor real-time  
193 effects on long-term colony health from chronic exposure to toxins used in commercial  
194 beekeeping and farming practices. Crucially, it would be transdisciplinary in  
195 incorporating traditional honeybee research with beekeepers' on-the-ground knowledge,  
196 along with sociologists and humanists versed in the social, economic, and political  
197 dimensions of scientific and agricultural practices.

198 More generally, the CCD case should lead us to consider the value and drawbacks  
199 of EPA's sound science approach to pesticide regulation. If sound science is not  
200 inherently superior to a precautionary approach, why should we use it? Should the federal  
201 government have regulatory policies whose scientific foundations systematically support  
202 the interests of some economic actors over others? If not, then debates that inform policy  
203 on pesticide regulation need to represent more equitably the methodological and  
204 epistemological commitments and values of a broader range of actors than what is  
205 currently occurring under the paradigm of sound science. A precautionary approach,  
206 broadly along the lines of what we have outlined, would allow scientifically justifiable  
207 and fairer means of serving environmental health and the interests of those involved in  
208 agricultural production.

209  
210  
211  
212