

Colony Collapse Disorder: Have We Seen This Before?

Robyn M. Underwood¹ and Dennis vanEngelsdorp^{1,2}

¹The Pennsylvania State University
Department of Entomology
University Park, PA 16802

²Pennsylvania Department of Agriculture
Department of Plant Industry – Apiculture
2301 N. Cameron St.
Harrisburg, PA 17110
(717)772-5225 (phone)
(717)705-6518 (fax)

“Colony Collapse Disorder” (CCD) is a new tag name presently being given to a condition that is characterized by an unexplained rapid loss of a colony’s adult population. Collapsed colonies have no or very few bees remaining, either in the dead hive or in the apiary. There are usually plenty of food stores in these colonies and if bees remain, the population consists of a queen and a small number of young workers. The stores appear to remain untouched by robbing bees or honey bee comb pests such as wax moths and small hive beetles for several weeks after the collapse.

Affected operations can be devastated by the condition. Some beekeepers have reported losses of 90% of their operation. A recent survey conducted by the Apiary Inspectors of America estimated that between 651,000 and 875,000 of the nation’s estimated 2.4 million colonies were lost over the winter of 2006 – 2007. While a majority of these losses were attributable to known bee threats, over 25% of beekeepers were considered to have CCD [1]. In the mid-Atlantic region, continuing surveys from several sources have demonstrated recurring periods of heavy winter losses. Specifically, beekeepers reported experiencing heavy losses in the spring of 2001, 2004 [2], and 2007 [3].

Large-scale losses are not new to the beekeeping industry (Table 1). Many of the symptoms similar to those expressed by CCD-affected colonies have been described before. Like today, in the past, the cause for the colony collapse has not been ascertained with certainty, although speculations as to the cause(s) are plentiful. In this paper we briefly review the past history of colony collapses that are reminiscent of the present situation.

The first published record of this disorder appeared in 1869. An anonymous author reported loss of bees which left behind hives with plenty of honey. It was speculated that the death was due to a lack of pollen, poisonous honey, or a hot summer [4]. Subsequently, Aikin [5] described losses in Colorado in 1891 and 1896 where large clusters disappeared or dwindled to tiny clusters with queens in May, hence the name “May disease”. Investigations at the time identified various fungi with these collapses. Burnside [6] was able to isolate, culture, and reproduce symptoms very similar to CCD with a strain of *Aspergillus* fungi.

Stonebrood, caused by the fungus *Aspergillus flavus*, affects both immature and adult bees. Infected larvae turn into solid, hard mummies that are not easily removed by the bees [7, 8]. Stonebrood-infected adults fly or crawl a considerable distance from colonies before dying [6]. Superficially, the adults appear normal [6]. It is believed that stonebrood is spread through the sharing of infected combs [9], as the fungus has been isolated on combs [10]. In addition, Giauffret [11] believed that disruption of the intestinal flora of bees due to antibiotic use may allow the fungus to spread. It is yet to be determined if the losses that are being seen today will, like with stonebrood, appear suddenly and then disappear [12].

In three epidemics between 1905 and 1919, 90% of the honey bee colonies on the Island of Wight in the United Kingdom died [13, 14]. Bees afflicted with this disorder could not fly, but crawled from the entrance [15, 16]. Researchers disagreed as to the cause of this affliction. Some concluded that the losses were due to acarine disease or the honey bee tracheal mite, *Acarapis woodi* [13]. Others believed that starvation was the cause of the losses [14, 17]; while still others thought Nosema disease caused the high losses [18]. Some affected beekeepers over the years have blamed their losses on the so-called “Isle of Wight disease” whenever they could not find another cause [14]. Bullamore [17] noted that genetics likely played a role and emphasized the need to dispose of colonies after a maximum of 3 years.

In the Stawell district of Australia in 1910, 59% of colonies were lost and many more were severely weakened [19]. Beuhne [19] noted that colonies that did not have their honey

extracted and that were allowed to gather honey late in the season did especially poorly. He concluded that honey made from *Eucalyptus leucoxylon* was too high in moisture, presumably fermented, and so was not suitable for consumption by the bees [19]. The author also mentioned that reliable accounts of severe losses from as far back as 1872 have been noted “at intervals of some years” [19].

Instances of large-scale losses were also reported in 1915 in Portland, Oregon [15] and from Florida to California in that same year [20], but these losses were not well documented. In 1917, widespread losses were reported in New Jersey, New York, Ohio, and Canada [21]. This time, an overabundance of pollen was blamed and bees were found dead in front of the hives. Root and Root [15] noted that this “disappearing disease” disappears within a short time without treatment.

In the 1960's, many reports of losses were published. In Texas and Louisiana, bees disappeared in the fall and winter [20]. In the Rio Grande River region of Texas in particular, the losses occurred after a period of unseasonable cold followed by 2 weeks of rain [22]. In Louisiana, bees were tested and found to be free of nosema disease, septicemia, honey bee tracheal mites, external parasites, and paralysis virus [23]. Roberge [24] noted that the bees had suddenly stopped clustering and believed the problem was genetic. Bees were also lost in California [25]. The remaining bees in the dwindled colonies appeared healthy and had plenty of food stores.

In Australia, losses were high in 1975 [26]. Termed “disappearing syndrome”, losses seemed to be due to dampness, poor nutrition, and stress [23]. Olley [26] noted that the syndrome could be transmitted between closely adjacent colonies through robbing and suggested that a virus was the cause. At about the same time, losses were reported in Mexico [27]. The losses in Mexico, called “disappearing disease”, were blamed on the environment, as research ruled out genetic factors [28]. In addition, paralysis virus and cold weather did not play a role [27]. Many factors, such as diseases, poor nutrition, and genetics, could have worked together to cause this syndrome [28]. An extensive survey conducted in 1975 indicated that the disorder could be found in 27 states [29]. Witherell [30] ruled out poisonous pollen, poisonous nectar, pesticide poisoning, and diseases caused by microorganisms, including viruses, as possible causes.

In the late 1970's, losses occurred in winter and spring in Florida [31]. Pathogens, food or lack of it, weather, genetics, and management were all suspected causes. Studies of the effects of protein sources on bee longevity and brood rearing showed that inadequate pollen substitutes could cause bee loss [32]. Losses also occurred in Seattle, Washington, where bees failed to cluster and were observed flying on a very cold day [33]. Losses in Texas at the time were not due to nosema disease or septicemia [22].

In the mid-1990's, losses again became evident in the northeastern United States [34]. Research showed that bee mortality could be reduced when Apistan, Terramycin extender patties, and Fumidil-B were used, but that menthol and grease patties did not prevent loss. This suggests that good nutrition and pest control may keep bees healthy and enable them to resist this malady and that honey bee tracheal mites did not likely contribute to the losses.

During the winters of 1998-1999 and 1999-2000, heavy losses were reported in France. Research showed that known honey bee diseases were present, alone or in combination with each other, in 76% of the effected apiaries [35]. There were no combinations of diseases that were more likely than the others, so the main suspects became colony mismanagement, nutrient

deficiencies, and chemicals in the environment. Then, as now, the cause of the losses was generally unknown.

Pesticides are often suspected as the cause for honey bee mortality because many that are used on bee-pollinated crops are toxic to honey bees. Tests commonly focus on finding doses of pesticides that are lethal to honey bees. However, the sublethal effects can also lead to problems at the colony level. At sublethal doses, the effects are more subtle. Honey production suffers [36] and foragers seemingly disappear [37]. For example, when colonies are placed near crops of sunflower treated with imidacloprid, foraging is disrupted and colonies dwindle and die as foragers fail to return to the hive [38]. This may be explained by the fact that some pesticides can cause a disturbance in the dance language or in the orientation abilities of worker bees [39, 40, 41]. In addition, the lifespan of workers can be reduced by sublethal pesticide exposure [42, 43].

Recently, a new species of nosema has been described [44] that has been suspected to cause losses similar to those being experienced at the present time. The presence of the parasite causes non-specific symptoms, such as low honey yields, higher than normal fall/winter colony mortality, and gradual depopulation of colonies [45]. The protozoan was first found in the Asian hive bee, *Apis cerana*, but is now reported from European bees, *Apis mellifera* [44, 45, 46]. In Spain, this protozoan was found in samples of bees where unexpected losses and poor honey yields were reported [46]. However, a recent survey of *N. cerana* prevalence in the U.S. has found the parasite to be widespread and to be in samples collected as far back as 2000 [47]. Very recent work has concluded that *N. ceranae* is not likely the cause of CCD [48].

The losses that have been occurring for over 100 years could be completely separate events or part of a cycle of disappearance. So far, we can only speculate. The cause of the recent honey bee colony losses, termed Colony Collapse Disorder, is still unknown [see MAAREC.org) for the latest information]. Scientists are working hard to determine what is killing our bees. It is hoped that, armed with many new tools, such as a complete mapping of the honey bee genome and modern molecular techniques, the cause of this latest outbreak will be determined.

Table 1. Past years of large-scale colony losses

Year	Location	Citation
1868	Kentucky, Tennessee	[4]
1872	Australia	[19]
1906	Isle of Wight	[17]
1910	Australia	[19]
1915	Portland, Oregon	[15]
1915	Florida to California	[20]
1917	United States	[15]
1917	New Jersey, Canada	[21]
1960's	Louisiana, Texas	[49]
1960's	Louisiana, Texas	[50]
1960's	Louisiana	[24]
1963-64	Louisiana	[23]
1964	California	[25]
1970's	Mexico	[27]
1970's	Seattle, Washington	[33]
1974	Texas	[22]
1975	Australia	[26]
1977	Mexico	[28]
1978	Florida	[31]
1995-1996	Pennsylvania	[34]
1999-2000	France	[35]
2002	Alabama	[20]
2002-2003	Sweden, Germany, etc.	[51]

References

1. vanEngelsdorp, D., D. Cox Foster, M. Frazier, N. Ostiguy, and J. Hayes. 2007. "*Fall dwindle disease*": *Investigations into the causes of sudden and alarming colony losses experienced by beekeepers in the fall of 2006. Preliminary report: First revision.* <http://maarec.cas.psu.edu/pressReleases/FallDwindleUpdate0107.pdf>
2. Burdick, E., and D.M. Caron. 2006. *MAAREC beekeeper survey.* <http://maarec.cas.psu.edu/pdfs/MAARECSurveyPub.pdf>
3. Caron, D.M., personal communication.
4. Anonymous, 1869. *Report of the Commissioner of Agriculture for the year 1868.* U. S. Government Printing Office, Washington, D. C. Pp. 272-281.
5. Aikin, R.C., 1897. *Bees evaporated; a new malady.* Gleanings in Bee Culture **25**:479-480.
6. Burnside, C.E., 1930. *Fungous Diseases of the Honeybee.* U.S. Department of Agriculture Technical Bulletin 149:1-43.
7. Maassen, A. 1906. *Die Aspergillusmykose der Bienen.* Mitteilungen aus der Kaiserlichen Biologischen Anstalt für Land- und Forstwirtschaft **2**:30-31.
8. Gilliam, M., and J.D. Vandenberg, 1997. *Fungi.* In Honey Bee Pests, Predators, and Diseases, Third Edition, R. A. Morse and K. Flottum [eds.]. The A. I. Root Company, Medina, OH., Pp. 81-110.
9. Betts, A.D. 1919. *Fungus diseases of bees.* Bee World **1**:132.
10. Rana, B.S., and M.T. Hamid, 1988. *Occurrence of some fungal parasites on bees (Apis mellifera L.) comb – a new report.* Indian Bee Journal **50**:23.
11. Giauffret, A., and Y.P. Tahercio, 1967. *Les mycoses de l'Abeille (Apis mellifica L.) etude de quelques antimycosiques.* Bulletin Apicole **10**:163-174.
12. Zander, E., 1919. *Die Brutkrankheiten und ihre Bekämpfung.* In Handbuch der Bienenkunde in Einzeldarstellung, Eugen Ulmer, Stuttgart. 69 pp.
13. Adam, B., 1968. "*Isle of Wight*" or acarine disease: *its historical and practical perspectives.* Bee World **49**:6-18.
14. Bailey, L., 1964. *The 'Isle of Wight disease': the origin and significance of the myth.* Bee World, **45**:32-37.
15. Root, A.I., and E.R. Root, 1923. *The ABC and XYZ of Bee Culture.* The A. I. Root Company, Medina, Ohio. 959 pp.
16. Anderson, J., 1930. "*Isle of Wight disease*" in bees. Bee World **11**:37-42.
17. Bullamore, G.W., 1922. *Nosema apis and Acarapis (Tarsonemus) woodi in relation to Isle of Wight disease.* Parasitology **14**:53-62.
18. Fantham, H.B., and A. Porter, 1912. *The morphology and life history of Nosema apis and the significance of its various stages in the so-called 'Isle of Wight' disease in bees (Microsporidiosis).* Annals of Tropical Medicine and Parasitology **6**:163-195.
19. Beuhne, R., 1910. *Bee mortality.* Journal of the Department of Agriculture of Victoria **7**:149-151.
20. [Tew, J.R., 2002. *Bee Culture's beeyard: Disappearing disease - An urban myth? Is this a disease? How can you tell?* Bee Culture .](#)
21. Carr, E.G., 1918. *An unusual disease of honey bees.* Journal of Economic Entomology **11**:347-351.
22. Kauffeld, N.M., J.H. Everitt, and E.A. Taylor, 1976. *Honey bee problems in the Rio Grande Valley of Texas.* American Bee Journal **116**:220-222, 232.

23. Oertel, E., 1965. *Many bee colonies die of an unknown cause*. American Bee Journal **105**:48-49.
24. Roberge, F., 1978. *The case of the disappearing honeybees*. National Wildlife **16**:34-35.
25. Foote, H.L., 1966. *The mystery of the disappearing bees*. American Bee Journal **106**:126-127.
26. Olley, K., 1976. *Those disappearing bees*. American Bee Journal **116**:520-521.
27. Mraz, C., 1977. *Disappearing disease south of the border*. Gleanings in Bee Culture **105**:198.
28. Kulinčević, J.M., W.C. Rothenbuhler, and T.E. Rinderer, 1984. *Disappearing disease: III. A comparison of seven different stocks of the honey bee (Apis mellifera)*. The Ohio State University, Research Bulletin 1160. Ohio Agricultural Research and Development Center, Wooster, Ohio, 21 pp.
29. Wilson, W.T., and D.M. Menapace, 1979. *Disappearing disease of honey bees: a survey of the United States*. American Bee Journal **119**:Part I:118-119, Part II:184-186, 217.
30. Witherell, P.C., 1975. *Conference on the disappearing disease of honey bees*. American Bee Journal **115**:300.
31. Kulinčević, J.M., W.C. Rothenbuhler, and T.E. Rinderer, 1982. *Disappearing disease. Part I. Effects of certain protein sources given to honey-bee colonies in Florida*. American Bee Journal **122**:189-191.
32. Kulinčević, J.M., W.C. Rothenbuhler, and T.E. Rinderer, 1983. *Disappearing disease. II. Effects of certain protein sources on brood rearing and length of life in the honey bee under laboratory conditions*. American Bee Journal **123**:50-53.
33. Thurber, F.F., 1976. *Disappearing-yes; disease-no!* Gleanings in Bee Culture **104**:260-261.
34. Finley, J., S. Camazine, and M. Frazier, 1996. *The epidemic of honey bee colony losses during the 1995-1996 season*. American Bee Journal **136**:805-808.
35. Faucon, J.P., L. Mathieu, M. Ribiere, A.C. Martel, P. Drajnudel, S. Zeggane, C. Aurieres, and M.F.A. Aubert, 2002. *Honey bee winter mortality in France in 1999 and 2000*. Bee World **83**:14-23.
36. Bendahou, N., C. Fleche, and M. Bounias, 1999. *Biological and biochemical effects of chronic exposure to very low levels of dietary cypermethrin (Cymbush) on honeybee colonies (Hymenoptera: Apidae)*. Ecotoxicology and Environmental Safety **44**:147-153.
37. Bonmatin, J.M., I. Moineau, R. Charvet, M.E. Colin, C. Fleche, and E.R. Bengsch, 2005. *Behaviour of imidacloprid in fields. Toxicity for honey bees*. In Environmental Chemistry, Green Chemistry and Pollutants in Ecosystems. Lichtfouse, R., J. Schwarzbauerm and D. Robert (eds), Springer, New York, Pp. 483-494.
38. Bortolotti, L., R. Montanari, J. Marcelino, P. Medrzycki, S. Maini, and C. Porrini, 2003. *Effects of sub-lethal imidacloprid doses on the homing rate and foraging activity of honey bees*. Bulletin of Insectology **56**:63-67.
39. Cox, R.L., and W.T. Wilson, 1984. *Effects of Permethrin on the behavior of individually tagged honey bees, Apis mellifera L. (Hymenoptera: Apidae)*. Environmental Entomology **13**:375-378.
40. Vandame, R., M. Meled, M.E. Colin, and L.P. Belzunces, 1995. *Alteration of the homing-flight in the honeybee Apis mellifera L. exposed to sublethal dose of deltamethrin*. Environmental Toxicology and Chemistry **14**:855-860.
41. Decourtye, A., J. Devillers, E. Genecque, K. LeMenach, H. Budzinski, S. Cluzeau, and M.H. Pham-Delègue, 2005. *Comparative sublethal toxicity of nine pesticides on olfactory*

- learning performances of the honeybee Apis mellifera*. Archives of Environmental Contamination and Toxicology **48**:242-250.
42. Smirle, M.J., M.L. Winston, and K.L. Woodward, 1984. *Development of a sensitive bioassay for evaluating sublethal pesticide effects on the honey bee (Hymenoptera: Apidae)*. Journal of Economic Entomology **77**:63-67.
 43. MacKenzie, K.E., and M.L. Winston, 1989. *Effects of sublethal exposure to Diazinon on longevity and temporal division of labor in the honey bee (Hymenoptera: Apidae)*. Journal of Economic Entomology **82**:75-82.
 44. Fries, I., F. Feng, A. daSilva, S.B. Slemenda, J.J. Pieniasek, 1996. *Nosema ceranae n. sp. (Microspora, Nosematidae), morphological and molecular characterization of a microsporidian parasite of the Asian honey bee Apis cerana (Hymenoptera, Apidae)*. European Journal of Protistology **32**:356-365.
 45. Fries, I., R. Martin, A. Meana, P. Garcia-Palencia, and M. Higes, 2006. *Natural infections of Nosema ceranae in European honey bees*. Journal of Apicultural Research **45**:230-233.
 46. Higes, M., R. Martin, and A. Meana, 2006. *Nosema ceranae, a new microsporidian parasite in honeybees in Europe*. Journal of Invertebrate Pathology **92**:93-95.
 47. Chen, Y., Pettis, J. Smith, B. and Evans, J. (submitted) *Nosema ceranae is a long-present and wide-spread microsporidian infection of the European honey bee (Apis mellifera) in the United States*. J of Invertebrate Pathology
 48. Pettis, J., personal communication.
 49. Williams, J.L., and Kauffeld, N.M., 1974. *Winter conditions in commercial colonies in Louisiana*. American Bee Journal **114**:219-221.
 50. Kauffeld, N.M., 1973. *Disappearing disease...a longevity problem?* Agricultural Research **22**:14.
 51. Svensson, B., 2003. *Silent spring in northern Europe?*
http://www.beekeeping.com/intoxications/silent_spring.htm, Accessed 15 February 2007.