Although such abrupt change in Fe distribution makes Fp and Pv denser and lighter, respectively, Irifune *et al*. found no anomaly in the net density of pyrolite. Indeed, the experimentally measured density of pyrolite matches the lower-mantle density profile deduced from seismology (*7*), thus supporting this traditional mantle composition model. On the other hand, the spin transition is known to strongly diminish the bulk modulus (incompressibility) and the electrical conductivity of pyrolite. Both experiment and theory have suggested an appreciable softening of the bulk modulus of Fp over the pressure range of spin transition (~4% reduction as pyrolite) (*8*, *9*) (see the figure). Such an anomaly is not found in the lower-mantle profile of seismologically observed bulk modulus (*7*), possibly because it is within the uncertainty of global seismic data (*9*). Recent laboratory measurements of electrical conductivity of pyrolite have shown that it decreases at depths greater than ~1200 km (*10*), likely attributable to the spin transition in Pv. Such measurements are not in agreement with geomagnetic field data, although observations constraining the lower-mantle conductivity are limited (*11*) (see the figure). The observed high electrical conductivity might suggest that the deep lower mantle is not pyrolitic in composition but includes a substantial amount of subducted oceanic crust, which exhibits much higher conductivity than pyrolite (*10*).

The results presented by Irifune *et al*. are an outcome of recent rapid developments in high-pressure experimental techniques combined with synchrotron x-ray radiation, which now enables precise density measurements up to 47 GPa and 2073 K (corresponding to 1200 km depth) in a large-volume press. These experiments have much better control of sample temperature than the other techniques, such as laser-heated diamond-anvil cell, with which similar experiments were previously performed. Nevertheless, much remains unknown about the composition and properties at greater depths. The nature of the spin transition in Pv is still an open question. A range of geophysical and geochemical observations suggest chemical stratification below ~1600 km depth (*10*, *12*, *13*). Additionally, the lowermost mantle, the bottom several hundred kilometers of the mantle, exhibits complex seismic-wave velocity structure. The recent discovery of silicate post-perovskite, a high-pressure phase of Pv, helps to explain the abrupt shear velocity increase at around 2700 km depth (*14*, *15*). On the other hand, supposed strong chemical heterogeneities in plume upwelling regions underneath Africa and the Pacific are yet to be examined. Further progress in high-pressure experimental techniques will allow us to tackle these unsolved problems in the deep Earth.

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ECOLOGY

Clarity on Honey Bee Collapse?

The worldwide losses of honey bee colonies continue to puzzle researchers and the beekeeping industry.

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Over the past few years, the media have frequently reported deaths of honey bee (*Apis mellifera* L.) colonies in the United States, Europe, and Japan. Most reports express opinions but little hard science. A recent historical survey (*1*) pointed out that extensive colony losses are not unusual and have occurred repeatedly over many centuries and locations. Concern for honey bees in the United States has been magnified by their vital role in agriculture. The California almond industry alone is worth $2 billion annually and relies on over 1 million honey bee hives for cross-pollination. So what is killing honey bee colonies worldwide, and what are the implications for agriculture?

In fall 2006 and spring 2007, many U.S. beekeepers encountered hives without adult bees but with abandoned food and brood. It was widely believed that these were symptoms of a new and highly virulent pathogen. In the absence of a known cause, the term "Colony Collapse Disorder" (CCD) was coined. What have we learned about this condition since then? Are the symptoms really novel?

CCD has stimulated a flurry of explanations, ranging from mobile phones and genetically modified crops, which have been dismissed by scientists (*2*, *3*), to pests and diseases, environmental and economic factors, and pesticides, which have received more serious consideration and stimulated much research. This week, for example, comprehensive surveys of honey bee losses in general in 16 countries in North America and Europe are reported (*4*). Although full explanations for these losses are still debatable, the consensus seems to be that pests and pathogens are the single most important cause of colony losses.

There is also growing evidence that the ability of a particular pathogen to kill colonies may depend on other factors, such as the ectoparasitic mite *Varroa destructor*. CCD-like symptoms have often been reported in Europe in colonies infected with this mite (*5*). Its original host was the Asian honey bee *Apis cerana*, but it colonized *A. mellifera* when this bee species was introduced to Asia. *V. destructor* is now present in all major beekeeping regions worldwide except Australia, where CCD symptoms have not been observed. It is not the mite itself that causes bee death, but a range of normally innocuous bee viruses that it carries. Experimental studies (*6*) have shown that *V. destructor* transmits viruses previously considered unimportant to honey bee biology, including slow paralysis virus and Kashmir bee virus, thus causing colony death. Field studies have demonstrated that the incidence and abundance of viral infections in *A. mellifera* have increased substantially since the mite colonized this species of bee. For example, in one study in the UK, the incidence of infection of experimental colonies with deformed

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wing virus increased from 0% in 1994–1995 to 100% once the mite was firmly established in the bee population during 1997–1998 (*7*). *V. destructor* has been controlled in various ways, including by acaricides, but in many areas, especially the United States and Europe, the mite has evolved resistance to the most effective chemicals used.

Mite interactions alone cannot, however, account for all losses attributed to CCD. One paradox noticed by researchers early on in the U.S. CCD story is that although *V. destructor* is universally present in affected colonies, mite numbers were often claimed to be small, whereas *V. destructor*–related colony losses elsewhere typically reported thousands of mites per colony (*8*). A possible resolution for the former lies in studies involving *V. destructor* and Kashmir bee virus (*9*), which report that the virus can persist in a colony's worker bees even in the absence of the mite, indicating that direct bee-to-bee virus transmission also occurs. This is not surprising, as this virus was present in *A. mellifera* before the bee was colonized by *V. destructor*. A study of U.S. CCD colonies using whole-genome microarrays found much evidence of viral infection, including by Kashmir bee virus (*10*).

In 2007, a metagenomic study (*11*) compared worker honey bees from dead or dying colonies showing CCD symptoms with workers from thriving hives. The analysis showed that Israeli acute paralysis virus, a previously esoteric virus, was the pathogen most commonly associated with CCD. Although the authors did not claim a causal relationship, this seemed reasonable, given that closely related viruses such as acute bee paralysis virus and Kashmir bee virus can kill colonies when in association with *V. destructor*. However, a 2009 study paints a less clear picture (*12*). Further studies on the pathology of bee infection by Israeli acute paralysis virus are needed and may be guided by studies on the related viruses linked to colony death.

Another pathogen that may be killing colonies is the microsporidian gut parasite *Nosema ceranae*, which also originated in the Asian hive bee *A. cerana*. *N. ceranae* affects adult bees and was recently found in collapsing *A. mellifera* colonies in Spain. Experimental results suggest that it is more virulent than *Nosema apis*, which has long been known to infect *A. mellifera*. However, molecular studies show that *N. ceranae* occurs in thriving colonies in many countries, and analyses of stored bee extracts showed that it was present in *A. mellifera* decades before the onset of CCD. More research is needed to determine how virulent *N. ceranae* really is (*13*).

Foraging honey bees and even whole colonies can be killed by chemicals intended to target other insects. Neonicotinoid systemic insecticides have been blamed for extensive colony collapse, and this has caused much debate. In France, the neonicotinoid compound imidacloprid was banned as a treatment on sunflowers and maize because of concerns that it could contaminate nectar or pollen and thus kill bees, but colony losses continued. After 10 years of research (*14*), it seems unlikely that imidacloprid was responsible for the French bee deaths, but it is conjectured that subtle, sublethal effects of either the compound or its metabolites may occur, perhaps making bees more susceptible to disease.

The mighty honey bee. Research is still needed to help beekeepers maintain healthy colonies and to determine what is killing colonies in colony collapse disorder. Shown is *A. mellifera.*

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The first annual report of the U.S. Colony Collapse Disorder Steering Committee, published in July 2009 (*15*), suggests that CCD is unlikely to be caused by a previously unknown pathogen. Rather, it may be caused by many agents in combination—the interaction between known pests and pathogens, poor weather conditions that diminish foraging, lack of forage (*16*), and management factors such as the use of pesticides and stress caused by long-distance transport of hives to nectar sources or pollination locations. The increasingly technical process of beekeeping itself merits further research as far as its impact on colony health. For example, although pollen substitutes are now widely used, little is known about the interactions between nutrition and disease susceptibility. Further research is also needed to develop effective ways of keeping colonies healthy through good hive management based on appropriate chemical, and other treatments such as "hygienic" bees that remove diseased brood and can be bred using conventional methods. In Europe, the COLOSS (COlony LOSS) network, consisting of 161 members from 40 countries worldwide, is coordinating research efforts and activities by scientists and the beekeeping industry to address these and other issues related to honey bee losses, including CCD (*2*).

In February 2009, the high pollination fee, combined with a temporary reduction in pollination demand due to drought and reduced almond prices, resulted in a surplus of hives in California available to pollinate almonds. But this leaves no room for complacency. Almond pollinating beekeepers had a poor summer in 2009 in the Dakotas and neighboring states, where hives spend the summer making honey, with heavy rains delaying and reducing the honey crop. This delayed chemical treatments for *Varroa* mites, and many colonies were probably in worse than usual condition going into winter back in California. It will be interesting to see what happens in February 2010 when the almonds bloom. On a longer time scale, there is a worrying downward trend in U.S. hives, from six million after World War II to 2.4 million today. Is the future of U.S. commercial beekeeping going to be based on pollinating a few high-value crops? If so, what will be the wider economic cost arising from crops that have modest yield increases from honey bee pollination? These crops cannot pay large pollination fees but have hitherto benefited from an abundance of honey bees providing free pollination.

Given the importance of the honey bee to mankind, the progress made in understanding CCD and colony losses in general is encouraging. But further research on honey bee health and well-being is needed.

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