By Philip T. Starks and Noah Wilson-Rich

Experiments in Epidemiology

How honey bees could have helped control swine flu.

Sometime during the end of March or early April 2009, a 5-year-old Mexican boy living down the road from a pig farm got sick. By mid-April, news reports blasted throughout the world press of a new type of influenza outbreak in Mexico City—where half the population of the boy's town commutes to work.

Within weeks, over 80 local people were dead from complications associated with swine flu infection. All schools and public parks were closed. Sunday church services were cancelled. Antiviral drugs were distributed to residents. Facial masks were disseminated to all citizens living within the vast metropolitan area in one of the world's most populated cities. This strain of influenza type A, 2009 H1N1, is remarkable in that it is a virus never before encountered by human kind.

As epidemiologists work tirelessly implementing and tweaking control strategies with each new country to which it spreads, an opportunity arises for prospective, population-level disease transmission studies.

Models for disease transmission facilitate our understanding of how infections spread, taking into account levels of genetic diversity, population density, and mechanisms for their control. New outbreaks such as swine flu provide valuable data with which to test these models and enhance their accuracy, yet scientists must sit and wait until these new data become available. Without natural outbreaks of disease, epidemiological modelers are unable to test their hypotheses.

The field would benefit greatly from the development of a new, financially viable, manipulable model system that mimics human populations with respect to genetic diversity, population density, and disease susceptibility. Ideally, this system should also present analogous behavioral responses to disease and should be amenable to chemical treatments.

The honey bee is a strong candidate.

Each colony of honey bees contains a single queen that has mated with as many as 30 unrelated males. That is as many as 30 patrilines within a single family. Apiaries can be organized to have anywhere from a single hive to hundreds of hives. That's the equivalent of everything from a single family up to multiple major cities. Honey bee hives are susceptible to—and are naturally associated with—arthropod, bacterial, fungal, and viral pathogens. Genetic diversity, population density, and disease susceptibility are all manipulable with artificial insemination, apiary management, and pathogen cultivation. And manipulable at a fraction of the cost necessary for similar experiments with rodents (which provide none of these advantages).

Like humans, honey bees display a variety of disease defense mechanisms. They mount a fever when infected. They groom. They dispose of the dead. We may know more about hygienic behavior in honey bees than in any other potential model system. Like medical doctors, apiculturists have developed disease management strategies. We have chemical treatments for some diseases. As a whole, not only can we manipulate genetic and population characteristics, but the system mimics humans with respect to behavioral responses and the availability of chemical preventatives and treatments.

It is unlikely that the physiological responses of honey bees replicate those of humans. But we are not arguing that honey bee research should replace other methods; rather, disease research would complement existing methods for epidemiological study. In this one experimentally manipulable, behaviorally rich system, we can prospectively study disease transmission and disease suppression within and across multiple populations differing in population density, genetic diversity, and behavioral response in ways directly analogous to those in human populations. And we can do it without breaking the bank.

Philip T. Starks and Noah Wilson-Rich are at the Department of Biology, Tufts University.