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Defense against diabetes

Type 1 diabetes, an autoimmune disease, is becoming increasingly common in developed countries. This suggests that the disease is affected by environmental conditions such as lifestyle and diet, which determine exposure to microbes. Studies have shown that diabetes-prone mice that are exposed to bacteria are slightly less likely to develop the disease than are mice in a sterile environment.

A new study led by Alexander Chervonsky (University of Chicago, IL) suggests that resident microbes in the intestine, as well as MyD88, a protein that regulates the immune response to these microbes, are key players in the development of type 1 diabetes (*Nature* published online 21 September 2008; doi:10.1038/nature07336). The authors engineered mice from a diabetes-prone strain to lack MyD88. Unlike most normal mice from this strain, knockout mice did not develop diabetes when exposed to intestinal microbes. When knockout mice were housed in a sterile environment, however, they developed the disease.

In normal diabetes-prone mice, MyD88 may prevent microbes from fending off disease. Thus, when the protein is lacking, the microbes can work to keep diabetes from developing. In a microbe-free environment, however, nothing stands in the way of disease development, and the absence or presence of MyD88 has no effect.

New porcine model for cystic fibrosis

Cystic fibrosis (CF) affects about 1 in 4,000 newborns in the US. This inherited disease involves the mucus glands of the lungs, liver, pancreas and intestines, causing progressive disability from multiple system failures. About 20 years ago, it was determined that CF is caused by mutation of the gene *CFTR*. Despite that discovery, however, the underlying disease process is still not well understood, and CF remains incurable. Even with treatment, most people with CF die at relatively young ages, many from lung failure. Mouse models of CF have been developed but do not recapitulate many of the characteristics of CF in humans, limiting their utility in studying CF pathology.

Now a new breakthrough from Michael Welsh (University of Iowa, Iowa City), Randall Prather (University of Missouri, Columbia) and colleagues offers researchers the chance to examine CF more closely. Welsh and Prather developed a pig model of CF by using genetic engineering to disable the gene *CFTR*. Newborn pigs with both copies of *CFTR* disabled have many of the same symptoms as humans with CF: defective salt transport, intestinal blockage and damage to the pancreas and liver (*Science* **321**, 1837–1841; 2008). It is hoped that the pig model will aid scientists in developing better CF treatments.

FDA regulation of GE animals

Genetic engineering technology has become commonplace in recent years, and though genetically engineered (GE) animals have not yet been released to the market, they probably will be in the near future. To address potential public health concerns, the US Food and Drug Administration (FDA) has released a draft guidance that explains the process by which certain GE animals are regulated (http://www.fda.gov/cvm/geanimals.htm).

The guidance, which is not yet binding, currently focuses on GE animals that are intended for consumption, for human therapeutic use (tissue transplants or pharmaceutical products) or for direct interaction with humans (pets or industrial animals). Producers of such animals must obtain FDA approval before introducing them to the market, and they must prove both that the animals are safe for use and that they carry the traits that they were engineered to express. At this point, most research scientists who produce GE animals for pure experimental purposes and not for consumption do not need to request approval from the FDA. "In general we're not interested in having an application from every post-doc and graduate student who's making a knockout mouse for this, that or the other thing," explains Larisa Rudenko, Senior Advisor for Biotechnology at the FDA Center for Veterinary Medicine.