How the plant immune system can drive the formation of new species

September 4 2007

Example of thale cress suffering from hybrid necrosis. Left and right are the healthy parents; center is the sick hybrid offspring. Credit: Image: Kirsten Bomblies

Plant geneticists know that not all plants from the same species can be successfully bred. Apparently, there are reproductive barriers that not only prevent the exchange of genes between well-established species (which goes to the very definition of a species), but also between varieties of one and the same species. How these barriers arise is of central importance if one wants to understand the origin of biodiversity.

A research team led by Detlef Weigel from the Max Planck Institute of Developmental Biology in Germany and Jeff Dangl from the University of North Carolina has now shown that a mis-regulated plant immune

system can establish reproductive barriers and might be a first step toward speciation.

The international collaboration studied a genetic incompatibility known as hybrid necrosis, using thale cress, Arabidopsis thaliana. The new study, reported in the latest edition of the open-access journal *PLoS Biology*, was based on the observation that unfit hybrids arising in different plant species are very similar. Their growth is retarded, the leaves become yellow and necrotic, the tissue collapses, and they often do not survive to make flowers; the syndrome is generally known as hybrid necrosis. “We suspected that hybrid necrosis is always caused by the same biochemical mechanism,” explains Weigel, director at the Max Planck Institute.

To test this hypothesis, the scientists took 280 genetically different strains of Arabidopsis from all over the world, which they crossed in 861 different combinations. Most of the hybrid plants were strong and grew normally, but 20—or two percent—of the crosses produced only small necrotic and unhealthy plants. Genomics-based experiments showed that these hybrids all had a comparable profile of gene activity: A common group of some 1000 genes were either more strongly or more weakly active in the hybrids than in their healthy parents. Moreover, this pattern was very similar to what is seen with a strong defense response mounted against pathogens during a normal infection. The plant immune response typically involves the sacrifice of a few cells at and around the infection site. But in the wimpy hybrids, healthy tissue also suffered, despite there being no pathogen around. The hybrid plants apparently mistook their own cell s for dangerous germs.

Although the genes that determined the abnormal autoimmunity were different in most crosses, the researchers discovered that often only two genes were required to cause the necrotic hybrid response: one from the father, the other from the mother. In one case that the researchers

studied in more detail, they found that the gene that causes necrosis only in hybrids, but not in the parents, is normally used to sense the presence of a pathogen. The scientists emphasize, however, that the hybrids are not the victims of malfunctioning genes. In contrast to many hereditary diseases, the necrosis is not due to each parent carrying a defective copy of the same gene; rather, there is a destructive interaction between two different genes, each of which evolved differently in the two parents. The genes on their own are harmless or even beneficial, since the parents are healthy. Only the combination of the altered gene variants creates problems. These types of genetic malfunction are ofte n known as Dobzhanshy-Muller incompatibilities, after the two giants of early modern genetics who first studied these necrotic hybrids in fruit flies.

The results of the German-American team challenge the classical definition of a species, according to which individuals of one species can mate at will and produce fertile offspring. Apparently, there are barriers to the free interchange of genes even within a species; after all, one out of 50 crosses in this study was not successful. “The formation of new species thus needs to be understood as a gradual process, where barriers within a species continually increase, until two groups cannot be crossed at all anymore” says Weigel.

While this view is widely accepted today, it is mostly unclear why such genetic barriers arise in the first place. Which advantage has the plant, when sometimes all seeds from a cross die" The current study offers a possible explanation. The plant genome changes under pressure from pathogens. “Plant and pathogen are locked in a race,” says Dangl, professor and expert in the genetics of plant pathology at the University of North Carolina. The pathogens tirelessly develop new strategies to attack the plant and evade its immune system. The plant, in turn, tries to prepare against as many new microbe “weapons” as possible. Armed to the teeth, it can happen that a harmless protein variant from a more distant relative is all of a sudden classified as dangerous and attacked.

The scientists are optimistic that their insights can be applied to other species. Common traits indicate that hybrid necrosis in crops such as wheat is caused by the same mechanisms as in thale cress. Dangl, therefore, believes that Arabidopsis can serve as a useful model for the understanding of hybrid necrosis in general. “Such a model would be very useful for breeding, since such genetic incompatibilities prevents some of the crosses breeders would like to make,” according to Dangl. The finding that only a few genes are responsible for each case of hybrid necrosis is particularly encouraging. It seems that only a few genetic changes are required to circumvent crossing barriers and to achieve a desired new combination of genetic traits. The flip side of the coin is that only minimal modifications in the genome can be sufficient to suppress the free exchange of genes between relatives, and that perhaps not much is needed to form a new species.

Citation: Bomblies K, Lempe J, Epple P, Warthmann N, Lanz C, et al. (2007) Autoimmune response as a mechanism for a Dobzhansky-Muller-type incompatibility syndrome in plants. PLoS Biol 5(9): e236. doi:10.1371/journal.pbio.0050236.

Source: Public Library of Science