

Fitness Effects of Transgenic Disease Resistance in Sunflowers

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Fears about transgene escape have focused attention on the potential for hybridization between crops and their wild relatives. Although transgenes will often escape from cultivation (1), their rate of spread will be mainly governed by their fitness effects, not the migration rate (2). Thus, only highly advantageous transgenes will spread rapidly enough to have a substantial ecological impact. Therefore, research on the risks associated with transgene escape should focus on the fitness effects of the gene(s) in question.

Here, we examined the fitness effects of a transgene conferring resistance to white mold (*Sclerotinia sclerotiorum*) in sunflower (*Helianthus annuus*). Unfortunately, attempts to breed white mold resistance have met with little success, and chemical control methods are costly and often ineffective. Therefore, attention has turned to genetic modification. An oxalate oxidase (OxOx) transgene has now been used to enhance white mold resistance in cultivated sunflower [supporting online material (SOM) text], presumably by degrading oxalic acid, which contributes to white mold pathogenicity (3).

Most of the sunflower acreage in the United States occurs within the range of wild sunflower, and many fields flower coincidentally with neighboring wild populations (4). Where they come into contact, cultivated and wild sunflower often hybridize (5), making transgene escape a virtual certainty. Combined with the efficacy of the OxOx transgene, this raises the possibility of hybridization giving rise to a more invasive wild sunflower.

To simulate the early stages of escape, we backcrossed the OxOx transgene into wild sunflower and grew the resulting plants in containment cages at field sites in Indiana, North Dakota, and California (6). Just before flowering, we inoculated half of all the plants with white mold and

then monitored them for symptoms of infection.

Presence or absence of the OxOx transgene had no effect on seed output ($P = 0.25$), indicating that there was no cost of resistance in the absence of a pathogen challenge (Fig. 1A). In terms of infection rates, the OxOx transgene did provide protection against white mold ($P = 0.002$) (Fig. 1B). The transgene did not, however, have any effect on seed output after inoculation ($P = 0.84$) (Fig. 1C). Though the transgene provided protection against white mold infection, it had no effect on reproductive output. This result has a simple explanation: Variation in the likelihood of infection was offset by variation in the severity of infection (compare Fig. 1, B and D). In California, where the transgene provided the most protection against infection, disease onset had no effect on seed output. In contrast, white mold infection caused a severe decline in seed output in Indiana, but infection rates were unaffected by the transgene. Thus, the transgene had a significant effect on the likelihood of infection, and infection had a negative effect on seed output ($P \leq 0.0001$) (Fig. 1D), but the disease effect varied across locations ($P = 0.001$), nullifying any advantage of the transgene.

Our results suggest that the OxOx transgene will do little more than diffuse neutrally after its escape. This is especially true because our experiment simulated the worst-case scenario, in which early generation hybrids faced a severe pathogen challenge.

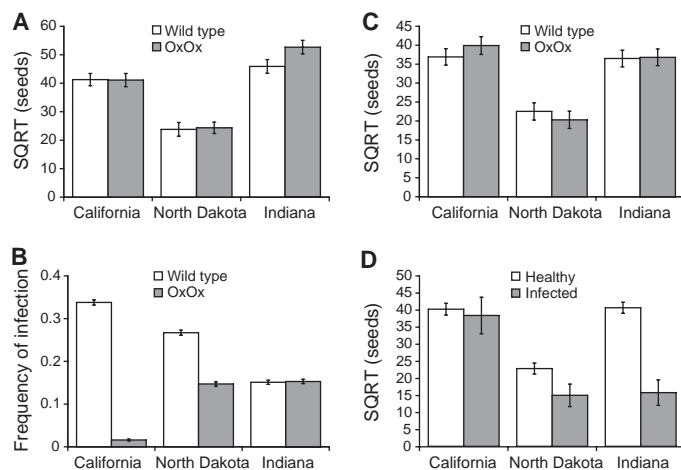


Fig. 1. Effects of the OxOx transgene and white mold infection on crop × wild sunflower hybrids. (A) Seed output of control individuals. (B) Frequency of infection after inoculation. (C) Seed output after inoculation. (D) Effect of infection on seed output after controlling for presence or absence of the transgene. All values are expressed as least-squares means \pm one standard error.

These results also illustrate the importance of quantifying fitness directly, rather than using a presumptive correlate such as disease incidence; had we relied solely on infection rates, our conclusions would have been quite different. Although the mechanism(s) responsible for the decoupling of infection rate and disease severity are unclear, one possibility is that conditions favoring white mold infection differ from those favoring development of the disease once it has been acquired. The hotter, drier climate of California might, for example, make plants more susceptible to infection but hinder development of the disease. Regardless, it appears that, by giving the OxOx transgene to wild sunflower, we gave it something that it already had: some degree of white mold resistance.

One caveat is that this work was performed within a single season and on a single genetic background. Therefore, our results may not be generalizable. Another caveat is that plants in this experiment were not subjected to environmental stresses such as drought; plants grown under stress may be less able to protect themselves from disease, and thus derive a greater benefit from transgenic disease resistance. Of course, stressful conditions could also reveal a cost of resistance not otherwise observed.

Future studies assessing the environmental impact of transgenes should not only be replicated over space and time, but should also examine the effects of genetic background and environmental stresses. Regardless of the form of future research, an informed judgment of the risks and benefits of genetic modification on a case-by-case basis is preferable to either the dismissal of transgenic approaches entirely, or the introduction of transgenic crops in the absence of appropriate scientific scrutiny.

References and Notes

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6. Materials and methods are available as supporting material on Science Online.
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Supporting Online Material

www.sciencemag.org/cgi/content/full/300/5623/1250/DC1
SOM Text
Materials and Methods

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