oped from the few survivors of a mass selection lost their resistance (*4*).

In the field, the level of selection declines from 100% to 0% over the margin of a treated area, and here the changes of gene frequency are a function of the selection pressure. Gene flow into the selected area allows the resistant factors to be assembled into the resistant strain.

The response to directed selection depends not only on the change of frequency of the resistant factors but also on the response to selection for a genetic background required for fitness of the resistant strain (*5*).


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*Response*: We agree with Tabashnik that the optimal dose depends on a number of factors, as emphasized in several places in our article. We did not imply that a low dose applied to a population with incipient resistance will always aggravate the problem.

Our point is that the optimal dose and use patterns vary widely; there are no simple solutions, and populations with suspected incipient resistance create a dilemma, particularly in determining the dose. The major problem here is, as we pointed out, that it is not possible to detect incipient resistance development with currently available technology; therefore, computer models are not yet of much *practical* help.

We do not advocate the use of high doses in any situations, but instead, in the article, recommended the maintenance of refugia (Tabashnik's points i and ii), genetic studies to characterize the inheritance of the resistance allele (Tabashnik's point iii), and intensive monitoring of resistance gene frequencies (Tabashnik's point iv) to make the best judgment.

We agree with Tabashnik that the use of a high insecticide dose most likely will cause more problems than it can solve, presents serious risks for undesirable side effects, and runs counter to integrated and any other sensible use of insecticides.

Therefore, we think the only way the phrase Tabashnik quotes ("exposing a population with incipient resistance to a low insecticide dose leads to rapid fixation of resistance. . .") can be misconstrued as a recommendation to use a high dose of insecticide is to read it out of its context.

McEnroe makes the interesting and valid point that resistance development depends not only on the gene (allele) directly responsible for a certain resistance mechanism but also on genetic fitness factors or a "genetic background required for fitness."

We did not mention this in our article because not much can be said about this phenomenon in relation to resistance in agricultural insects.

McEnroe's point deserves significantly more attention than has been afforded it so far.


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## "Mazel Tov" Usage

I would like to respond to Stephen A. Ockner's comment (Letters, 17 Oct., p. 261) about "Mazel tov."

The current use of the term "Mazel tov" certainly does mean "congratulations," rather than "good luck." This is not, however, its original meaning. The Yiddish term "mazel," stems from the Hebrew "mazal," which means "constellation." Tov just means "good." The direct translation of "mazel tov" is therefore, "A good constellation!" The idiom originated from the astrological interests of the Jews in ancient Israel, which are well known from surviving mosaics on floors of certain synagogue ruins. In less idiomatic terms, when an ancient Israeli said, "Mazel tov!" he was wishing a good astrological influence for the recipient of his statement. This can be loosely termed, "Good luck!" The usage of the idiom, however, can also follow the event for which luck was being wished. Rather than, "May you be blessed by a good constellation," it becomes, "You must have been blessed by a good constellation," or "Congratulations!" Either way, it comes out "Mazel tov."


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