complicated relationship between the precise masses of the top quark and the W boson and the predicted mass of the still-unseen Higgs boson. However, the legend in the figure illustrating this relationship may mislead readers about the source of the precision measurements of these particles' masses.

The original legend in the figure supplied by the Collider Detector at Fermilab (CDF) Collaboration read "World average measurements," but it appeared in *Science* as "CDF plus other measurements." This alteration does not give important credit to the DZero collaboration at Fermilab and the UA2 collaboration at the European Organization for Nuclear Research (CERN), in Geneva, Switzerland; both have made crucial contributions to the average. The main point of the plot is to show how precise measurements of the top quark and W boson masses give indirect information on the Higgs particle mass, one of the most important parameters in high-energy physics. The limit on the Higgs mass is driven primarily by the W mass measurement, and DZero currently has the world's most precise measurement of this quantity.

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The error in the legend in the figure occurred during editing. *Science* regrets the error.—***Eds.***

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Controlling Cotton Pests

Contrary to the characterization in Jocelyn Kaiser's article "Pests overwhelm *Bt* cotton crop" (News & Comment, 26 July, p. 423), the Bollgard *Bacillus thuringiensis* (*Bt*) gene by Monsanto is providing economic and environmental benefits to cotton growers and is performing as expected given this year's severe pest conditions.

Bollgard was evaluated in 6 years of field tests before commercialization. The vast majority of these tests were done in full public view by scientists at the U.S. Department of Agriculture, universities, and extension facilities. Control of the pests targeted for this product—tobacco budworm, pink bollworm, and bollworm—was excellent. In a few of the field tests, high infestation levels have required application of pesticides to supplement the control provided by the *Bt* gene.

Monsanto is well aware of the potential for pests to adapt to the *Bt* protein. Because we—along with many others—have much to lose if this happens, we have worked long and hard with resistance management experts to develop strategies to delay the onset of resistance. Strategies vary depending on the insect involved. With the bollworm, the key strategy is refugia, host plants where the insect can escape exposure to the *Bt* protein. Nonselected populations that develop on these refuges help dilute and suppress any resistance genes that may develop in the Bollgard fields. The bollworm has a multitude of hosts—both wild and crop plants. With Bollgard, resistance management is taken even further by requiring growers to plant refuges with cotton that does not contain the Bollgard gene. When both the natural and mandated refuges are combined, resistance development in the bollworm can be delayed significantly.

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The bollworm outbreak on *Bt* cotton is not a manifestation of physiological resistance predicted in 1991 (*1*); rather, the epidemic apparently arises from extant populations that have the inherent ability to discriminate

among tissues with varying concentrations of the toxin within a plant. Whereas physiological resistance in insects to pesticides and plant resistance (biotypes) is well documented (*2*), inherent behavioral capabilities of insects to discriminate in a toxin mosaic superimposed on their host or habitat is not well understood (*3*). Insect-resistant transgenic plants appear to provide an ideal substrate in which entomologists can explore this previously recalcitrant and otherwise neglected subject. Nonlethal repellency of preadapted populations from human-valued resource tissues to yield insensitive tissues offers the prospect of conserving the extant insect genome by these intraplant refugia and of increasing mortality in cannibalistic species by concentrating populations in smaller areas. Realization of such possibilities would be accelerated if prepared minds were combined with proprietary technology in a context of production agriculture.

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References

1. M. K. Harris, *Science* **253**, 1075 (1991).
2. R. L. Metcalf and R. A. Metcalf, in *Destructive and Useful Insects* (McGraw-Hill, New York, 1993), pp. 7.42–7.47; *Biology and Breeding for Resistance* M. Harris, Ed. (Texas Department of Agriculture Commission, Texas A&M University, College Station, TX, 1980).
3. A. Brown, J. Haworth, A. Zahar, *J. Med. Entomol.* **13**, 4 (1976).

It is regrettable, but not surprising, that *Bt* cotton in its initial year of commercialization has fallen victim to the bollworm. During 1994, when bollworm numbers were extremely high in North Carolina, peak boll damage and yield reductions in two of our *Bt* cotton tests exceeded 20% (*1*).

Our data (*1*, *2*) were summarily ignored in favor of data acquired when there were low numbers of "wild" bollworms or from test sites artificially infested with laboratory-cultured larvae. The wave of euphoria created by *Bt* cotton swept across the cotton-belt and carried many entomologists with it.

In *Bt* cotton, biotechnology has provided cotton farmers with a most powerful tool to assist in the management of insect pests; however, for success and sustainability the tool must be strategically integrated with other management tactics into systems designed for specific areas.

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References

1. J. S. Mahaffey, J. R. Bradley Jr., J. W. Van Duyn, *Proc. 1995 Beltwide Cotton Conf.* **2**, 795 (1995).
2. J. S. Mahaffey, J. S. Bacheler, J. R. Bradley Jr., J. W. Van Duyn, *Proc. 1994 Beltwide Cotton Conf.* **2**, 1061 (1994); A. L. Lambert, J. R. Bradley Jr., J. W. Van Duyn, *Proc. 1996 Beltwide Cotton Conf.* **1**, 931 (1996).

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HIV Fusion

Recently, an assemblage of evidence (*1*, *2*) demonstrated that any one of four β-chemokine receptors can act as human immunodeficiency virus (HIV) cofactors. In some interpretations (R. Weiss, "HIV receptors and the pathogenesis of AIDS," Viewpoints, 28 June, p. 1885) (*1*), these host cell receptors have been prematurely assigned HIV coreceptor function. Back-to-back reports drew contradictory conclusions about the specificity of one such HIV cofactor, suggesting the complicity in HIV infection of additional unaccounted variables.

For a component to be classified as a receptor, three criteria must be met: the component must demonstrate (i) saturable binding of the ligand to the receptor; (ii) specific binding of the ligand to the receptor that is competitively inhibited at the binding site; and (iii) a rate-dependent biologic response to the ligand (*3*). When the meth-