## Plants Talk—But Can They Listen?

K. BROWN'S ARTICLE "SOMETHING TO SNIFF at: unbottling floral scent" (News Focus, 28 June, p. 2327) highlights the spectacular advances recently made in the field of plant volatiles through a combination of ecological, molecular, and evolutionary techniques. The sidebar within the article ("Plants 'speak' using versatile volatiles," p. 2329) attempts to tie these advances to the question of what have been called "talking trees." Several researchers, including J. Tumlinson and I. Baldwin, have demonstrated that insects have exquisite abilities to detect volatile compounds emitted by plants and that both herbivorous and predatory insects can respond quite strongly to certain phytogenic volatiles. Such results fit well with research on the physiology and neurobiology of insect olfactory systems.

If a tree talks, is anyone listening?

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What is not yet known, however, is whether plants growing under natural (or agricultural) conditions respond directly to volatile signals from other plants. Many of the recent experiments on this question have been conducted under laboratory conditions that artificially (and, possibly, artifactually) raise the concentrations of the volatile compounds under consideration. Simple calculations of biogenic flux and turbulent diffusion rates suggest that most plants growing outdoors see concentrations of biogenic volatiles several orders of magnitude lower than those commonly used in lab and growth chamber experiments. We still lack convincing evidence that plants respond to volatile signals from other plants when turbulence conditions are realistic and concentrations approach those seen in nature. In contrast to insects, plants appear to lack highly evolved reception and transduction systems for volatile signals. The question about plants is not whether they can talk. The question is, do they listen?

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## Something in the Eye of the Beholder

BECAUSE OF THE INTENSE INTEREST IN THE stem cell field, even reports of failure to replicate previous findings have appeared in prominent journals. For example, two recent reports question whether adult bone marrow–derived cells contribute to central nervous system (CNS) neurons because the authors failed to see markers of such cells in brains ("Failure of bone marrow cells to transdifferentiate into neural cells in vivo," R. F. Castro *et al.*, Brevia, 23 Aug., p. 1299; "Little evidence for developmental plasticity of adult hematopoietic stem cells," A. J. Wagers *et al.*, Reports, 27 Sept., p. 2256; published online 5 Sept.; 10.1126/science.1074807). These conclusions are in marked contrast with previous reports by us and others (*1*–*3*) that found that bone marrow–derived cells transit to the CNS in adult mice, express proteins typical of neurons in the olfactory bulb, and contribute to well-defined subsets of neurons such as Purkinje cells in the cerebellum. Efforts to replicate discoveries are critical to the scientific process, and convincing failures to do so are important contributions to the literature. The fundamental issue is defining what makes a study convincing and, therefore, what should be the criteria for overturning previously published findings. The findings by Castro *et al.* and Wagers *et al.* underscore the need to establish criteria for publishing negative reports.

First, a prerequisite for proving a null finding is the clear ability to detect a positive control. A case in point is Castro *et al.*, who fail to detect not just neurons but also bone marrow–derived microglial cells within the CNS. At least 20 reports over the past 15 years have shown that bone marrow transplantation results in readily detectable replacement of a large proportion of microglial cells in the brain (*4*–*8*). Moreover, following a stab wound, the presence of such cells in the brain would be impossible to miss, as they are localized in great abundance at the site of the wound (*9*). Thus, the lack of detection of microglia by Castro *et al.* suggests that their system was unable to detect marrow-derived cells that should have been present in the brain.

If such controls fail, the fidelity of an assay must be questioned. Perhaps the major problem in the findings of Castro *et al.* lies in the use of ROSA26 transgenic mice that constitutively express β-galactosidase (β-Gal) in most cells. The expression of β-Gal by these mice is very weak at the single cell level and can be difficult to distinguish from endogenous mammalian β-Gal activity, especially in the brain in cells at high magnification. We know this from personal experience, as we, like Castro *et al.*, used ROSA26 bone marrow donors for an entire year to track marrow-derived cells within the brains of recipient mice. We ultimately rejected the ROSA26 approach because it lacked specificity and sensitivity in the brain and, therefore, took pains to redo all of our experiments for 2 subsequent years with a marker that has no endogenous counterpart, green fluorescent protein, before publishing our report (*1*).

The report by Wagers *et al.* exemplifies another concern regarding the publication of negative findings. In the absence of an adequate description of the methodologies used, the experimental results are difficult to interpret or compare with previous results. Indeed, scientists may well be comparing apples with oranges. For example, in Wagers *et al.*, in the case of the brain, it is unclear what regions were assayed, and a different marker was used from those published previously (*1*–*3*). In the case of skeletal muscle, the particular muscles sampled were not identified. This choice could have profound effects on the results obtained, as there are hundreds of