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COMMENTARY

Genes do not form channels

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Glutamate receptors and related proteins are important players in plant ion transport, cellular signalling, and ion toxicity, as substantial recent work has shown (see reviews by Dietrich et al. 2010; Kronzucker and Britto 2011; Maathuis 2007; Zhang et al. 2010). A new article on the subject, published in the journal *Science*, however, comes with the unsettling title "Glutamate Receptor–Like Genes Form Ca^{2+} Channels in Pollen Tubes and Are Regulated by Pistil D-Serine" (Michard et al. 2011). This paper discusses the role of "ionotropic *GLRs* [glutamate-receptor-like genes]…identified in the genome of *Arabidopsis*", and declares in its final paragraph that "Genes for putative cyclic nucleotide-gated channels were the first reported as plausible Ca^{2+} channels…." Thus, at several junctures, the manuscript rather boldly conflates genetic and functional attributes.

This requires some editorial comment. Surely, the authors of this paper, its referees, and the journal's editors must be aware that genes do not form channels, mediate ion fluxes (i.e. behave "ionotropically"), or perform any known catalytic or transport function in living organisms. Yet, the very term "glutamate receptor-like gene" is itself a misnomer: surely, a *gene* cannot be likened to a glutamate receptor. Moreover, the genes in question are not themselves regulated by the amino acid D-serine, contrary to what the paper's title unequivocally states.

To be fair, this sort of category error is not without precedent in the literature; for example, it can be seen in other articles on GLR channels by Chiu et al. (2002), Li et al. (2005), Meyerhoff et al. (2005), and Roy et al. (2008). While the conflation of gene and protein seems unusually rife in this area, it is by no means restricted to it, nor is it restricted to plant biology. Examples abound of statements that genes "transport" substances (Dean et al. 2003), "synthesize" or "produce" other substances (Malik et al. 2009; Pulkkinen et al. 2000; Weiner et al. 1993), and "catalyze" chemical reactions (Chen et al. 2005; Drakas et al. 2005; Lewinsohn et al. 2001; Metherall et al. 1996; Ono et al. 1999; Powell et al. 2008; Ullrich and van Putten 1995).

Common usage of an error does not make it any less erroneous. However, the example of Michard et al. is particularly egregious in that it has introduced this manner of speech into the pages of one of the world's most highly esteemed scientific journals. With this stamp of approval, it propagates a rather stunning

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biological inaccuracy to the broad readership that *Science* reaches, with the potential of misleading laypeople, non-specialists and students of biology, who may not know better. In addition to the confusion between gene and protein, it offers the false promise that catalytic DNA has been discovered, similar to the finding, three decades ago, that RNA can have catalytic properties (Kruger et al. 1982; Guerrier-Takada et al. 1983). The implication that the gene itself is regulated by serine also inadvertently glosses over the importance of post-translational regulation of gene products, which is central not only to the findings of the Michard et al. paper itself, but is also one of the most important areas of plant biology (e.g. Amtmann and Blatt 2009; Kaiser and Huber 2001; Plaxton 1996).

We suggest that such linguistic errors are far from semantically trivial, but are part of a deeper issue, that of the erosion of language in the biological sciences, to the point at which misleading verbal constructs become permissible at the highest level of publication. The reasons for this development are, no doubt, complex, but might be linked to deep changes in university-level biology curricula (Tyree 2003). While the paper by Michard et al. is not without scientific merit, its authors would do well to heed the caution expressed by Orwell, that imprecise language "makes it easier for us to have foolish thoughts" (Orwell 1946).

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