

other farming practices have caused the hypoxic condition, and changes in farm policy can correct the problem with relatively little cost to farmers and with little or no loss of agricultural productivity. The idea proposed by Winstanley and E. Krug that modern agriculture has a "cleansing" influence on water quality is absurd, as discussed in the critique by M. David and G. McIsaac, to which the Ferber article refers. The vast majority of the science points to the need to change farming practices in the United States if we are to solve the problem of the dead zone and the deteriorated water quality elsewhere in our country.

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IN DAN FERBER'S REPORT ON HYPOXIA, HE tells a good-girl/bad-boy story about N. Rabalais and me rather than describing legitimate differences of scientific opinion. In so doing, he makes inaccurate and misleading statements.

(i) Ferber states that Edward Krug and I conclude that modern agriculture has greatly cleansed the Illinois River. We in fact conclude that reduction in the concentration of nitrogen is attributable to both point- and non-point-source pollution control. The discharge of nutrients from Chicago wastewater has decreased by more than 90% since the 1920s.

(ii) Ferber reports that I argue that the dead zone in the Gulf of Mexico is a natural phenomenon. My position is that the relative contribution of offshore sources of nutrients from upwelling waters of the continental slope is unknown. The White House Committee on the Environment and Natural Resources recognizes that algal blooms in shallow coastal zones can be caused by upwelling of deep nutrients (1). The coastal zone of the Pacific Ocean from Canada south to Chile experiences natural hypoxia (2).

(iii) Ferber also reports that I have led an effort to clear the Mississippi Basin's name. This is false. As the CENR report itself acknowledges, a large body of addi-

tional data is available, but was not used. The failure of the report to incorporate a large body of scientific data reflects a bias with the report, not with me.

(iv) Ferber knows that I support the implementation of agricultural best management practices, but does not mention this in his report.

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References and Notes

1. CENR, *National Assessment of Harmful Algal Blooms in US Waters* (White House, Washington, DC, 2000).
2. R. J. Diaz, R. Rosenberg, *Oceanog. Mar. Biol.* **33**, 245 (1995).

Response

WINSTANLEY HAS CRITICIZED WHAT HE CALLS

"the fertilizer hypothesis" in Congressional testimony, in public comments he ghostwrote for Illinois officials, and in several speeches, including one at the annual convention of a major farm lobbying group, the American Farm Bureau Federation. (Snyder's claim that fertilizer nitrogen has not been clearly linked to Mississippi River nitrogen dismisses several modeling studies, including Goolsby's, that point to nitrate from fertilizer as the major source.) Also, while the federal action plan does refer to all sources of nitrogen, it clearly aims to cut fertilizer use in the Mississippi Basin by calling for funding of agricultural best management practices, which help reduce wasteful fertilizer application.

DAN FERBER

A Global Strategy to Defeat Invasive Species

IN "BLACK CARP AND SICK COWS" (EDITORIAL, 13 Apr., p. 169), Donald Kennedy illustrates that globalization of trade, travel, and transport can have unintended negative consequences, namely, the relocation and establishment of invasive species (1). He is justifiably concerned that there is too little awareness of this international threat and no general strategy for dealing with the invaders.

In 1996, this same concern was voiced by representatives of 80 countries and the United Nations (2). This led The Scientific Committee on Problems of the Environment, The World Conservation Union, CAB International, and invasive species experts from a wide array of disciplines to establish the Global Invasive Species Program (GISP) in 1997. GISP's mission is to employ its scientific and technical expertise to increase the ability of all nations to minimize the spread and impact of invasive species.

In its first phase of operations, GISP

has produced four books (3) and designed a database for the world's worst invaders (www.issg.org) and a toolkit of best management practices. GISP's global strategy recommends actions that governments and other bodies can take to address the invasive species problem. Its recommendations informed the development of the United States' first national invasive species management plan, released by the National Invasive Species Council in January (available at www.invasivespecies.gov).

GISP's studies indicate that prevention is more economical and feasible than controlling outbreaks of invasives. Thus, the improvement of prevention systems and their expansion to incorporate agricultural and environmental threats should be an international goal. Many invasive species have "lag periods" after introduction when small populations can be eradicated or contained; therefore, limited resources are best expended to detect and respond to newly established invasives. Ultimately, a nation's ability to address its invasive species problems is determined by its access to global information sources, the strength of its taxonomic capacity, and its willingness to cooperate with other countries.

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References and Notes

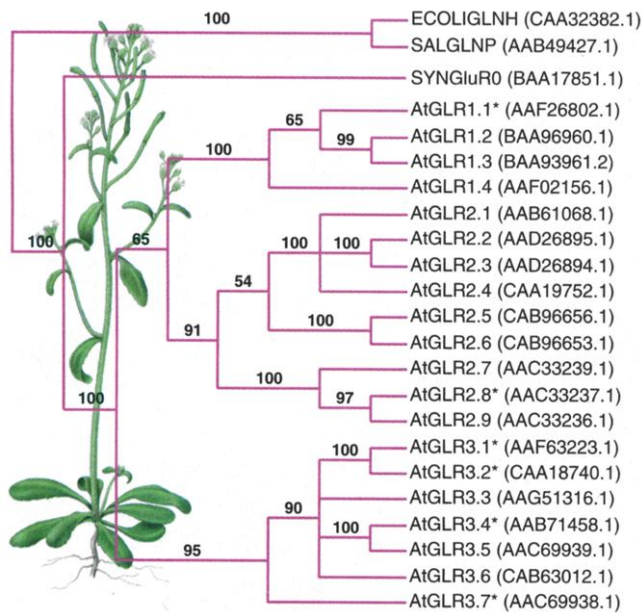
1. "Invasive species" means an alien (non-native) species whose introduction does or is likely to cause economic or environmental harm or harm to human health. U.S. Executive Order 13112, 3 Feb. 1999.
2. O. T. Sandland et al., Eds., *Norway/United Nations Conference on Alien Species* (Norwegian Directorate for Nature Management and Norwegian Institute for Nature Research, Trondheim, Norway, 1996).
3. J. A. McNeely, Ed., *The Great Reshuffling: Human Dimensions of Invasive Alien Species* (World Conservation Union, Cambridge, MA, 2001); H. A. Mooney, R. J. Hobbs, Eds., *Invasive Species in a Changing World* (Island Press, Washington, DC, 2000); C. Perrings et al., Eds., *The Economics of Biological Invasions* (Edward Elgar, Northampton, MA, 2000); C. Shine et al., *A Guide to Designing Legal and Institutional Frameworks on Alien Invasive Species* (IUCN, World Conservation Union, Bonn, Germany, 2000).

The Identity of Plant Glutamate Receptors

ION CHANNELS ARE IMPORTANT IN THE perception and transduction of environmental signals in essentially all organisms. Plants are no exception. Completion of the *Arabidopsis* genome-sequencing project has revealed that among the 600 *Arabidopsis* genes predicted to encode membrane transport proteins of one sort or another are 20

Letters to the Editor

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Phylogenetic relationships within the Arabidopsis GLR gene family. The accession number is in parentheses. Asterisks, genes with an identified full-length cDNA. Amino acid sequences can be found at <http://www.pasteur.fr/recherche/banques/LGIC/LGIC.html>

apparent homologs of animal ionotropic glutamate receptors (GLRs). In animals, these ligand-gated ion channels conduct cations across nerve cell membranes after being activated by glutamate and related neurotransmitters. The plant and animal genes share an overall secondary structure and six domains of functional importance (1), but they are sufficiently divergent that their function can-

not be deduced from sequence alone. The evidence obtained to date indicates that they participate in light signal transduction and Ca^{2+} homeostasis (2). Here, we would like to propose the adoption of a naming convention that is based on the phylogenetic relationship of the group.

In this scheme, the 20 *Arabidopsis* glutamate receptor genes are divided into three phylogenetically distinct clades, on the basis of results from parsimony analysis with bacterial amino acid binding proteins as outgroups (1). Each node is strongly supported by high bootstrap values (91–100). Each clade was assigned a number X, and the genes within a clade were each numbered consecutively with a separate value Y. Our proposal is that each gene be named AtGLRX.Y. Splice variants are denoted with lower-case letters (AtGLR3.1a and AtGLR3.1b for Genbank AF079999 and AF038557, respectively, for example). GLRs from other plant species are also accommodated by this nomenclature. For ex-

ample, the *Brassica napus* glutamate receptor (Genbank AF109392) belongs to sub-family 2 and thus would be named BnGLR2.Y.

Widespread adoption of this nomenclature will eliminate confusion as efforts intensify to learn more about the functions of these plant genes.

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References and Notes

1. J. Chiu et al., *Mol. Biol. Evol.* **16**, 826 (1999).
2. H. M. Lam et al., *Nature* **396**, 125 (1998); E. R. Brenner et al., *Plant Physiol.* **124**, 1615 (2000); S. A. Kim et al., *Plant Cell Physiol.* **42**, 74 (2001); K. L. Dennison, E. P. Spalding, *Plant Physiol.* **124**, 1511 (2000).
3. K. Q. Chen et al., *Nature* **402**, 817 (1999).

CORRECTIONS AND CLARIFICATIONS

NEWS FOCUS: "Perfecting the art of the science deal" by David Malakoff (4 May, p. 830). In the "Science lobby standouts" table, the final entry should have read "Consortium for Oceanographic Research & Education." In the "Tools of the trade" sidebar (p. 833), the first sentence stated the number of members of Congress incorrectly. The number is 535, not 545.

NEWS OF THE WEEK: "Smithsonian Institute: Plan to close zoo lab draws fire" by Elizabeth Pennisi (13 Apr., p. 183). This last sentence of the article should have read "...[the Smithsonian Institute's] stated mission of the increase and diffusion of knowledge...."

PERSPECTIVE: "A kinase to dampen the effects of cocaine?" by A. Gupta and L. -H. Tsai (13 Apr., p. 236). In the first paragraph, the sentence "The CNS usually adapts to chronic cocaine exposure by rendering the pathways that are stimulated by cocaine more resistant to the activity of this drug."

THE ATGLRS FAMILY

cDNA			Genomic (BAC)			
Full-length			Protein ID			
I	<i>AtGLR1.1</i> [*]	AF079998	AC016829	T6K12.27	At3g04110	AAF26802.1
	<i>AtGLR1.2</i> [*]		AB020745	MJE7.3	At5g48400	BAA96960.1
	<i>AtGLR1.3</i> [*]		AB020745	MJE7.4	At5g48410	BAA96961.2
	<i>AtGLR1.4</i> [*]		AC009853	F21O3.23	At3g07520	AAF02156.1
II	<i>AtGLR2.1</i> [*]		AF007271	T21B4.10	At5g27100	AAB61068.1
	<i>AtGLR2.2</i> [*]		AC007266	F27A10.3	At2g24720	AAD26895.1
	<i>AtGLR2.3</i> [*]		AC007266	F27A10.2	At2g24710	AAD26894.1
	<i>AtGLR2.4</i> [*]		AL031004	F28M20.100	At4g31710	CAA19752.1
	<i>AtGLR2.5</i> [*]		AL360314	F21I11.100	At5g11210	CAB96656.1
	<i>AtGLR2.6</i> [*]		AL360314	F21I11.70	At5g11180	CAB96653.1
	<i>AtGLR2.7</i> [*]		AC005315	T9I4.20	At2g29120	AAC33239.1
	<i>AtGLR2.8</i> [*]	AJ311495	AC005315	T9I4.19	At2g29110	AAC33237.1
	<i>AtGLR2.9</i> [*]		AC005315	T9I4.18	At2g29100	AAC33236.1
III	<i>AtGLR3.1</i>	AF079999	AC002329	F5J6.2	At2g17260	AAF63223.1
	<i>AtGLR3.2</i>	AF159498	AL022604	F23E12.150	At4g35290	CAA18740.1
	<i>AtGLR3.3</i> [*]		AC025815	T8D8.1	At1g42540	AAG51316.1
	<i>AtGLR3.4</i> [#]	AF167355	AC000098	YUP8H12.19	At1g05200	AAB71458.1
	<i>AtGLR3.5</i> [*]		AC005700	T32F6.9	At2g32390	AAC69939.1
	<i>AtGLR3.6</i> [*]		AL133452	F26O13.120	At3g51480	CAB63012.1
	<i>AtGLR3.7</i> ^{**}	AF210701	AC005700	T32F6.8	At2g32400	AAC69938.1

^{*}AGI, *Nature*, **408**, 796 (2000). [†]AtGLR1.1 was named AtGLR1 in Lam et al. (2). [‡]AtGLR2.1 was named AtGLR3 in Chiu et al. (1). [§]CDNA was cloned and named GluR9 (AJ311495). ^{||}AtGLR3.1 was named AtGLR2 in Lam et al. (2). A cDNA representing a splice variant of AtGLR3.1 was also cloned (ACL1) and its sequence was submitted to genbank (AF038557). [¶]AtGLR3.2 was named AtGLR2 in Kim et al. (2). [#]AtGLR3.4 was named AtGLR4 in Chiu et al. (1). A cDNA was cloned and named GLR3 (AF167355). A cDNA representing a splice variant was also cloned and named GLR4 (AF183932). ^{**}cDNA was cloned and named GLR5 (AF210701).