



**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  $\text{Ca}^{2+}$  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 opiate" should have read "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> <sup>†</sup>	AF079998	AC016829	T6K12.27	At3g04110	AAF26802.1
	<i>AtGLR1.2</i> <sup>†</sup>		AB020745	MJE7.3	At5g48400	BAA96960.1
	<i>AtGLR1.3</i> <sup>†</sup>		AB020745	MJE7.4	At5g48410	BAA96961.2
	<i>AtGLR1.4</i> <sup>†</sup>		AC009853	F21O3.23	At3g07520	AAF02156.1
II	<i>AtGLR2.1</i> <sup>†</sup>		AF007271	T21B4.10	At5g27100	AAB61068.1
	<i>AtGLR2.2</i> <sup>†</sup>		AC007266	F27A10.3	At2g24720	AAD26895.1
	<i>AtGLR2.3</i> <sup>†</sup>		AC007266	F27A10.2	At2g24710	AAD26894.1
	<i>AtGLR2.4</i> <sup>†</sup>		AL031004	F28M20.100	At4g31710	CAA19752.1
	<i>AtGLR2.5</i> <sup>†</sup>		AL360314	F2111.100	At5g11210	CAB96656.1
	<i>AtGLR2.6</i> <sup>†</sup>		AL360314	F2111.70	At5g11180	CAB96653.1
	<i>AtGLR2.7</i> <sup>†</sup>		AC005315	T9I4.20	At2g29120	AAC33239.1
	<i>AtGLR2.8</i> <sup>†</sup>	AJ311495	AC005315	T9I4.19	At2g29110	AAC33237.1
	<i>AtGLR2.9</i> <sup>†</sup>		AC005315	T9I4.18	At2g29100	AAC33236.1
III	<i>AtGLR3.1</i> <sup>†</sup>	AF079999	AC002329	F5J6.2	At2g17260	AAF63223.1
	<i>AtGLR3.2</i> <sup>†</sup>	AF159498	AL022604	F23E12.150	At4g35290	CAA18740.1
	<i>AtGLR3.3</i> <sup>†</sup>		AC025815	T8D8.1	At1g42540	AAG51316.1
	<i>AtGLR3.4</i> <sup>†</sup>	AF167355	AC000098	YUP8H12.19	At1g05200	AAB71458.1
	<i>AtGLR3.5</i> <sup>†</sup>		AC005700	T32F6.9	At2g32390	AAC69939.1
	<i>AtGLR3.6</i> <sup>†</sup>		AL133452	F26O13.120	At3g51480	CAB63012.1
	<i>AtGLR3.7</i> <sup>†</sup>	AF210701	AC005700	T32F6.8	At2g32400	AAC69938.1

<sup>†</sup>AGI, *Nature*, 408, 796(2000). <sup>†</sup>AtGLR1.1 was named AtGLR1 in Lam et al. (2). <sup>†</sup>AtGLR2.1 was named AtGLR3 in Chiu et al. (1). <sup>†</sup>cDNA was cloned and named GluR9 (AJ311495). <sup>†</sup>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). <sup>†</sup>AtGLR3.2 was named AtGLR2 in Kim et al. (2). <sup>†</sup>AtGLR3.4 was named AtGLR4 in Chiu et al. (1). A cDNA was cloned and named GLUR3 (AF167355). A cDNA representing a splice variant was also cloned and named GLR4 (AF183932). <sup>†</sup>cDNA was cloned and named GLR5 (AF210701).