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COLD1: a cold sensor in rice

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Rice (*Oryza sativa* L.) is one of the most important food crops in the world. Originating in tropical and subtropical regions, rice cultivars are grouped into two major subspecies, *indica* (*O. sativa* ssp. *indica*) and *japonica* (*O. sativa* ssp. *japonica*). *indica* cultivars grown in low latitude areas are sensitive to low temperature, while *japonica* cultivars with more resistance to cold temperature are grown in temperate and frigid zones [1,2]. Low temperature is a major factor that affects rice production and distribution worldwide. Scientists have been making great efforts to identify genes that control chilling tolerance traits of rice. However, the molecular mechanism underlying the chilling adaptation of *japonica* is unknown.

Recently, the research group led by Kang Chong in Institute of Botany, Chinese Academy of Sciences successfully cloned a QTL named *COLD1* (*Chilling-Tolerance Divergence 1*) [3]. One of the SNPs in this locus arisen during domestication confers chilling tolerance in *japonica* subspecies. This study also discovered that *COLD1* interacts with G-protein α subunit and functions in accelerating GTPase activity. Moreover, they provided elegant data showing that extracellular Ca^{2+} influx and net cytosolic Ca^{2+} concentration are mediated by *COLD1* in response to chilling stress. Their study, for the first time, uncovers a potential membrane-localized cold sensor in plants.

To identify chilling-tolerance-divergence (COLD), Ma et al. first generated recombinant inbred lines by crossing a chilling tolerant *japonica* cultivar Nipponbare (NIP) and a chilling-sensitive *indica* cultivar 93-11. Using molecular markers and QTL analysis, they defined *COLD1* on chromosome 4. Fine mapping and DNA sequence comparisons revealed that a single nucleotide A at the 15th nucleotide in the 4th exon of *COLD1* in NIP is substituted to T in 93-11, leading to a change in 187th amino acid from Lys to Met. The homozygous $COLD1^{NIP/NIP}$ lines of near-isogenic lines (NILs) containing the $COLD1^{NIP}$ locus in the 93-11 genetic background exhibit higher chilling tolerance than 93-11. Furthermore, the authors showed that $COLD1^{jap}$-overexpression lines significantly enhance survival rates after chilling treatment compared to WT whereas *cold1-1* mutant and *COLD1* antisense transgenic lines are chilling sensitive. These results suggest that *COLD1* is a key gene modulating the chilling tolerance in *japonica* rice.

Importantly, Ma et al. discovered that a single-nucleotide polymorphism SNP2 in the 4th exon is associated with chilling tolerance. By sequencing the full-length *COLD1* gene in 127 rice cultivars, they found that all *japonica* accessions and five *O. rufipogon* samples originating from China with stronger chilling tolerance have nucleotide A at the SNP2 sites. The chilling sensitive *indica* cultivars, however, have either T or C at the SNP2 site. From a geographical point of view, these *japonica* cultivars are mostly distributed in northeast Asia, the US, and higher elevation areas of the southeast Asia, whereas, *indica* cultivars are grown in southern and south-eastern Asia. Phylogenetic and nucleotide diversity analysis suggested that $SNP2^{A}$ in *COLD1* is likely to be derived from the Chinese *O. rufipogon* during *japonica* rice domestication.

Cold shock could activate the Ca^{2+} signaling pathway in plant cells, stimulating downstream cold signal transduction

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and transcription factor activation [4]. Little is known about the molecular mechanism of low temperature sensing and Ca^{2+} signaling. Ma et al. found that *COLD1* encodes a protein localized on plasma membrane and endoplasmic reticulum (ER). Like its orthologues GTG1/2 in *Arabidopsis*, COLD1 interacts with rice G-protein α subunit 1 (RGA1). However, COLD1 is different from GTG1/2 in intrinsic GTPase activity [5]. COLD1 alone does not have GTPase activity, but it can promote GTPase activity of RGA1. Therefore, COLD1 is a G-protein signaling regulator. Strikingly, they found that co-expressing $COLD1^{jap}$ and *RGA1* rapidly activates inward current after cold treatment by using electrode voltage clamp approach. Furthermore, both Ca^{2+} influx and Ca^{2+} concentration in the cytoplasm showed a remarkable increase upon cold shock. Similar to TRPV which is an ion channel acting as a temperature sensor in mammalians, COLD1 is strongly suggested as a cold sensor in plants. In further study, it would be exciting to find out if COLD1 itself works as a potential calcium channel or a subunit of such ion channel.

In summary, Ma et al. reported the first plasma and ER-membrane protein involved in cold sensing in plants. Their findings contribute to our understanding of cold perception in plants. Meanwhile, using these NILs to confer chilling tolerance without negative effects on grain yield via molecular breeding techniques will definitely benefit human beings.

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