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农学院张国平教授团队在大麦耐盐调控机制研究上取得新进展

编辑：傅炜琳 来源：农业与生物技术学院办公网 时间：2020年06月24日 访问次数:184

2020年6月18日，Plant Physiology在线发表了浙江大学作物科学研究所张国平教授团队题为“Calmodulin HvCaM1 negatively regulates salt tolerance via modulation of HvHKT1s and HvCAMTA4”的研究论文，揭示了大麦钙调蛋白HvCaM1负向调控耐盐性的新机制，对作物耐盐遗传改良具有重要意义。

土壤盐害是全球作物生产面临的主要非生物胁迫，而Ca²⁺信号及其结合蛋白介导的盐响应信号转导途径一直是植物耐盐分子调控机理研究的热点。在禾谷类作物中，大麦 (*Hordeum vulgare* L.) 的耐盐性显著强于水稻和小麦等其它作物，但其耐盐分子机制尚未阐明。张国平教授团队前期从西藏野生大麦 (XZ16和XZ26等) 根蛋白组中鉴定到一个响应盐胁迫的新型钙调蛋白HvCaM1 (Wu和Shen等, Proteomics, 2014; Shen等, Plant Physiol Biochem, 2018)。

系统发育树分析表明CaM1起源于绿藻，是植物中序列高度保守的Ca²⁺结合蛋白。作为典型的Ca²⁺结合蛋白，HvCaM1在大麦根的表达量高于地上部，且在根中柱和维管束组织中表达丰度较高。RNAi沉默HvCaM1基因显著增强大麦的耐盐性，同时降低Na⁺从根部往地上部的转运。HvCaM1与一个转录激活因子HvCAMTA4互作，通过下调HvHKT1;5的表达减少木质部Na⁺向上运输，并通过上调HvHKT1;1的表达增加Na⁺向根部回流，从而降低地上部Na⁺积累，增强大麦耐盐性。此前，该课题组报道了HvHKT1;5负向调控大麦耐盐的分子机制 (Huang和Kuang等, Plant Physiol, 2020)。目前，HvCaM1和HvHKT1;5研究的相关发明专利均已获得授权 (ZL201710923685.0和ZL201710958435.0)。HvCaM1和HvHKT1;5协同调控大麦耐盐性的发现，不仅丰富了作物耐盐理论，对大麦以及其它禾谷类作物的耐盐遗传改良具有重要指导价值。

浙江大学作物所沈秋芳博士后为该论文第一作者，张国平教授为通讯作者，吴德志副教授和西悉尼大学陈仲华教授等参与了该研究的部分工作。该研究受国家自然科学基金、现代农业产业技术体系以及中国博士后科学基金等项目的资助。

论文链接：<http://www.plantphysiol.org/content/early/2020/06/18/pp.20.00196>

(作物科学研究所供稿)

Other

Calmodulin HvCaM1 negatively regulates salt tolerance via modulation of HvHKT1s and HvCAMTA4

Qifang Shen, Liangbo Fu, Tingting Su, Lingzhen Ye, Lu Huang, Liuliu Kuang, Liyuan Wu, Dezhi Wu, Zhonghua Chen, Guoping Zhang

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Abstract

Calcium (Ca²⁺) signaling modulates sodium (Na⁺) transport in plants; however, the role of the Ca²⁺-sensor calmodulin (CaM) in salt tolerance is elusive. We previously identified a salt-responsive calmodulin (HvCaM1) in a proteome study of barley (*Hordeum vulgare*) roots. Here, we employed bioinformatic, physiological, molecular and biochemical approaches to determine the role of HvCaM1 in barley salt tolerance. CaM1s are highly conserved in green plants and probably originated from ancestors of green algae of the Chlorophyta order. HvCaM1 was mainly expressed in roots and was significantly up-regulated in response to long-term salt stress. Localization analyses revealed that HvCaM1 is an intracellular signaling protein that localizes to the root stele and vascular systems of barley. After treatment with 200 mM NaCl for 4 weeks, HvCaM1 knock-down (RNAi) lines showed significantly larger biomass but lower Na⁺ concentration, xylem Na⁺ loading, and Na⁺ transportation rates in shoots compared with overexpression (OE) lines and wild-type plants. Thus, we propose that HvCaM1 is involved in regulating Na⁺ transport, probably via certain Class I high-affinity potassium transporters (HvHKT1.5 and HvHKT1.1)-mediated Na⁺ translocation in roots. Moreover, we demonstrated that HvCaM1 interacted with a CaM-binding transcription activator (HvCAMTA4), which may be a critical factor in the regulation of HKT1s in barley. We conclude that HvCaM1 negatively regulates salt tolerance, probably via interaction with HvCAMTA4 to modulate the downregulation of HvHKT1.5 and/or upregulation of HvHKT1.1 to reduce shoot Na⁺ accumulation under salt stress in barley.

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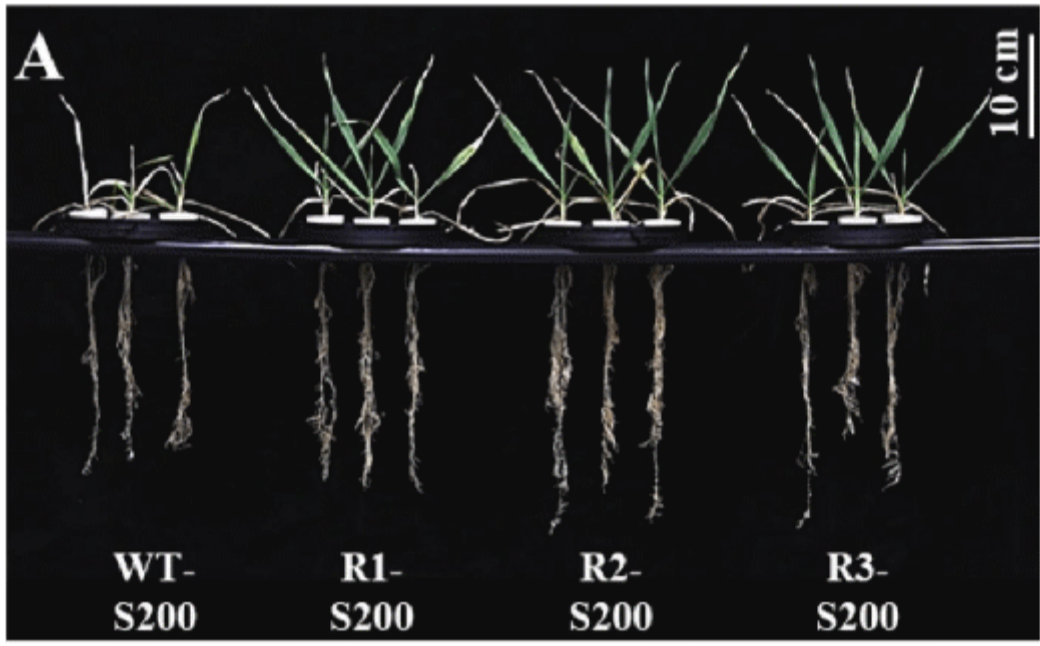
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The HKT Transporter HvHKT1.5 Negatively Regulates Salt Tolerance
Li Huang et al. *Plant Physiology*, 2020

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Magdalena M. Jankowska, *Plant Physiology*, 2019

A Na⁺ Transporter Negatively Regulates Salt Tolerance
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Calmodulin methylation: another layer of regulation in calcium signaling
Nancy R. Hofmann, *Plant Cell*, 2013



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