

植物抵御冷胁迫的分子机制研究进展

李金涛¹, 景莉清¹, 逯丹阳¹, 闫欣¹, 李露婷¹, 唐乐瑶¹, 吴一凡¹,
郑亚亚¹, 赵强², 樊海燕^{1*}

(1. 信阳师范大学 生命科学学院, 河南 信阳 464000;

2. 河南省科研平台服务中心, 河南 郑州 450003)

摘要: 冷胁迫是一种普遍存在的非生物胁迫因子, 严重制约植物的生长发育与全球粮食安全。系统综述了冷胁迫(0~15℃)引发的关键生理生化损伤(包括电解质渗漏、光合与呼吸抑制、活性氧(ROS)爆发及水分调节失衡); 深入探讨了植物感知低温信号的分子机制(涉及COLD1/RGA1、Ca²⁺通道、RLKs等关键感受器)及其信号转导通路(如Ca²⁺信号流、MAPK级联反应和ICE1-CBF-COR核心模块); 并详细阐述了植物抵御低温的多种分子策略, 涵盖渗透保护系统、ROS清除机制、海藻糖信号传导、S-酰基化翻译后修饰以及低温保护蛋白(LEA、AFP、CSPs)的诱导表达。最后, 展望了植物耐冷性研究的未来方向, 强调深化冷感受器结构与功能解析、揭示物种间调控网络差异、探索新型调控机制(如相分离、表观遗传)以及推动多基因协同编辑育种的重要性, 为作物耐冷性遗传改良提供理论基础与创新思路。

关键词: 植物; 生理生化; 冷胁迫; 信号转导; 分子机制

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Molecular mechanisms of plant resistance to cold stress

LI Jintao¹, JING Liqing¹, LU Danyang¹, YAN Xin¹, LI Luting¹, TANG Leyao¹,
WU Yifan¹, ZHENG Yaya¹, ZHAO Qiang², FAN Haiyan^{1*}

(1. College of Life Sciences, Xinyang Normal University, Xinyang 464000, China;

2. Henan Scientific Research Platform Service Center, Zhengzhou 450003, China)

Abstract: Cold stress is a pervasive abiotic stress factor that severely constrains plant growth and development and threatens global food security. The key physiological and biochemical damages triggered by cold stress (0–15 °C), including electrolyte leakage, inhibition of photosynthesis and respiration, reactive oxygen species (ROS) burst, and water homeostasis dysregulation were systematically synthesized. The molecular mechanisms underpinning low-temperature signal perception in plants, involving critical sensors such as COLD1/RGA1, Ca²⁺ channels, and receptor-like kinases (RLKs), along with their downstream signal transduction pathways (e.g., Ca²⁺ flux, MAPK cascade reactions, and the ICE1-CBF-COR core module) were comprehensively explored. Furthermore, the diverse molecular strategies employed by plants to counteract low temperatures, encompassing osmotic protection systems, ROS scavenging mechanisms, trehalose signaling, S-acylation post-translational modifications, and the induced expression of cryoprotective proteins (LEA, AFP, CSPs) were elaborated. Finally, future research trajectories were outlined in plant cold tolerance, emphasizing the imperative to: decipher the structure-function relationships of cold sensors, reveal interspecies regulatory network divergences, explore novel

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作者简介: 李金涛(1982—), 男, 河南新郑人, 副教授, 博士, 主要从事植物分子生物学研究; 樊海燕(1983—), 女, 山东临朐人, 讲师, 博士, 主要从事作物分子育种研究。

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regulatory mechanisms (e.g., phase separation, epigenetic regulation), and advance multi-gene coordinated editing for breeding. This review aims to provide a solid theoretical foundation and innovative perspectives for the genetic improvement of crop cold tolerance.

Key words: plant; physiological and biochemical; cold stress; signal transduction; molecular mechanism

0 引言

全球气候变化导致极端低温事件频发,对农业生产构成严峻挑战。水稻 (*Oryza sativa* L.)、玉米 (*Zea mays* L.)、大豆 (*Glycine max* L.) 等作为全球主粮作物,其驯化起源多集中于热带与亚热带地区^[1-3]。这种起源特性使得众多现代栽培品种对冷胁迫高度敏感。低温冷害可导致作物显著减产,例如,水稻苗期和孕穗期遭遇低温常引起死苗、结实率低(减产高达30%~40%),造成严重经济损失^[4-5]。因此,深入解析植物响应与抵御冷胁迫的分子网络,发掘关键耐冷基因资源,并将其应用于作物种质遗传改良,对于增强作物环境适应性、稳定粮食产量、保障全球粮食安全具有重大战略意义。

本文旨在综述近年来植物耐冷性分子机制研究的重要进展,重点关注冷胁迫造成的生理生化影响、冷信号的感知与转导途径以及植物进化出的多层次分子防御策略,并对未来研究方向提出见解。

1 冷胁迫对植物生理生化的影响

植物在0~15℃时发生的低温胁迫,称为冷胁迫。水稻遭受冷胁迫后,会引起植株叶片皱缩、生长迟缓甚至死亡等现象。生理生化方面,会引起细胞膜流动性减慢、细胞质渗漏、脂质过氧化、叶绿素降解、ROS积累等方面损伤^[4-5]。

1.1 水分调节失衡

低温环境下,植株体内水分流失的同时,根系吸收能力下降,水分平衡失调,造成植株脱水。研究表明,水通道蛋白参与了冷胁迫下水分平衡的调节^[6-7]。拟南芥中,水通道蛋白AtPIP2;5通过增加水分的传导提高植株的耐寒能力^[8]。水稻中,水稻12号染色体的耐冷位点CTS-12通过保卫细胞调节气孔开闭控制水分流失,增强植株抗寒性^[9]。

1.2 细胞膜系统破坏,电解质渗透

细胞膜是植物最早感受冷胁迫的场所。遭受冷胁迫后,膜脂排列紊乱变为凝胶态,膜蛋白排列无序,膜出现裂隙甚至破裂,通透性增大,电解质

外泄。提高*OsVPI*基因表达水平后,水稻植株电解质渗透性降低,植株对冷胁迫具有更强的耐受性^[10]。相反,*OsLPXC*基因突变后,水稻植株的电解质渗透性增加,植株对冷胁迫更敏感^[11]。

1.3 光合作用、呼吸作用和氧化还原反应受损

冷胁迫影响气孔开合和叶绿素的生物合成,阻碍叶绿体发育,造成光系统II(PS II)损伤^[12]。光合速率下降,导致植物对氧气的利用率降低,植物新陈代谢过程中ROS的产生和清除的平衡遭到破坏,导致了线粒体膜硬化和活性氧SOD积累过多,最终对植物体的生命活动造成毒害作用^[13-14]。

2 植物中冷信号感知与传递的分子机制

细胞膜是植物细胞感知冷胁迫的场所,COLD1/RGA1、Ca²⁺通道和质膜定位受体样激酶(Receptor-like kinases, RLKs)等是植物中重要的冷感受器^[15]。其中,COLD1/RGA1和Ca²⁺通道研究较多。*COLD1*编码一个G蛋白调节因子,定位于质膜和内质网,是水稻中发现的首个冷感受器。当植物遭受冷胁迫时,COLD1与G蛋白 α 亚基RGA1发生互作而激活Ca²⁺通道,促进Ca²⁺内流,触发低温响应的信号通路^[16]。植物感知低温后,通过MAPK级联信号传递和ICE1-CBF-COR模块快速对冷胁迫做出响应(图1)^[17]。

2.1 冷信号的感知

环核苷酸门控CNGCs通道(Cyclic Nucleotide-Gated Ca²⁺ Channels)、谷氨酸受体样通道(Glutamate Receptor-Like Channels, GLRs)和MCA1/MCA2互补活性通道(Mid1-Complementing Activity 1/2)是主要的冷信号感知Ca²⁺通道蛋白^[18]。CNGCs是一类定位于质膜的非选择性阳离子通道,通过结合G蛋白耦联受体,激活cAMP或cGMP环化酶活性,释放cAMP或cGMP开启Ca²⁺通道^[19]。水稻中,环核苷酸门控通道OsCNGC14、OsCNGC16和OsCNGC9均正调控水稻抵御冷胁迫的过程。OsCNGC14和OsCNGC16基因突变后,低温诱导的钙离子内流减低,植株对冷胁迫敏感^[20]。OsCNGC9控制的Ca²⁺通道开闭受OsSAPK8激酶磷酸化调节,冷胁迫

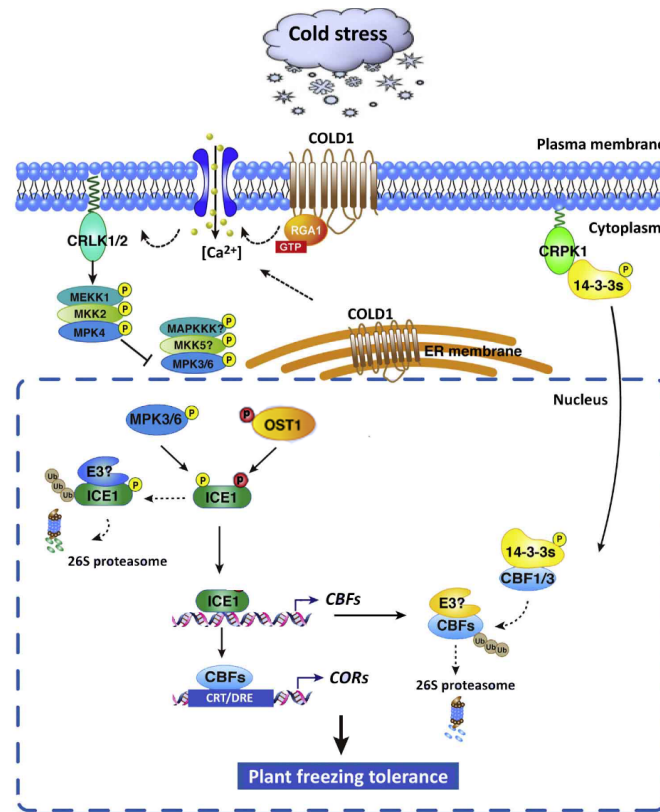


图 1 植物冷信号转导过程

Fig. 1 Low-temperature signal transduction in the plant

迫条件下,OsCNGC9被磷酸化, Ca^{2+} 通道开放,水稻植株耐寒性增强^[21]。MCA1和MCA2是拟南芥中的两个钙渗透性机械敏感通道蛋白,通过感知胞外的牵张、压力、重力和渗透压等机械信号转变为胞内 Ca^{2+} 流,传递冷信号^[22]。

2.2 MAPK级联反应传递冷信号

植物激酶级联反应是植株冷胁迫应答的重要过程,通过MAPK级联反应,将冷信号与细胞应答联系起来^[23]。MAPKK与2个钙调蛋白调节的受体激酶CRLK1和CRLK2(Calmodulin-Regulated Receptor-Like Kinase)接收到冷信号后,通过MEKK1-MEK2-MPK4激酶途径传递信号。进一步,MEKK1-MEK2-MPK4通过拮抗MKK4/5-MPK3/6反应,抑制MPK3/6对ICE1(Inducer of CBF expression 1)磷酸化过程,增强植物抵御冷胁迫能力^[24]。此外,冷胁迫下,CRPK1蛋白激酶(Cold-Responsive Protein Kinase 1)能够磷酸化14-3-3蛋白,磷酸化后的14-3-3作为穿梭蛋白从细胞质进入细胞核,与细胞核定位的CBF1/3结合并促进其降解,负调控植物耐冷过程^[25]。

2.3 ICE1-CBF-COR冷响应过程

ICE1是CBF(C-Repeat Binding Factor)的主要调节因子,属于bHLH转录因子家族成员,通过ICE1-CBF-COR通路正调控植物耐冷能力。ICE1能够结合到CBF基因的启动区调节CBF的表达,而CBF作为转录因子激活低温应答基因COR(Cold-Responsive)的表达抵御冷胁迫过程^[17]。拟南芥中,MPK3/MPK6通过磷酸化ICE1(Inducer of CBF expression 1),引起其稳定性降低,负调控植物耐冷过程。相反,水稻中,ICE1被OsMPK3磷酸化后稳定性提高,冷胁迫耐受性增强^[26]。这些结果表明冷信号感知与传递的核心分子模块在不同物种间存在功能分化。拟南芥OST1,也称为SnRK2.6(Sucrose Non-Fermenting 1-Related Protein Kinase 2),是冷环境信号转导的关键调控因子。冷胁迫下,OST1能够结合并磷酸化ICE1,阻遏ICE1被26S蛋白酶体降解,提高CBF转录活性,增强植株耐冷能力^[27, 28]。HOS1(High Expression Of Osmotically Responsive Gene 1)编码一个E3 ubiquitin ligase,通过泛素化ICE1负调控植株耐冷过程,蛋白激酶BIN2(Brassinosteroid-

Insensitive2)通过磷酸化ICE1,促进HOS1泛素化并降解ICE1,调节植株耐冷反应^[29-30]。

3 植物抵御低温的机制研究

植物对低温冷害的适应是一个复杂的生物学过程,为减少低温造成的伤害,植物进化出一系列耐冷机制,包括渗透保护系统、氧化还原系统、海藻糖、S-酰基化以及低温保护蛋白等,以此来抵御冷胁迫造成的细胞内渗透压失衡和有毒物质积累,维持细胞完整性及内部平衡。

3.1 渗透保护系统在植物抵御冷胁迫中的作用

冷胁迫使植物体内水分流失,渗透保护系统通过有机渗透调节物质(统称为渗透保护剂,如糖类、脯氨酸以及季氮化合物等),从环境中吸收水分缓冲植物体内缺水^[31],进而减轻冷胁迫造成的伤害。拟南芥中,麦芽糖和葡萄糖分别通过叶绿体上的糖转运体MEX1(Maltose Transporter)和pGlcT(Plastid Glucose Transporter)运输到细胞质中^[32-33]。液泡膜定位糖输出转运蛋白SWEET16(Sugars Will Eventually be Exported Transporter 16)具有转运葡萄糖、果糖和蔗糖等可溶性糖的活性,其过量表达后能显著增强拟南芥植株的耐冷能力^[34]。质子泵偶联外排蛋白ERDL6(Early Response To Dehydration Like 6)介导葡萄糖从液泡排出,其过量表达后引起拟南芥植株的耐冷能力降低^[35]。相似地,过表达拟南芥转录因子基因*bZIP62*,同样能够激活脯氨酸合成酶基因*P5CS1*(Pyrroline-5-Carboxylic Acid Synthetase)基因表达,提升游离脯氨酸含量增强抵御冷胁迫的能力^[36]。番茄(*Solanum lycopersicum*)中,*SIWRKY51*基因敲除植株对冷敏感,光合能力降低,活性氧(ROS)积累增加,脯氨酸含量明显降低。深入研究发现,*SIWRKY51*通过直接激活脯氨酸生物合成关键基因*D-1-pyrroline-5-carboxylate synthetase1 (P5CS1)*的表达调控耐冷过程^[37]。甘氨酸甜菜碱属于一类季铵型生物碱,提高茶树甜菜碱醛脱氢酶基因*CsBADH*表达水平,能够提升甘氨酸甜菜碱的水平,增加拟南芥、烟草和茶树的耐冷能力^[38]。

3.2 活性氧清除系统在植物抵御冷胁迫中的作用

冷胁迫发生后,植物细胞内膜系统遭到破坏,电子传递和氧化还原动态平衡被打破,大量的活

性氧被积累。为应对氧化损伤,植物进化出了一系列的活性氧清除系统,包括过氧化氢酶(Catalase, CAT)、超氧化物歧化酶(Superoxide Dismutase, SOD)、过氧化物酶(Peroxidase, POD)和抗坏血酸过氧化物酶(Ascorbate Peroxidase, APX)等。CAT主要负责清除光合作用及乙醛酸循环中的过氧化氢。柚子中,转录因子PtrbHLH通过正调节CAT基因的表达量控制ROS水平,赋予植株耐寒能力^[39]。木薯中,增强Cu/Zn-SOD和CAT的表达水平,能提高植株对冷胁迫的耐受性^[40]。芥菜*RCI35*(Rare Cold-Inducible 35)属于Ⅲ型POD基因家族,烟草中异源表达*CbRCI53*,植株SOD酶活增强,ROS清除能力增加,植株抵御冷胁迫能力提升^[41]。水稻中,过表达抗坏血酸过氧化物酶基因*OsAPX1*和*OsAPX2*,能显著提高植株耐受冷胁迫能力^[42]。*OsPUS1*编码一个定位于叶绿体的假尿苷合成酶,参与了叶绿体核糖体生物合成。低温条件下,*OsPUS1*突变后引起线粒体超氧根阴离子(O₂⁻)积累造成叶片白化^[43]。水稻转录因子*OsbZIP72*,通过直接结合并激活活性氧清除基因*Glutathione S-transferase*和*CDSF32*(*Thioredoxin*)的表达,调节活性氧稳态的方式增强水稻苗期耐冷性^[44]。玉米中,叶绿素水通道蛋白*TIP4;3*抑制气孔关闭,促进ROS积累,下调冷响应基因表达,负调控植株耐冷。328 bp的转座子插入造成*TIP4;3^{Hap2}*基因型品种H3K9me2和H3K9me3甲基化水平增加,引起*TIP4;3*基因表达抑制,增强了植株的耐冷性^[7]。因此,ROS是植物抵御冷胁迫中关键的信号分子,适度的ROS水平将激活防御通路,过度积累将导致植物细胞氧化损伤。

3.3 海藻糖在植物抵御冷胁迫中的功能

海藻糖是一种稳定的非还原性二糖,作为葡萄糖的储存与运输分子、能量来源以及在胁迫条件下发挥保护作用的关键应激化合物。水稻中,通过提高海藻糖合成途径中两个关键酶*OsTPS1*和*OsTPP1*的表达量,能显著提高海藻糖含量达到增强水稻植株的耐寒性^[45]。低温诱导的*OsPP2C27*能够使磷酸化的OsMAPK3和OsbHLH002直接去磷酸化,从而负调控OsMAPK3-OsbHLH002-OsTPP1信号通路,阻止冷胁迫的正调控途径持续激活,并在低温条件下维持正常生长^[46]。甜瓜中,海藻糖通过调控质膜

水通道蛋白基因 *CmPIP2;3* 的表达,促进质外体过氧化氢 (H_2O_2) 信号转运至细胞内,诱导幼苗耐冷性增强^[47]。然而,海藻糖在大多数植物体内含量极低,暗示这种非还原性二糖更倾向于发挥信号传导功能而非代谢功能。

3.4 S-酰基化在植物抵御冷胁迫中的功能

S-酰基化也称作棕榈酰化(S-acylation 或 S-palmitoylation),是一种可逆的翻译后修饰,由 S-酰基转移酶 PATs 介导将长链脂肪酸通过硫脂键连接到底物蛋白的半胱氨酸残基上。最近研究发现,在非胁迫条件下,S-酰基转移酶 MtPAT9 能够与 MtNAC80 作用并发生 S-酰基化修饰,介导其膜定位。在冷胁迫条件下,发挥去乙酰化作用的硫酯酶 MtAPT1 聚合成有酶活的四聚体^[48],催化 MtNAC80 发生去 S-酰基化并进入细胞核。在细胞核中,MtNAC80 直接激活 *MtGSTU1* 基因表达,清除 MDA 和 H_2O_2 ,发挥抵御冷胁迫的功能^[49]。

3.5 低温保护蛋白在植物抵御冷胁迫中的功能

冷胁迫条件下,胚胎晚期丰富蛋白(Late Embryogenesis Abundant Protein, LEA)、抗冻蛋白(Antifreeze Protein, AFP)和冷休克蛋白(Cold Shock Proteins, CSPs)等保护蛋白在植物体内诱导表达,缓解冷胁迫造成的伤害。胚胎晚期丰富蛋白(LEA)蛋白是一类亲水性的固有无序蛋白,有水分时保持无序状态;当细胞发生脱水时,LEA 蛋白富集,结构发生折叠、蛋白纤维交织成纤维网,维持脱水下细胞的稳定性。小麦中,提高 LEA3 蛋白水平能够增强小麦的耐冷的能力^[50]。过表达一个玉米 LEA 蛋白基因 *Dehydrin Protein*,能显著提高植株抵御冷胁迫的能力^[51]。抗冻蛋白也称为重结晶抑制蛋白(Ice Recrystallization Inhibition, IRI),越冬植物通过分泌抗冻蛋白抑制冰晶生长,防止细胞脱水。AFP I 可以作为冷保护剂,经过 AFP I 处理的番茄种子在冷胁迫下萌发率升高^[52]。对抗冻蛋白的研究多数研究集中于其抑制冰晶重结晶的体外活性,但 AFP 在植物体内是如何精准定位到冰晶形成位点(如细胞间隙或质外体)的,并不清楚。冷休克蛋白(CSPs)中的冷休克结构域(CSD)是高度保守的核酸结合结构域,识别并结合单链 DNA/RNA。冷胁迫下,冷休克蛋白作为 RNA 伴侣,保护 RNA 的正常构象不受破坏赋予植株耐冷功能^[53]。拟南芥中,过量表达 *AtCSP3* 基因,转基因植株耐寒性增强^[54]。尽管已

知 CSPs 能结合 RNA,但低温下具体保护的 RNA 靶点(如 mRNA、rRNA 或非编码 RNA)及其对翻译效率的影响仍有待解析。

4 展望

提升作物耐冷性是应对气候变化、保障粮食安全的核心课题之一^[55-56]。近年来,随着分子生物学技术的飞速发展,在主要作物中鉴定并克隆了大量关键耐冷基因(例如水稻的 *COLD1*、*OsbZIP72*、*OsSAPK6*,玉米的 *bZIP68*、*HSF21*,小麦的 *TaSnRK2.8*、*TaCIPK*,番茄的 *SINAC3* 等)^[57],显著深化了对植物冷响应调控网络的理解。先进的表型组学技术(如高光谱成像、微 CT、超分辨率显微镜)为在微观和动态层面精准量化冷胁迫表型(如膜完整性、叶绿素荧光、ROS 分布)提供了强大工具。空间转录组与单细胞测序技术的结合,有望以前所未有的分辨率揭示冷胁迫下不同组织、细胞类型中基因表达的时空特异性与互作网络,解析复杂组织(如分生组织、维管束)响应低温的异质性。CRISPR/Cas9 基因编辑技术因其高效、精准的特性,已成为同时操控多个耐冷相关基因(如优化 *CBF* 表达调控网络、修饰关键激酶/磷酸酶活性、增强保护蛋白积累)、创制聚合优良耐冷等位基因新种质的革命性手段^[58-59]。

基于当前研究进展和领域发展趋势,未来植物耐冷研究应重点关注以下几个方向:

(1)冷感受器的精细解析:尽管已知 *COLD1*、 Ca^{2+} 通道、RLKs 等参与冷感知,但其精确的感知机制(如温度敏感性结构域)、激活后的构象变化,以及与其他膜蛋白/脂质的相互作用网络仍知之甚少。利用结构生物学(冷冻电镜、X 射线晶体学)结合生物物理手段深入研究冷感受器的结构与动态变化,是揭示“温度如何转化为初始生化信号”这一根本问题的关键。

(2)物种特异性与保守性机制的深度挖掘,核心通路(如 ICE1-CBF-COR)在不同物种间存在显著功能分化(如 ICE1 磷酸化后稳定性调控的差异)。未来需系统比较不同生态型、不同驯化程度作物(特别是耐冷性差异显著的种质)的冷响应机制,鉴定关键的物种/生态型特异性调控因子和等位变异,为定向设计适应特定温度地区的耐冷品种提供靶点。

(3)超越转录调控的复杂网络:除转录因子

外,翻译后修饰(PTMs,S-酰基化^[48-49]、磷酸化、泛素化)、相分离、非编码RNA(尤其是长链非编码RNA和环状RNA)、表观遗传调控(DNA甲基化、组蛋白修饰)在冷胁迫响应中的精细调控作用亟待深入探索。例如,S-酰基化如何动态调控更多冷响应蛋白的亚细胞定位与活性?冷胁迫是否诱导形成特定的生物分子凝聚体(Condensate)以隔离或激活信号分子?这些研究将揭示调控网络的更高层次复杂性。

(4)多基因模块协同设计与智能育种:耐冷性是多基因控制的复杂数量性状。利用系统生物学整合多组学数据(基因组、转录组、蛋白组、代谢组、表型组),构建更精确的基因调控网络模型,识

别关键调控节点和协同模块至关重要。在此基础上,结合人工智能(AI)驱动的基因编辑设计工具,实现多基因/模块的协同优化(如平衡抗逆性与生长发育),并利用预测模型指导亲本选配和后代筛选,将极大加速培育高产、稳产、广适的智能化耐冷新品种。

综上所述,深化基础机制研究与推动技术创新的深度融合,是未来突破植物耐冷性研究瓶颈、实现作物抗逆与稳产育种目标的核心驱动力。这不仅是提升我国农业生物技术国际竞争力的关键,更是应对全球气候变化挑战、保障国家粮食安全的迫切需求。

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