



评述

水稻理想株型基因*IPA1*研究进展侯鹰翔^{1,2}, 费思恬^{1,2}, 宋松泉¹, 王洁敏^{3,4,5}, 王伟平^{3,4,5*}, 张超^{1,2*}

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摘要 随着水稻功能基因组学的深入解析, 越来越多的水稻(*Oryza sativa*)产量相关基因被相继克隆. 理想株型基因*IPA1*(*OsSPL14*)编码SQUAMOSA(SQUA)植物特异性转录因子, 协同调控水稻的产量与抗病性, 打破了单个基因不可能同时实现增产和抗病的传统观点. 本文综述了*IPA1*基因在调控水稻理想株型、其他生长发育和生物与非生物胁迫中的重要功能, 以及育种应用现状, 提出了*IPA1*调控网络中需要进一步研究的问题, 展望了未来的应用潜力与育种策略. *IPA1*通过整合多种信号途径, 形成了复杂的分子调控网络, 从而能够协同增产与抗性, 为水稻高产与抗性育种提供了重要的理论基础和基因资源.

关键词 水稻, 理想株型, *IPA1*, 产量, 胁迫, 育种

水稻(*Oryza sativa*)作为世界上最重要的粮食作物之一, 养育全球50%以上的人口, 在亚洲以稻米为主食的人口比例达到60%, 提高水稻单产是育种家们普遍追求的目标. 我国水稻育种史上出现过两次重要突破, 第一次是始于20世纪50年代的矮化育种, 以黄耀祥先生培育的“广场矮”^[1]和国际水稻研究所育成的品种IR8^[2]为标志, 实现了水稻产量的第一次飞跃, 被誉为我国水稻育种的第一次“绿色革命”. 第二次则是70年代, 袁隆平先生领衔的科学家们成功实现杂交水稻三系配套让水稻单产在矮秆品种的基础上又一次大幅提升, 随后光温敏雄性核不育材料的发现使两系法杂交

稻比三系法杂交稻产量更上一个台阶, 杂种优势的利用使我国水稻单产再次出现革命性飞跃^[3]. 为了进一步实现水稻产量的第三次突破, 科学家寄希望于籼粳亚种间杂种优势利用和理想株型育种等^[4,5].

20世纪60年代, 澳大利亚科学家Donald首次提出作物理想型(crop ideotype)概念^[6], 即有利于作物充分利用光照和营养、促进生长从而提高群体产量的株型模式. 通过塑造理想株型培育水稻新品种, 对提高产量和资源利用效率具有重要意义. 过去的几十年, 科学家们在育种实践中提出了众多理想株型模式, 包括“半矮秆丛生快长”模式^[7]、“立叶直穗”模式^[8]、“重穗型”模

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式^[9]、“少蘖大穗”模式^[10]和“上三叶长、直、窄、凹、厚”模式^[11]等。综合来看,理想株型是指通过改变植株结构,包括株高、叶形、粒型、穗型等,使多种农艺性状实现最佳组合,群体光合效率达到最大,从而获得最大限度的群体产量^[12]。2007年,中国科学家李家洋院士在此基础上正式提出理想株型(ideal plant architecture)概念^[13],并通过*IPA1*(ideal plant architecture 1)基因的克隆和鉴定验证了此概念^[14]。21世纪以来,随着分子生物学的发展,理想株型的分子机制研究取得重要突破,克隆了诸多调控株型的重要基因,例如*IPA1*^[14,15],*DEP1*(dense panicle 1)^[16]等,为水稻分子设计育种奠定基础。

2010年,李家洋团队^[14]和Motoyuki Ashikari团队^[15]在*Nature Genetics*期刊“背靠背”发表了*IPA1*的研究文章,揭示了*IPA1/WFP*(wealthy farmer's panicle)是水稻理想株型的主效基因。理想株型基因*IPA1*优异等位使水稻分蘖减少、穗子增大、每穗粒数增多和千粒重增加,同时根系发达、茎秆粗壮、抗倒伏能力增强,从而提高水稻产量^[14,15]。*IPA1*的发现被认为是水稻科学的重大突破,Wang和Wang^[17]认为*IPA1*可能是下一个“绿色革命”基因。近年来,围绕*IPA1*的功能与机制研究取得了诸多进展,*IPA1*除调控株型外,在响应生物和非生物胁迫上也发挥重要作用,展现出强大的正向多效性,具有很高的应用价值。本文综述前人对理想株型基因*IPA1*的研究成果,探讨需要进一步研究的科学问题,试图为*IPA1*在水稻育种上的应用提供参考。

1 *IPA1*调控理想株型

株型通常是指植物地上部分各个组织形态特征及其在空间上的分布方式,水稻株型主要体现在株高、茎粗、叶型、分蘖数目、分蘖角度和穗部形态等方面^[18]。株型作为多个复杂农艺性状的集合体,是决定作物产量的关键要素。现有研究表明,*IPA1*介导的株型发育是一个复杂的分子网络,涉及转录与转录后以及翻译与翻译后修饰等调控路径(图1)。

*IPA1*是一个半显性基因,编码转录因子OsSPL14(souamasa promoter binding protein-like 14),是微小核糖核酸OsmiRNA156的直接靶标^[14]。*OsSPL14*的一个点突变扰乱了OsmiRNA156对*OsSPL14*的调控,从而产生了一种“理想”水稻植株,表现为分蘖数减少、抗

倒伏能力增强、籽粒数和千粒重增加,产量提高^[14]。在营养生长期,*OsSPL14*控制水稻分蘖;在生殖生长期,*OsSPL14*高表达促进了穗分枝;且将*OsSPL14*等位基因引入品种日本晴(Nipponbare)中,使得水稻产量得以提高^[15]。Lu等人^[19]证实*IPA1*是一种功能性转录激活因子,并通过染色质免疫沉淀测序分析,分别在茎尖和幼穗中鉴定出了1067个和2185个潜在的*IPA1*结合位点。同时,该研究发现,*IPA1*能够直接结合到水稻分蘖芽生长的负调控因子*OsTB1*(*teosinte branched 1*)^[20,21]基因的启动子上,促进*OsTB1*表达以抑制水稻分蘖,并能激活直立穗型基因*DEP1*的表达^[16,22,23],从而影响植株高度和穗长^[19]。有趣的是,*IPA1*的一个互作蛋白Os-SH11(short internodes 1),能够直接结合到*OsTB1*和*DEP1*的启动子区域的顺式作用元件(T/GCTCTAC),阻碍*IPA1*对*OsTB1*和*OsDEP1*启动子的结合,抑制了*IPA1*的转录激活活性,从而导致分蘖数增加以及穗变小^[24]。Luo等人^[25]鉴定到一个编码热激蛋白的基因*NAL11*(*narrow leaf11*)的等位变异*NAL11*^{-923del-1552},该等位基因表达增加,会产生相对较少的分蘖、粗茎和大穗的理想株型表型。进一步研究表明,*IPA1*能直接与*NAL11*^{-923del-1552}启动子缺失片段中的元件结合,并负调控*NAL11*的表达,而*NAL11*通过调节赤霉素(GA)的内稳态来调控理想株型^[25]。水稻花序结构是构成穗粒产量的基础,植物激素细胞分裂素(CK)已被证实能够调控水稻花序的发育^[26-28]。Chun等人^[29]鉴定到一个细胞分裂素受体*OHK4/OsHK4*的失活突变体*pal1*(*panicle length1*),该突变体由于花序分生组织活性降低,导致穗变小。研究发现,B型反应调节因子(type-B response regulator)OsRR21能直接激活*IPA1*的表达,而OsRR21的活性依赖于OHK4,*pal1*导致OsRR21不能将CK信号传导至*IPA1*,从而影响穗发育。此外,*IPA1*通过与2个TCP转录因子,PCF1和PCF2^[19],相互作用结合到*OHK4*启动子上并促进其表达,从而形成一个正反馈回路^[29]。

*IPA1*蛋白水平调控的研究极大地丰富了*IPA1*调控株型的分子机制。Wang等人^[30]鉴定到一个在细胞核内与*IPA1*互作的蛋白IPI1(*IPA1 interacting protein 1*),它是一种RING-finger E3连接酶,通过在稻穗中以K48连接的多聚泛素链促进*IPA1*的降解,而在茎尖部位以K63连接的多聚泛素链起到稳定*IPA1*的作用,最终以组织特异性的方式泛素化*IPA1*,进而精准调节水稻不

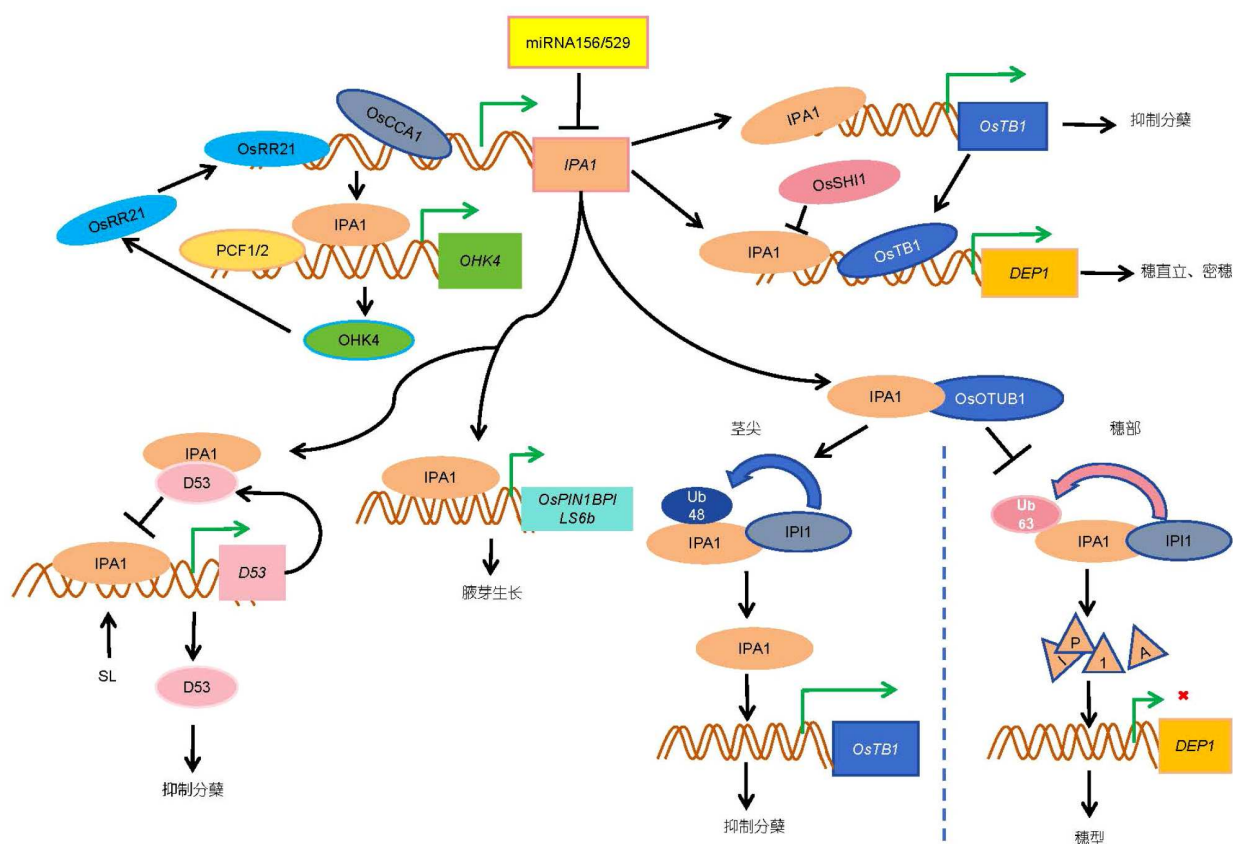


图 1 *IP41* 调控株型机制示意图

Figure 1 Schematic of the mechanism by which *IPAI* regulates plant architecture

同组织中IPA1蛋白的水平来影响植株形态。另一项研究发现, 一个调控水稻株型的主效数量性状位点 *qNPT1(new plant type 1)*, 该位点编码一个去泛素化酶 OsOTUB1(otubain-like protease with deubiquitination activity 1)。OsOTUB1能与IPA1相互作用, 抑制后者被 K63连接的泛素化, 这反过来又促进了IPA1依赖于K48连接的泛素化降解^[31]。泛素化酶IPI1与去泛素化酶 OsOTUB1协调作用, 实现了对IPA1蛋白水平在植株不同组织内的精准调控。IPI1功能缺失突变体植株表现出分蘖增加, 稻穗增大以及单株产量提高^[30]。这无疑拓宽了IPA1在分子设计育种中的应用途径, 例如, 改良特定品种时, 通过敲除IPI1增加分蘖数和穗长; 或利用IPI1和OsOTUB1作用的“跷跷板”关系, 来更加精密地将IPA1蛋白水平在相应组织器官中调节到最佳状态, 从而提高产量潜力。此外, Zhang等人^[32]在超级稻甬优12中鉴定到一个数量性状位点 *qWS8/ipa1-2D*, 该位点产生于IPA1上游的3'端天然串联重复序列, 它降

低了 *IPA1* 的 DNA 甲基化修饰, 保持 *IPA1* 启动子区域的染色质处于松散状态, 从而减轻了由附近异染色质介导的对 *IPA1* 的表观遗传抑制作用, 使植株维持适当的分蘖数目。该项研究表明, *IPA1* 对分蘖数、茎粗和一次枝梗数的调控呈剂量依赖性, 自然等位变异 *ipa1-2D* 通过精细调节 *IPA1* 的表达水平来平衡分蘖数目与穗部性状, 以塑造更好的理想株型, 从而实现更高的产量, 这展示了一条高效设计优良超级稻品种的途径。

分蘖直接决定了水稻有效穗数,进而影响产量。植物激素独脚金内酯(SLs)被证实通过抑制腋芽伸长调控植物分蘖形成与发育^[33,34]。早先的研究认为, *IPA1* 对水稻分蘖的调控,是独立于SL通路的另一条路径^[35]。然而,更新的证据表明, *IPA1* 是SL介导的分蘖调控信号通路中的下游关键转录因子^[36]。多蘖矮秆基因 *D53 (dwarf53)* 编码一个与 I 类 ClpATP 酶类似的蛋白,是SL信号途径中的负调控因子^[37~39]。Song 等人^[36]发现, *D53* 与 *IPA1* 互作,并抑制后者的转录激活活性,而 *IPA1* 能够

直接结合到*D53*的启动子上促进*D53*的表达, 形成一个负反馈循环. 进一步研究发现, 用*rac-GR24*(SL类似物)处理*IPA1*功能缺失和过表达的植物既不能抑制芽的生长, 也不能诱导*D53*转录本的表达, 说明在SL信号通路中, *IPA1*在*D53*的下游发挥作用, 且SL对*D53*转录的诱导依赖于*IPA1*的正常表达^[36]. Wang等人^[40]报道了一个涉及生物钟、糖类以及SL通路的调控回路, 该回路可调控水稻分蘖芽及稻穗的发育. 水稻生物钟调节因子*OsCCA1*(*circadian clock associated1*)能够结合到*OsTBI*、多蘖矮秆基因*D14*(*DWARF14*)^[41~43]和*IPA1*等SL通路关键基因的启动子上直接调节它们的表达, 从而抑制分蘖; 同时, *OsCCA1*对*IPA1*的直接调控, 导致稻穗和籽粒变大; 此外, 糖类会抑制水稻*OsCCA1*在根系和分蘖芽中的表达, 从而促进分蘖芽的生长^[40]. 生长素输出载体*OsPIN1b*^[44~46]和*PILS6b*^[47,48]参与生长素应答和水稻分蘖发育. *IPA1*能结合在*OsPIN1b*和*PILS6b*启动子区域的GTAC顺式作用元件上, 激活它们表达, 通过精细调节生长素运输和分布, 调控水稻腋芽生长^[49].

水稻遗传改良过程中, 需要平衡因基因多效性和连锁累赘所导致的复杂权衡关系. 产量三要素中, *IPA1*虽然能增加每穗粒数和提高千粒重, 但会减少分蘖数. Song等人^[50]采用基于平铺删除的CRISPR-Cas9筛选技术, 鉴定到一个*IPA1*启动子区域缺失54 bp的突变体*IPA1-Pro10*, 能同时增加分蘖数和穗粒数, 进而显著提高单株粮食产量. 机制研究表明, *IPA1-Pro10*缺失的这个片段是驯化关键转录因子An-1^[51]的一个靶位点, An-1通过结合这个位点来抑制*IPA1*在稻穗中的表达, 从而特异性调控穗部表型. 此外, 该研究还确定了*IPA1*启动子中与An-1结合的位点负责调控穗、茎和根的多个性状^[50], 但不包括分蘖数的调控, 暗示*IPA1-Pro10*缺失片段中可能存在其他的靶基因位点来调控分蘖. 最近, Rong等人^[52]发现, *IPA1*能够通过与*miR408-5p*前体启动子相互作用来调节水稻叶片倾角. 研究表明, *IPA1*对叶倾角的调控依赖于*miR156-IPA1-miR408-5p-IAA30*生长素信号转导通路, 此外, *miR393*可使生长素受体沉默, 进而影响了该调控模块^[52].

*IPA1*介导的理想株型调控方式主要依赖于不同等位引起的自身转录或蛋白水平的改变, 进而对下游靶标进行调控. 转录层面, 如*IPA1*外显子区域一个点突变扰乱了*miRNA156*对*IPA1*的抑制^[14]; *ipa1-2D*中*IPA1*

上游串联重复序列, 削弱了*IPA1*的DNA甲基化修饰, 解除了*IPA1*的抑制^[32]; 基因编辑*IPA1*启动子产生的*IPA1-Pro10*, 失去了An-1的结合抑制位点, 激活了*IPA1*的表达^[50]. 蛋白层面, 如*D53*与*IPA1*互作反馈抑制了*IPA1*的转录激活活性而削弱了对*D53*的转录^[36]; *IPI1*与*IPA1*互作并以不同方式泛素化修饰后者, 此过程又被去泛素化酶*OsOUTB1*所干预, 其抑制*IPA1*的K63泛素化修饰而促进其以K48泛素化修饰, 维持*IPA1*蛋白水平在不同组织部位的丰度^[30,31]. 此外, Abbai等人^[53]在3000份水稻资源中, 鉴定出*IPA1*存在15种以上等位类型, 表现出不同的分蘖水平, 其中以*IPA1-H14*为最优, 出现频率为6.64%. 因此, *IPA1*通过多层次的分子互作精确协调株型发育, 而调控网络中的等位基因组与互作模式决定了*IPA1*在不同水稻品种中的功能分化.

2 *IPA1*调控其他生长发育

2.1 *IPA1*调控根系及穗部苞叶生长

氮素是维持水稻生长的主要营养元素, 除铵态氮(NH_4^+)外, 硝酸盐(NO_3^-)也是水稻根系能吸收和利用的氮源^[54]. Sun等人^[55]发现, 水稻会通过改变根系形态来响应外环境中的硝酸盐, *IPA1/OsSPL14*和*OsSPL17*作用于SL信号的下游, 调节硝态氮营养下水稻根系的伸长. 研究表明, *D53*与*OsSPL14/17*互作, 并抑制后者介导的对*OsPIN1b*的转录激活. *OsSPL14/17*和*OsPIN1b*的突变导致根伸长反应对 NO_3^- 和*rac-GR24*施用不敏感. 进一步研究表明, *D14*对SL信号的感知会通过蛋白酶体系统导致*D53*降解, 解除了其对*OsSPL14/17*的抑制, 从而激活了*OsPIN1b*的转录, 进而在硝酸盐供应的情况下导致根系伸长^[55]. Song等人^[50]利用基因编辑技术, 获得三个缺失片段不同的突变体*IPA1-Pro10*, *IPA1-Pro11*和*IPA1-Pro12*, 正常生长情况下, 根系均较野生型更粗壮. 此外, Srikanth等人^[56]发现, 低氮条件下, *IPA1*与水稻产量构成要素紧密关联.

在水稻中, 苞片是指生殖转变后产生的叶, 着生在穗分枝的基部, 通常情况下, 苞片的生长受到抑制, 在稻穗上一般看不到明显的、像营养叶一样的叶片. Wang等人^[57]发现, 过表达*miR156/529*或下调其靶标*OsSPL14*和*OsSPL17*, 均表现出苞片过度生长表型. *osspl14*单突以及*osspl17 osspl14 osspl17*组合的双突和

三突, 旋生叶序处均出现明显的苞叶, 突变体穗轴显著短于野生型, 三突稻穗最终表现为富含营养枝的多叶表型, 育性降低, 表明OsSPLs通过终止/抑制营养生长来促进和/或维持生殖生长的建成, 对穗部苞叶的生成有抑制作用^[57]。

2.2 *IPA1*调控种子休眠

种子休眠是植物中常见的现象, 低水平的休眠可能产生收获前萌发(pre-harvest sprouting), 而高水平的休眠则抑制迅速和整齐的萌发, 二者都会影响作物的产量与质量^[58,59]。种子休眠作为一个受遗传和环境因素影响的复杂性状, 受植物激素的调控, 脱落酸(ABA)和GA被认为是调控种子萌发的主要因素, ABA诱导休眠抑制萌发, 而GA释放休眠和促进萌发, 二者相互拮抗^[60-62]。Miao等人^[63]发现, 突变*miR156*促进水稻种子休眠并抑制穗发芽, 且几乎不影响水稻形态和籽粒大小。机制研究表明, *miR156*突变解除了对其靶基因*IPA1*的抑制作用, 而*IPA1*能直接调控多个GA生物合成、信号传导和失活基因的表达, 如*KAO*, *CPS1*, *GNP1*, *SD1*, *SLR1*, *GA2ox6*和*EUI1*, 从而增强种子休眠性^[63](图2)。除*IPA1*外, 部分其他SPL基因也是

*miR156*的靶标, *miR156*还可能通过靶向*SPL2-SPL4*, *SPL7*, *SPL11-SPL13*和*SPL16-SPL18*来调控GA通路^[63]。在另一项研究中, Qin等人^[64]验证了这一推测。在水稻种子中特异性过表达*IPA1/OsSPL14*和*OsSPL12*, 能够增强水稻种子的休眠性并抑制穗萌, 并证实*OsSPL12*也直接调控GA代谢途径中的多个基因的表达来促进种子休眠^[64]。此外, 在种子中特异性过表达*IPA1*还能使籽粒增大, 进而提高产量^[64]。Shen等人^[65]筛选到一个Os**H**HLH004转录因子的突变体, 其种子休眠性增加。研究发现, Os**H**HLH004负调控ABA生物合成的关键酶基因*OsNCED3*^[66]和GA分解代谢关键酶基因*OsGA2ox6*^[67]的表达, 抑制了ABA的合成和GA的降解, 从而降低种子休眠性。深入研究发现, 两个磷脂酰乙醇胺结合蛋白OsMFT1和OsMFT2(mother of FT and TFL 1 and 2), 与Os**H**HLH004以及*IPA1*互作, 抑制了Os**H**HLH004与*OsNCED3*和*OsGA2ox6*的结合, 促进了*IPA1*对*OsNCED3*和*OsGA2ox6*的转录激活, 从而促进种子休眠^[65](图2)。He等人^[68]发现, *IPA1*通过GA和WRKY途径干扰淀粉代谢, 从而负调控种子萌发及幼苗早期生长。机制研究表明, *IPA1*能够直接激活*OsWRKY51*和*OsWRKY71*的表达, 影响了受GA诱导的转录因

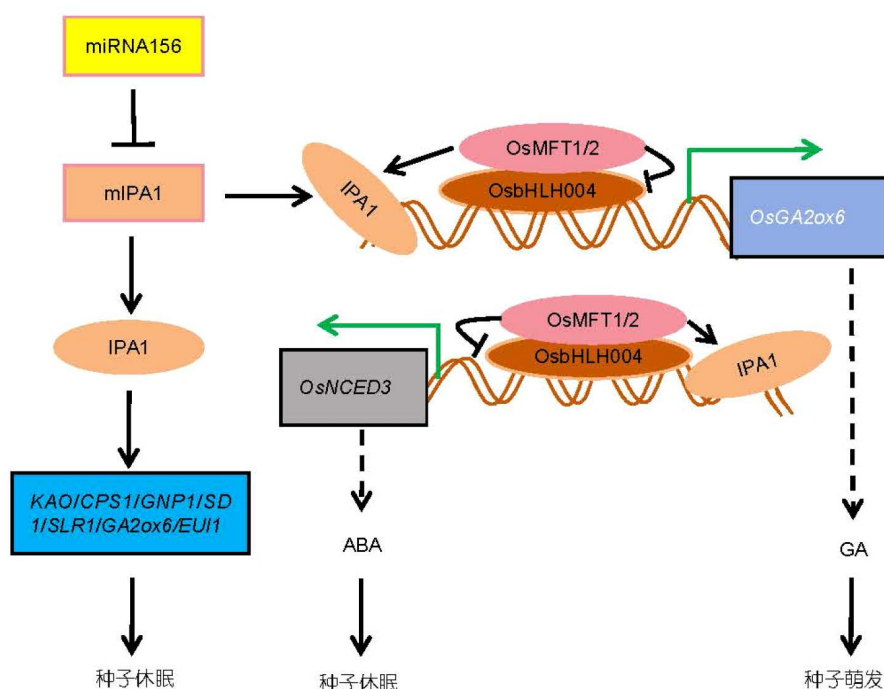


图2 *IPA1*调控种子休眠机制示意图

Figure 2 Schematic of the mechanism by which *IPA1* regulates seed dormancy

子OsGAMYB的结合亲和力,进而抑制 α -淀粉酶基因的表达^[69],导致种子萌发和幼苗生长受阻.

3 *IPA1*响应生物胁迫

植物在面对病原体侵入时,往往会消耗过多的能量和营养物质用于免疫防御,而影响其他正常生理过程的资源分配,导致作物减产等^[70,71]. Wang等人^[72]研究发现, *IPA1*在提高稻瘟病抗性的同时,还能增加产量,打破了单基因不能同时增加产量和抗性的传统观念,为培育高产抗病水稻新品种提供了重要理论基础和应用途径. *IPA1*在正常条件下促进生长发育,而在稻瘟病菌侵染时,其DNA结合结构域内的第163位丝氨酸发生磷酸化,并且这种磷酸化改变了*IPA1*的DNA结合特异性. 磷酸化的*IPA1*会与病原体防御基因*WRKY45*的启动子结合并激活其表达,进而增强抗病性. 有趣的是,在受到侵染后的48小时内, *IPA1*会恢复到非磷酸化状态,非磷酸化的*IPA1*能够结合*DEP1*启动子并激活其表达,重新开始为高产所需的生长提供支持(图3). 因此, *IPA1*通过维持生长和免疫之间的平衡,既能促进产量提升,又能增强抗病能力^[72]. 最新研究表明,磷酸化的*IPA1*对*WRKY45*的调控依赖于一个

含环指结构的E3连接酶IPI7^[73]. IPI7通过与*IPA1*互作,促进*IPA1*发生K29多聚泛素化修饰,但不影响*IPA1*蛋白质稳定性. IPI7以非水解的K29泛素化*IPA1*受稻瘟病菌(*Magnaporthe oryzae*)侵染所诱导,并且对于磷酸化的*IPA1*激活*WRKY45*表达以产生免疫应答是必需的,但对于正常条件下*IPA1*促进穗发育的作用来说并非必需(图3). 因此, *IPI7*基因敲除会损害*IPA1*介导的免疫能力,但不会影响产量^[73].

在另一项研究中, *miR156*的表达下调,以及*miR156*的两个靶基因*IPA1*和*OsSPL7*的过量表达,增强了水稻对白叶枯病的抗病能力,但会降低水稻产量^[74]. 研究发现, *IPA1*和*OsSPL7*通过与DELLA蛋白SLR1 (slender rice 1)互作,降低由GA介导的疾病易感性. *IPA1*对免疫和生长的调控作用部分依赖于SLR1蛋白. 此外,小RNA调控基因*OsHEN1*(*hua enhancer 1*)启动子含有Ta19a的结合位点,受水稻白叶枯病菌诱导^[75]. 通过*OsHEN1*基因启动子诱导性过表达*IPA1*,可使水稻既获得高产量又具有高抗病能力^[74].

4 *IPA1*响应非生物胁迫

水稻种植区域分布广泛,生态条件多样化,生长过

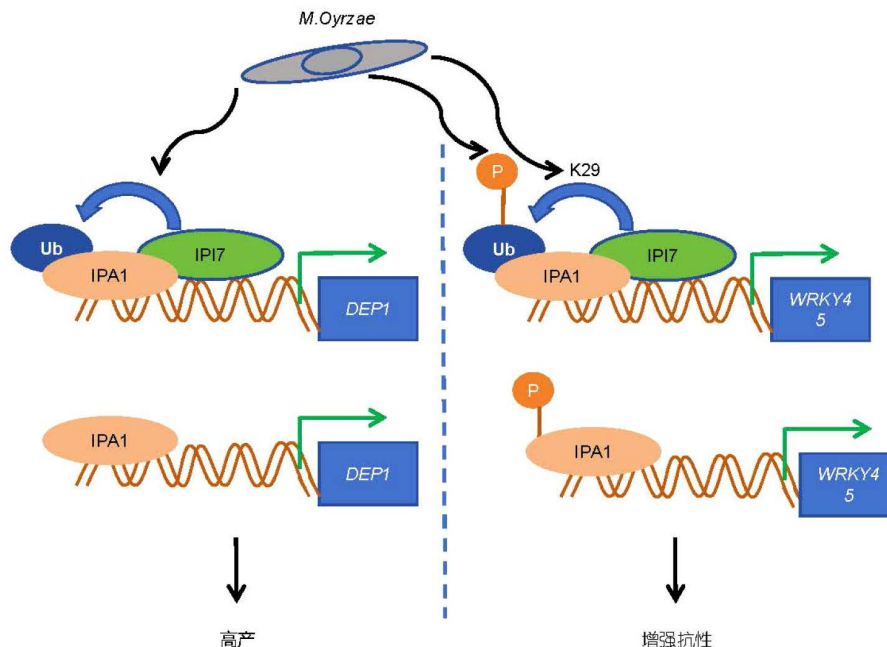


图3 *IPA1*平衡稻瘟病抗性与高产机制示意图

Figure 3 Schematic of the mechanism by which *IPA1* balances resistance to rice blast and high yield

程中可能会因遭受多种非生物胁迫, 比如高温、低温、干旱、洪涝、盐碱和重金属等, 从而带来产量损失. 深入解析水稻响应非生物胁迫的生理和遗传机制, 挖掘抗逆基因并应用于育种仍然是提高水稻非生物胁迫耐受性的主要途径之一. 研究发现, *IPA1*调控了水稻低温、干旱和盐胁迫. Zhu等人^[76]利用一对*IPA1/ipa1*近等基因系进行研究, 发现*IPA1*正调控水稻幼苗期的耐旱性. 研究表明, *IPA1*能直接激活同源异型盒蛋白12基因(*homeoBox12*)*OsHOX12*和转录因子基因*OsNAC52*的表达^[76]. 据报道, *OsHOX12*能促进ABA生物合成关键酶9-顺式-环氧类胡萝卜素双加氧酶(9-*cis*-epoxycarotenoid dioxygenase 1)*OsNCED1*的表达, 从而促进水稻茎基部ABA的生物合成^[77], 而*OsNAC52*则是ABA信号途径的正调控因子^[78]. 因此, *IPA1*通过调控*OsHOX12*和*OsNAC52*的表达以激活ABA途径来提高水稻幼苗的耐旱性(图4). Chen等人^[79]发现, 敲除*IPA1*会降低水稻幼苗的抗旱性, 而过表达*IPA1*则会提高水稻幼苗的抗旱性. 研究表明, *IPA1*直接激活活性氧(ROS)调控途径的关键转录因子基因*SNAC1*^[80,81]的表达, 通过影响ROS含量调节水稻抗旱性^[79].

低温是影响水稻生长、育性和灌浆的主要因素之一. 一批赋能水稻耐冷性的重要基因, 比如*COLD1*^[82],

CTB4a^[83], *HANI*^[84]等被发掘, 但水稻响应低温信号及其转导机制仍有待于深入解析. Jia等人^[85]报道*IPA1*是水稻耐冷性的正调控因子. 低温胁迫下, 渗透胁迫/脱落酸激活蛋白激酶*OsSAPK6*^[86,87]与*IPA1*互作, 并在Ser201和Ser213位点对*IPA1*进行磷酸化修饰, 积累并稳定*IPA1*蛋白, 从而促进*OsCBF3*(*c-repeat binding factor*)基因的表达, 增强水稻耐冷性^[85]. 研究表明, *IPA1*在Ser213位点的磷酸化对于其耐冷表型有关键作用, 将Ser213位点的丝氨酸突变成无法被磷酸化的天冬酰胺后, *ipa1*^{S213N}突变体表现出了冷敏感表型. *OsSAPK6*, *IPA1*和*OsCBF3*均为水稻抗寒性的正调控因子, *OsSAPK6*调控低温依赖于*IPA1*, 而过表达*OsCBF3*能恢复*ipa1*功能缺失突变体的冷敏感表型. 此外, 自然等位变异*ipa1-2D*能够增强水稻幼苗的耐冷性并增加籽粒产量^[85]. 该研究揭示了水稻的*OsSAPK6*-*IPA1*-*OsCBF3*低温信号级联, 扩展了对低温信号传导机制的认识, 为选育高产耐寒水稻品种提供了新思路 and 重要的遗传资源.

我国国土面积的1.3%是盐碱地, 是受土壤盐碱化影响最严重的国家之一, 盐碱胁迫是水稻生长面临的最严重胁迫之一^[88]. Jia等人^[89]在另一项研究中, 对*IPA1*功能缺失突变体*ipa1-10*和功能获得型突变体

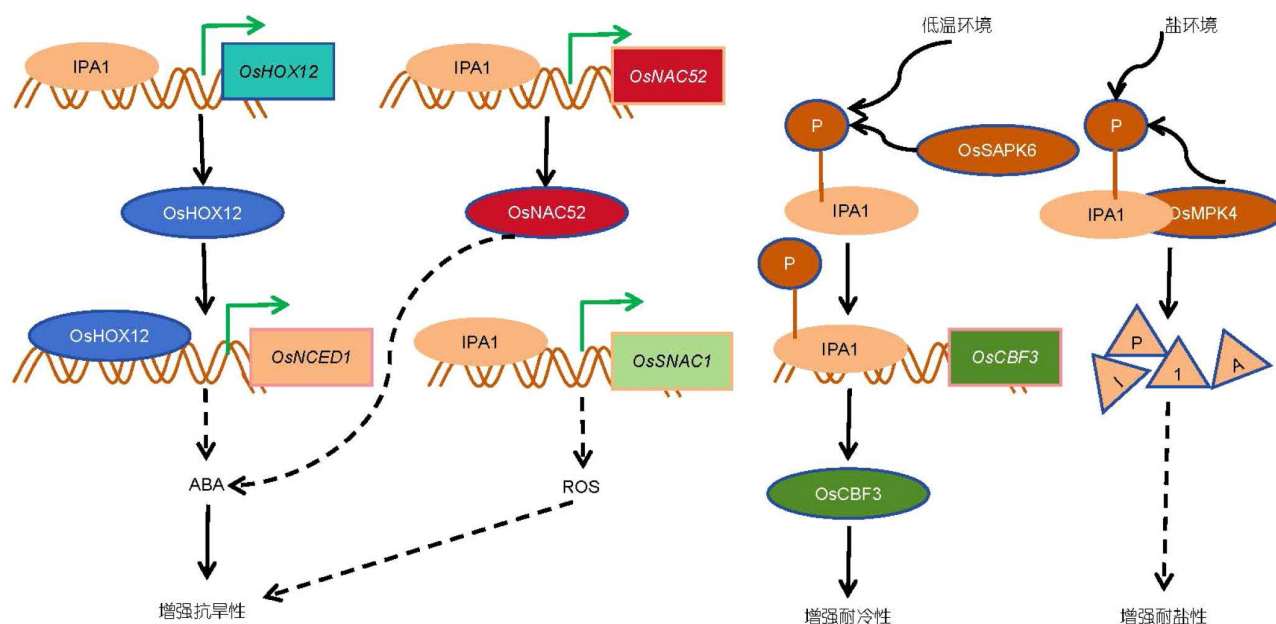


图4 *IPA1*响应非生物胁迫机制示意图

Figure 4 Schematic of the mechanism by which *IPA1* responds to abiotic stress

*ipa1-3D*的幼苗进行盐处理,发现*IPA1*负调控水稻的耐盐性.研究表明,丝裂原活化蛋白激酶OsMPK4与*IPA1*互作,盐胁迫下,OsMPK4被激活并磷酸化*IPA1*的Thr180位点,导致*IPA1*泛素化降解,从而降低*IPA1*的蛋白水平并最终提高水稻耐盐性^[89].值得注意的是,*IPA1*在Thr180处突变仅部分消除了OsMPK4介导的*IPA1*磷酸化,表明*IPA1*中存在其他位点可能也受OsMPK4磷酸化调控.同时,*osmpk4*突变体中盐胁迫诱导的*IPA1*磷酸化被部分抑制,暗示其他激酶可能也以*IPA1*为底物进行磷酸化修饰^[89].然而,*IPA1*在水稻耐盐中的下游机制及应用潜力有待进一步研究.

一般认为,植物已经进化出一种调节机制来平衡生长和对胁迫的抗性.一个可能的机制是植物在胁迫下可以诱导关键多功能蛋白的瞬时修饰.例如,*IPA1*在生物和非生物胁迫下,通过对特定定位点的磷酸化,实现对生长、免疫和抗逆性等多方面功能的动态调控.磷酸化修饰介导的对*IPA1*的精确调控赋予了*IPA1*蛋白功能的多样性,从而使水稻能够在不同环境压力下,灵活地平衡生长和防御,实现高产和抗逆性的结合.

5 *IPA1*在育种中的应用

*IPA1*在调控水稻理想株型、抗病性、抗逆性等方面展现出多效性功能,随着*IPA1*的表达调控机制、作用机理、信号途径和调控网络的不断阐明,越来越多科研人员将*IPA1*应用于育种实践中.自*IPA1*问世之初,李家洋团队^[14]便率先将*ipa1-1D*等位基因导入到粳稻常规品种秀水11中,发现可使穗粒数增加约60%,增产10%以上.Zhang等人^[32]在超级稻甬优12中鉴定到*IPA1*天然功能获得型等位变异*ipa1-2D*,在粗秆大穗的基础上,还能维持适当的分蘖,将其导入粳稻品种日本晴中可增产约37%~41%.Kim等人^[90]分别将穗粒数主效基因*Gn1a*^[91]和*IPA1*优势等位导入4个籼稻品种中,对比每穗粒数,发现*Gn1a*优势等位在籼稻品种中与对照没有显著差异,而*IPA1*优势等位在四个品种中的每穗粒数都增加了10.6%~59.3%,大田种植增产比例则高达28.4%~83.5%,比对照栽培品种IRRI156高出约64.7%.而Song等人^[50]针对*IPA1*启动子不同区域及3'UTR区域设计39个靶点进行连续敲除,发现在ATG上游72~125位置范围内的一段序列缺失,能增加穗二次枝梗数,不影响分蘖,最终使产量显著增加,平衡了穗粒数和

分蘖数之间的矛盾.Li等人^[92]在中花11中敲除*IPA1*,获得了*ipa1*突变体系列,表现出分蘖增多和分蘖减少两种表型,这取决于*OsmiR156*靶区诱导产生的变化,前者为基因功能完全丧失类型,而后者突变导致*miR156*识别位点改变,从而产生了分蘖较少、秆粗穗大的理想株型表型.在另一项研究中,Li等人^[93]利用CRISPR/Cas9系统在中花11中对*Gn1a*,*IPA1*和粒型基因*GS3*^[94]进行靶向编辑,产生的*gn1a*,*gs3*和*ipa1*突变体分别呈现出大穗、大粒、无效分蘖少以及茎秆粗壮的特征.随后通过常规杂交育种,培育出了*gn1a-gs3*,*gn1a-ipa1*,*gs3-ipa1*以及*gn1a-gs3-ipa1*四条聚合系,其表现优于单基因突变体,*gs3-ipa1*和*gn1a-gs3-ipa1*聚合系的粒重,比单基因突变体的粒重要重,*gn1a-gs3*和*gs3-ipa1*聚合系的单株籽粒产量均高于单基因突变体系.含有*ipa1*的聚合系,都具有与*ipa1*单突变体类似的“理想株型”表型,但在*gs3-ipa1*中却并未出现有效分蘖减少^[93],这一组合体现出良好的应用潜力.

上述研究证明了*IPA1*功能位点可有效用于水稻增产,具有极高的推广利用价值.李家洋团队^[95]将*IPA1*导入优质籼稻和粳稻亲本中,育成了适宜长江中下游稻区种植的“嘉优中科”系列水稻新品种.该品种聚合了控制理想株型、抗稻瘟病、结实率、生育期等优异性状的等位基因,具有株高适宜、分蘖适中、无效分蘖很少、茎秆粗壮、根系发达等明显的理想株型特征,且熟期早,抗逆性强,增产效果显著.“嘉优中科”系列杂交水稻品种有效地解决了水稻生产中兼具高产、优质和抗病的品种较少的难题,标志着利用*IPA1*进行分子设计育种从概念变为了现实,并将应用到更多的水稻育种实践中.

6 展望

综上所述,关于理想株型基因*IPA1*的功能与机制研究取得了重大进展,尤其是在解析*IPA1*调控株型的分子机理上,通过上下游基因鉴定、蛋白水平的调控及激素信号途径分析,构建出一个复杂且精细的株型调控网络.近些年,科学家们也鉴定出*IPA1*在水稻抗病、抗逆及调控种子休眠等的功能,拓展了*IPA1*的功能作用和潜在价值.此外,*IPA1*在育种实践中也取得了重大突破.然而,关于*IPA1*进一步的研究,仍然有诸多探讨之处.

6.1 *IPA1*调控下游靶基因及其生物学机制

Lu等人^[19]通过全基因组染色质免疫沉淀测序分析, 确定了数千个*IPA1*的潜在靶基因, 迄今为止仍只有少数基因被鉴定. 因此, *IPA1*如何调控其他下游靶点及其生物学效应仍有待充分研究, 这对于充分利用*IPA1*进行分子设计育种具有重要意义. 已知*IPA1*作为SL途径下游调控水稻分蘖的关键因子, 在此过程中, *IPA1*受D53抑制^[36], 而*IPA1*直接结合*OsTB1*负调控水稻分蘖^[19]. 然而, 在水稻中尚未观察到SL对*OsTB1*的转录激活, 事实上, 在水稻SL突变体中, *OsTB1*的表达并不总是与分蘖负相关^[96]. SL是否能够调控水稻芽特异性组织中*OsTB1*基因的表达, 以及这种调控是否依赖D53和*IPA1*, 还有待研究^[97]. 此外, *OsmiR156*对水稻分蘖的负调控效应并非完全依赖*IPA1*, 因为*OsmiR156*过表达系对SL响应, 而*IPA1*过表达系则没有^[35]. *OsmiR156*对分蘖的影响可能也通过其他SPLs转录因子, 如*OsSPL7*和*OsSPL17*, 其突变体也表现出分蘖变化表型^[98]. *NAL11*在*IPA1*的下游通过GA途径调控分蘖和穗分枝, 然而*NAL11*缺失突变后, SL相关基因表达上调, 暗示*NAL11*可能协调SL和GA之间的调控, 值得进一步研究^[25].

6.2 *IPA1*与*SD1*互作机制

植物免疫反应往往会牺牲产量^[99]. 然而, *IPA1*在病原菌刺激下, 能通过翻译后的泛素化和磷酸化修饰, 巧妙地同时促进免疫和产量^[72,73]. Wang等人^[72]认为, 通过蛋白质磷酸化改变DNA结合特异性来灵活地应对环境刺激可能是一种普遍现象. Jia等人^[85]的研究证实了这一点, 在低温胁迫下, *OsSAPK6*可以磷酸化*IPA1*, 提高其稳定性, 从而提高水稻耐寒性. 此外, 在Liu等人^[74]的研究中, *IPA1*过表达增加对水稻白叶枯抗性的部分原因是DELLA蛋白的积累, 而“绿色革命基因”*SD1*正是通过适度增加DELLA蛋白水平而大大提高了水稻的产量^[100,101]. 弄清楚*IPA1*与*SD1*的联系, 即确定*IPA1*和DELLA蛋白共同靶标, 将为作物育种提供新的策略.

6.3 *IPA1*同源基因的保守性

*IPA1*调控水稻理想株型的重大突破, 为解析其同源基因在其他作物中的功能提供了关键线索. *IPA1*在

小麦中的同源基因*TaSPL14*, 在调控小麦株型结构上的功能与*IPA1*相似. 过表达*TaSPL14*导致小麦分蘖数减少、穗粒数和粒重增加, 而敲除该基因则产生相反表型, 表明*TaSPL14*负调控分蘖、正调控穗粒数和粒重^[102~104]. 进一步研究表明, *TaSPL14*能够直接结合*TaTB1*和*TaPIN1*基因的启动子并激活其表达, 从而抑制分蘖^[104], 这与*IPA1*通过促进*OsTB1*^[19]和*OsPIN1b*^[49]的表达抑制水稻分蘖的机制一致. 而Liu等人^[105]的研究表明, SPLs介导的*TaTB1*的转录激活可以被TaD53在瞬时表达系统中抑制, 与*OsD53-IPA1-OsTB1*^[36]模块调控水稻分蘖的机制相似. 此外, *IPA1*在大豆中的同源基因*GmSPL9d*, 抑制大豆分枝的功能与*IPA1*抑制水稻分蘖类似^[106]. *IPA1*同源基因的功能保守性, 可能会为作物理想株型设计提供“通用蓝图”, 且*IPA1*促进水稻抗病、抗逆等功能是否能“迁移”至其他作物, 是未来值得关注的研究方向.

6.4 *IPA1*育种利用策略

*IPA1*通过增加穗粒数和粒重提升产量, 而对分蘖的负调控则限制了它的增产潜力. 自然功能获得型等位基因*ipal-2D*平衡了分蘖和穗部性状, 显著提高水稻产量^[32], 并促进了水稻抗寒性^[85], 为水稻育种提供了宝贵的遗传资源. *ipal-2D*已经成功培育了甬优、浙优等超高产杂交稻, 实现了亩产超过1000 kg的重大突破, 被认为是有可能实现新的绿色革命的重要位点, 然而, *ipal-2D*在2464份世界各地水稻品种的频率不到1%^[107], 应加大对这些稀有种质资源的育种利用, 以培育更多的高产高抗品种. 通过基因编辑方法可以产生具有更好*IPA1*表达的新型等位基因. 比如, 通过平铺删除筛选得到的突变体*IPA1-pro10*, 可以同时增加分蘖数和穗长, 从而大大提高产量^[50]. 此外, *IPA1*在促进营养元素吸收^[55,56]、抑制穗发芽^[63,64]、提高稻瘟病^[72,73]和白叶枯病^[74]抗性以及提升水稻苗期的耐旱^[76,79]、耐寒^[85]和耐盐^[89]性上发挥作用, 这对于培育不同生态环境、种植条件下的水稻品种具有重要意义. 随着时代的发展, 对水稻品种的需求已经不单单满足于高产, 而是要培育绿色安全、优质高产高效的全能型品种. 通过分子设计育种, 在理想株型基因*IPA1*的基础上聚合优质、抗病、抗逆、高效等基因, 无疑是实现这一目标的一条途径.

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Research progress on the ideal plant architecture gene *IPA1* in rice

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With the in-depth analysis of rice functional genomics, an increasing number of genes related to rice (*Oryza sativa*) yield have been cloned. The ideal plant architecture gene *IPA1* (*OsSPL14*) encodes a SQUAMOSA (SQUA) plant-specific transcription factor, which coordinately regulates the yield and disease resistance of rice, breaking the traditional viewpoint that a single gene cannot simultaneously achieve yield increase and disease resistance. This article mainly reviews the functions of the *IPA1* gene in regulating the ideal plant architecture of rice and other aspects of growth and development, as well as rice responses to biotic and abiotic stresses. It also reviews the current status of the applications of *IPA1* in plant breeding, proposes the issues that require further research in the *IPA1* regulatory network, and discusses the future application potential and breeding strategies. *IPA1* forms a complex molecular regulatory network by integrating multiple signaling pathways, which coordinates yield increase and resistance, and thus provides a theoretical basis and gene resources for high-yield and resistance breeding in rice.

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