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植物响应缺钾胁迫的生理与分子机制研究进展

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摘要: 钾(K)作为植物生长发育所必需的大量营养元素,是多种酶的关键组分,广泛参与植物的生理代谢与生化反应过程。然而,在农业生产中,土壤有效钾含量常难以满足作物需求,加上土壤环境退化及人为活动的影响,植物缺钾现象普遍存在。在长期进化过程中,植物逐步形成了多种应对低钾胁迫的调控机制。本文系统综述了植物响应缺钾胁迫的生理与分子机制,重点探讨了缺钾对植物光合作用、水分吸收、酶活性及激素平衡等生理过程的影响,并阐述了 K^+转运系统、信号转导途径、转录调控网络及相关基因表达在植物缺钾胁迫响应中的作用。最后,对未来植物缺钾胁迫研究的方向进行了展望,以期为作物耐低钾及钾高效利用的分子育种研究提供理论依据。

关键词: 缺钾胁迫; 钾营养; 生理响应; 分子机制; 转录调控
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Research progress on physiological and molecular mechanisms of plant responses to potassium deficiency

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Abstract: Potassium (K), as an essential macronutrient for plant growth and development, is a key component of various enzymes and is widely involved in physiological metabolism and biochemical reactions in plants. However, in agricultural production, the content of available potassium in soil is often insufficient to meet the needs of crops. Additionally, due to the degradation of soil environment and the impact of human activities, potassium deficiency in plants is a common phenomenon. During long-term evolution, plants have gradually developed a variety of regulatory mechanisms to cope with low-potassium stress. This article systematically reviewed the physiological and molecular mechanisms of plant responses to low-potassium stress, focused on the effects of potassium deficiency on plant physiological processes such as photosynthesis, water absorption, enzyme activity, and hormone balance. It also elaborated on the roles of K^+ transport systems, signal transduction pathways, transcriptional regulatory networks, and related gene expression in plant responses to low-potassium stress. Finally, the future research directions of plant low-potassium stress were prospected, aiming to provide a theoretical basis for molecular breeding of crops with low-potassium tolerance and efficient potassium utilization.

Key words: potassium deficiency stress; potassium nutrition; physiological response; molecular mechanism; transcriptional regulation

钾是植物生长发育过程中不可或缺的必需营养

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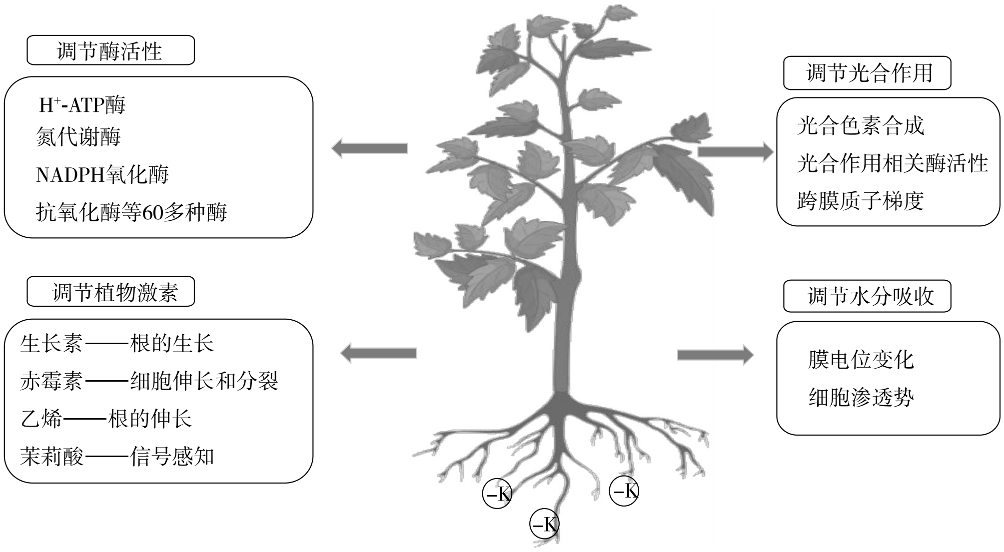
元素，在光合作用、酶激活及渗透调节等关键生理过程中起着重要作用[1]。然而，全球约 70%的耕地不同程度的缺钾，土壤有效钾匮乏已成为制约作物生产的主要因素[2]。尽管植物已进化出液泡钾储存与再动员、钾转运蛋白动态调控等适应性机制[3]，但长期缺钾仍会导致叶片萎蔫、生物量锐减[4-5]，造成产量损失。目前，相关研究主要集中于钾信号感知元件的分析鉴定、钾转运体功能调控机制、钾信号与胁迫应答网络的交叉调控。然而，关于系统整合植物生理响应与分子调控网络方面的研究较少，钾离子信号动态传递与根系构型重塑、气孔运动调控之间的分子调控网络尚未完全解析，钾信号与碳氮代谢途径、激素信号转导通路的互作机制也缺乏系统性研究。本文综述了缺钾胁迫下植物的适应性调控机制，从钾转运通道、缺钾信号转导通路、转录调控网络等层面揭示植物应对缺钾胁迫的层级响应策略，以期为作物钾高效利用遗传改良提供理论依据，并为钾肥增效技术开发和耐缺钾种质创制提供参考。

1　植物响应缺钾胁迫的生理机制

如图 1 所示，缺钾胁迫通过多种途径影响植物生长发育。在光合作用方面，钾离子通过调节保卫细胞渗透势调控气孔开闭，进而影响植物对 CO_2 的吸收效率，同时影响植物叶绿素合成与降解[6-7]。在水分运输方面，钾离子缺乏导致细胞膨压下降，并且钾离子通过抑制水通道蛋白活性降低水分运输效率[8]。在酶促反应中，钾离子作为多种酶的激活剂，其缺失会直接抑制光合碳同化、呼吸作用及抗氧化系统[9]。缺钾胁迫可重新调整植物体内的生长素、赤霉素、乙烯和茉莉酸的动态平衡，使植物适应缺钾胁迫[10-12]。



−K 表示缺钾；H^+-ATP：H^+-三磷酸腺苷；NADPH：还原型烟酰胺腺嘌呤二核苷酸磷酸。

图 1　植物响应缺钾胁迫的生理机制

Fig.1　Physiological mechanisms of plant responses to potassium deficiency stress

1.1　缺钾胁迫对植物光合作用的影响

钾离子通过调控气孔开闭、光合色素合成及酶活性影响植物光合作用。在气孔调节方面，钾离子通过调节保卫细胞的渗透压驱动气孔开闭[8]。缺钾条件下，保卫细胞中钾离子浓度降低，导致渗透势下降、膨压不足，引发气孔关闭，进而通过以下 2 种途径抑制光合效率：气孔导度降低直接限制植物对 CO_2 的吸收；蒸腾作用减弱引起叶片温度过高，间接抑制核酮糖-1，5-二磷酸羧化酶（*Rubisco*）活性和光系统Ⅱ（PSⅡ）电子传递速率[6-7]。在叶片色素合成方面，缺钾胁迫导致叶片栅栏组织与海绵组织细胞面积比增加，叶绿体发育异常，叶绿素合成受阻，叶片光捕获能力降低，同时叶绿体表面积减少进一步降低了光反应效率[13]。例如，缺钾胁迫下，玉米（*Zea mays* L.）中叶绿素含量显著降低，PSⅡ反应中心严重受损[14]。酶活性调控方面，缺钾胁迫显著抑制 *Rubisco* 羧化活性，导致卡尔文循环中 CO_2 的固定效率降低，PSⅡ电子传递链受阻，最终导致植物净

光合速率下降[15]。

1.2　缺钾胁迫对植物水分吸收的影响

K^+通过调节膜电位与渗透势维持植物水分平衡。正常条件下，K^+主要储存于液泡中，部分经木质部转运至地上部[16]。缺钾时，叶片相对含水量与水势降低，植株抗旱性减弱。缺钾胁迫下，水稻（*Oryza sativa* L.）气孔面积缩小、气孔导度降低，这直接抑制了气体交换，最终导致水稻减产和水分利用效率降低[17]。植物对钾离子的吸收量与水分消耗量呈正相关[18]。被钾离子转运蛋白吸收的钾离子，会随水分循环输送至各组织和器官。缺钾胁迫下，向日葵（*Helianthus annuus* L.）水分利用效率显著降低[19]。

1.3　缺钾胁迫对植物酶促反应的影响

K^+是60多种酶的必需激活因子[9]。在光合作用调控中，K^+通过直接激活H^+-三磷酸腺苷（H^+-ATP）酶提升植物光合速率[20]。对番茄（*Solanum lycopersicum* L.）施用外源K^+处理可显著提高其H^+-ATP酶活性[21]。在蛋白质合成方面，K^+参与核糖体组装、调控核糖体功能位点的活化和维持mRNA结构稳定性[22]。研究结果表明，K^+缺乏会导致棉花（*Gossypium hirsutum* L.）硝酸盐还原酶活性降低、蛋白质含量减少，同时伴随游离氨基酸的积累[23]。在碳氮代谢方面，缺钾会显著影响植物相关代谢酶活性、硝酸盐同化基因表达和硝酸盐转运能力。缺钾胁迫下，苹果（*Malus domestica*）幼苗根系对^{15}N的吸收能力和叶片对^{13}C的同化能力下降，导致根系向叶片中转运的^{15}N和叶片向根系中转运的^{13}C减少[24]。K^+还通过抑制还原型辅酶Ⅱ（*NADPH*）活性和激活抗氧化酶系统［包括脱氢抗坏血酸还原酶（*DHAR*）、谷胱甘肽还原酶（*GPX*）、超氧化物歧化酶（*SOD*）、过氧化氢酶（*CAT*）、抗坏血酸过氧化物酶（*APX*）、过氧化物酶（*POX*）］维持细胞氧化还原平衡[25]。缺钾会使参与光合作用的酶活性下降，从而导致植物的光合效率降低[26]。在能量代谢方面，K^+作为磷酸果糖激酶和丙酮酸激酶的活化剂，通过促进糖酵解和三羧酸循环过程参与ATP的合成[27]。此外，K^+能够激活淀粉合成酶，促进单糖合成蔗糖和淀粉；缺钾时，植物体内的糖和淀粉会水解成单糖，导致代谢紊乱[27]。缺钾还会破坏植物体内离子平衡，引发细胞内代谢紊乱并影响多种酶活性[28]。

1.4　缺钾胁迫对植物激素水平的影响

缺钾胁迫通过调控植物激素网络介导多层次的生理适应机制。研究结果表明，在缺钾胁迫下，生长素、赤霉素、乙烯和茉莉酸等植物激素通过复杂的互作网络调控植物的生长发育和胁迫响应[10-12,29-30]。

生长素作为调控细胞分裂、伸长和分化以及植物向性生长的关键激素，其合成和极性运输在缺钾条件下显著受阻[31]。研究发现，钾缺乏会干扰PIN家族蛋白介导的生长素极性运输，导致生长素空间分布失衡[32]。PIN蛋白作为定位于细胞膜的生长素转运载体，在低钾胁迫下，其表达和转运功能受到抑制[33]。钾转运体KUP9基因突变拟南芥（*Arabidopsis thaliana*）的根系发育受阻，表明拟南芥根系生长发育依赖于生长素和K^+的协同调控[34]。而低钾胁迫下，根尖生长素浓度降低，表明植物通过抑制根系生长以应对营养胁迫[35]。

赤霉素在促进茎伸长、种子萌发、开花和果实成熟等生理过程中发挥重要作用[36]。缺钾胁迫下，植物中赤霉素生物合成基因的表达受到抑制，导致赤霉素含量下降。赤霉素合成途径中的关键酶类包括萜类合酶、细胞色素P450单加氧酶和2-氧代戊二酸依赖性双加氧酶等，钾离子作为必需辅因子，在这些酶活性的维持中发挥重要作用[37]。缺钾胁迫通过抑制这些酶的活性，阻碍赤霉素的生物合成[38]。此外，赤霉素调控的细胞壁松弛蛋白表达也受到缺钾胁迫的影响[39]。值得注意的是，赤霉素与K^+之间存在协同调控关系，赤霉素可调节钾通道蛋白的表达，从而影响K^+的吸收和运输；而缺钾胁迫下，赤霉素合成酶活性降低，赤霉素含量减少，又会进一步减弱植物对钾的吸收能力[1,40]。

乙烯作为重要的植物激素，参与调控果实成熟、叶片衰老、不定根形成和种子休眠等生理过程[41]。研究结果表明，缺钾胁迫下，拟南芥（*Arabidopsis thaliana*）C_2H_2型锌指蛋白基因*ZFP5*的表达量升高，该基因通过调控乙烯信号通路进而影响根毛发育[42]。乙烯通过调控活性氧（ROS）水平，影响钾转运蛋白活性及根毛伸长，从而调控根系钾离子吸收与转运[43]。乙烯可通过促进拟南芥侧根和根毛发育增强植物对钾的吸收能力[44]。缺钾条件下，不同植物对乙烯响应存在物种特异性。在西瓜（*Citrullus lanatus* Thunb.）中，缺钾胁迫早期，乙烯信号转导途径相关基因表达量降低，而到后期，乙烯信号转导途径相关基

因表达量升高[45]。在梨(*Pyrus* spp.)中,低钾处理使乙烯生物合成及信号转导相关基因表达量显著升高,根系内源乙烯浓度提高[46]。在拟南芥中,乙烯通过诱导高亲和性钾转运蛋白 *HAK5* 基因的表达响应低钾胁迫[47]。在葡萄(*Vitis vinifera* L.)中,缺钾胁迫早期,乙烯响应因子(如 ERF 转录因子)基因表达量升高,而到后期,乙烯响应因子基因表达量降低,表明乙烯信号参与缺钾胁迫的调控[48]。

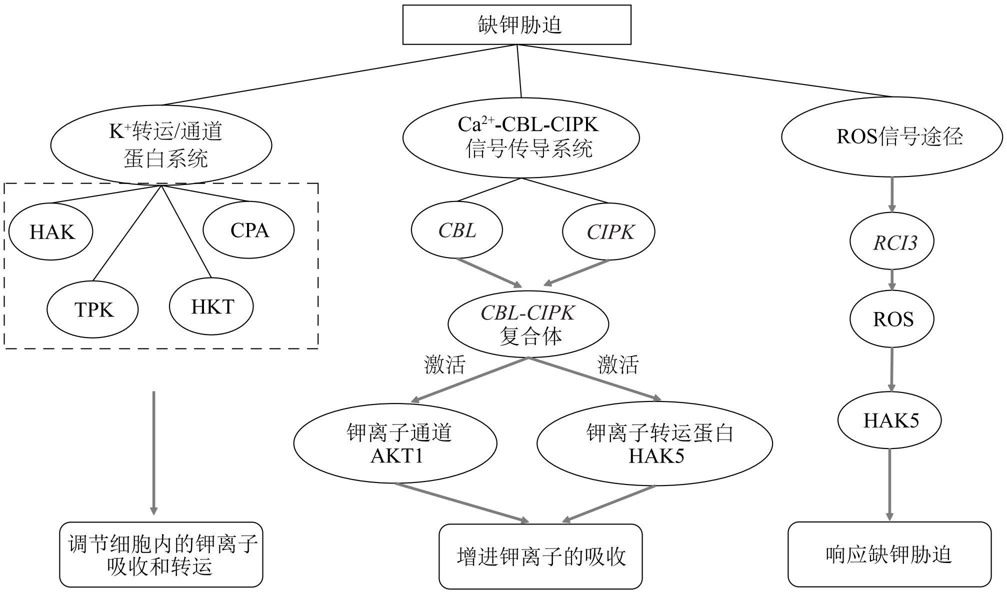
茉莉酸(JA)可以直接或间接激活钾离子转运蛋白。首先,缺钾胁迫会刺激茉莉酸衍生物(JA-Ile)的产生和积累,并由 F-box 蛋白 COI1 和 JAZ 蛋白形成的复合体受体识别。在 JA 信号传导过程中,COI1 与 JAZ 相互作用导致 JAZ 蛋白泛素化,进而通过泛素-蛋白酶体系统降解,JAZ 的解除使得其对下游响应因子的抑制作用被释放,从而启动 JA 应答[49]。在代谢调控网络中,雷帕霉素靶蛋白(TOR)可通过抑制 JA 信号通路响应外界营养胁迫,TOR 与 JA 信号通路之间的作用机制仍有待研究[50]。缺钾条件下,水稻(*Oryza sativa*)中茉莉酸-异亮氨酸衍生物(JA-Ile)含量显著升高,且在 *OsJAZ9* 过表达株系中其含量进一步增加,使 JA 信号响应增强,提高了植株的耐低钾能力[51]。在气孔调控方面,JA 与脱落酸(ABA)协同作用可促进 Ca^{2+} 内流,进而提高拟南芥保卫细胞外向 K^+ 通道蛋白基因 *GORK* 的表达量并增强其通道活性,最终导致 K^+ 外流,驱动保卫细胞收缩和气孔关闭[52]。番茄中,JA 通过减弱 SlJAZ1 对转录因子 SlWRKY81 的抑制作用,增强 Na^+/K^+ 转运蛋白的表达,促进 K^+ 吸收[53]。综上,JA 信号通路在维持植物体钾离子稳态中发挥重要作用,其通过多层次的调控机制影响钾离子通道活性和转运蛋白功能。

2 植物响应缺钾胁迫的分子机制

如图 2 所示,植物在长期进化过程中形成了精细的分子调控网络以响应缺钾胁迫,该网络主要包括钾离子动态平衡的跨膜转运系统、钙信号传递系统、活性氧介导的信号级联、多层级基因表达调控系统以及 microRNA 的调控系统[54-56]。

2.1 K^+转运通道与信号通路

在钾稳态维持方面,K^+转运蛋白与通道蛋白协同作用,参与根系吸收、木质部装载及液泡钾的再动员等过程,形成具有空间特异性的钾调控体系[57]。钙信号和活性氧作为关键信号分子,参与调控钾转运蛋白及通道蛋白活性[58]。这些机制在拟南芥、水稻、玉米等模式作物中既表现出一定的保守性,又存在物种特异性差异。



HAK、HKT:高亲和性钾离子转运体;CPA:阳离子-质子反向转运体;TPK:双孔钾离子通道;*CBL*:钙调磷酸酶 B 样蛋白;*CIPK*:CBL 互作蛋白激酶;AKT1:拟南芥钾转运体 1;HAK5:高亲和性钾转运蛋白 5;ROS:活性氧;*RCI3*:Ⅲ型过氧化物酶。

图 2 植物缺钾胁迫的信号调控网络

Fig.2 Signal regulatory network of plant potassium deficiency stress

植物在长期进化过程中形成了高度保守的钾离子转运系统,钾离子转运系统主要包括 3 类转运蛋白家族(KUP/HAK/KT、HKT 和 CPA)和 2 类通道蛋白家族(Shaker、TPK/KCO)[59],这些蛋白质主要定位于细胞膜和液泡膜系统,在维持钾离子稳态中发挥关键作用。

KUP/HAK/KT 蛋白是高等植物中普遍存在的高亲和性钾转运蛋白,如拟南芥 AtHAK5 和 AtKUP7、水稻 OsHAK5 以及玉米 ZmHAK1 和 ZmHAK5,它们通过调控钾离子在植物体中的分布增强植物对缺钾胁迫的适应性[57, 60-61]。HKT 家族是单子叶植物特有的钾转运蛋白,根据结构特征可分为Ⅰ类(Na^+单向转运)和Ⅱ类(Na^+/K^+共转运),在维持离子稳态和抗逆性中具有重要作用[62-63]。CPA 家族包含 NHX(Na^+/H^+交换蛋白)、CHX(阳离子/H^+交换蛋白)和 KEA(K^+外流逆向转运蛋白)3 个亚家族,它们通过协同调控 Na^+、K^+等离子稳态及细胞内的 pH 值动态平衡,在植物响应盐胁迫过程中发挥重要作用[64-65]。

Shaker 家族钾通道蛋白是维持细胞钾稳态的重要蛋白,其中拟南芥 AKT1 是典型的电压门控钾通道,其活性受 CBL1/9-CIPK23 复合物的磷酸化调控,在水稻和木薯等作物中也发现了 AKT 蛋白这一调控机制[66-69]。值得注意的是,拟南芥特有的钾离子通道蛋白 AtKC1 可通过与 AKT1 互作调节其活性,而在水稻中没有这一调控机制[69-70]。TPK 家族是液泡膜定位的钾通道蛋白,在缺钾条件下,液泡中的 K^+被运输到细胞质以维持细胞质的 K^+浓度[3]。拟南芥 AtTPK1 活性受钙依赖性蛋白激酶(*CDPK*)调控,AtTPK2/3/5 和 AtKCO3 受 CBL2/3-CIPK3/9/23/26 复合体的钙依赖性调控,表明钙信号在钾稳态调控中具有重要作用[71-72]。

2.2 Ca^{2+}-CBL-CIPK 信号传导系统

在缺钾胁迫条件下,植物通过 Ca^{2+}信号级联系统启动多层次的适应性调控网络。低钾环境首先激活质膜 Ca^{2+}通道,诱导胞质 Ca^{2+}浓度瞬时升高,从而触发钙信号转导途径[54]。该信号被钙调磷酸酶 B 样蛋白(CBL)识别,进而激活 CBL 互作蛋白激酶(*CIPK*),形成功能性 CBL-CIPK 复合体。研究结果表明,植物主要通过增强钾离子吸收以及促进液泡钾库的再动员应对钾缺乏,这 2 种机制分别由不同的 CBL-CIPK 复合体调控[3, 58]。其中,CBL1/9-CIPK1/9/23 复合体通过磷酸化激活质膜钾通道蛋白 AKT1[67]和钾转运蛋白 HAK5[73],促进细胞对钾离子的吸收;而 CBL2/3-CIPK3/9/23/26 复合体则通过调控液泡膜钾通道蛋白 TPK1/3/5 促进液泡钾离子释放到细胞质中[74]。在 K^+充足的条件下,CBL1/9、CBL2/3、CIPK9/23 和 AKT1 等蛋白质水平较低且处于非磷酸化状态;随着 K^+浓度降低,这些蛋白质逐渐积累并发生磷酸化[75]。值得注意的是,在钾缺乏条件下,CBL2/3 蛋白优先被磷酸化,激活液泡内储存的 K^+释放到细胞质中,使胞质 K^+浓度稳定[75]。

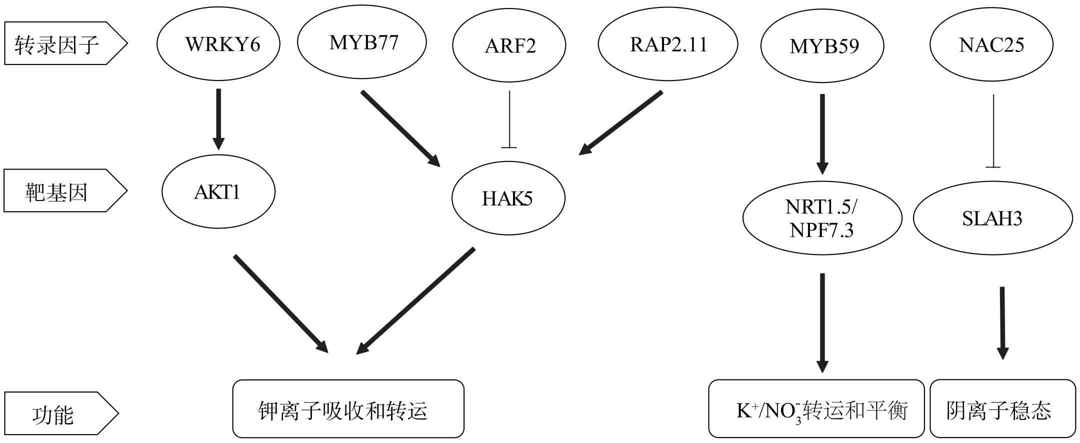
2.3 活性氧信号途径

缺钾胁迫触发植物体内活性氧(ROS)信号网络的级联响应,其通过特异性积累调控多种适应性机制。研究结果表明,在低钾条件下,拟南芥根部 ROS 含量显著增加,ROS 信号通过诱导高亲和性钾转运蛋白 HAK5 的表达响应低钾胁迫[76]。在低钾条件下,低钾敏感番茄(*Solanum lycopersicum*)品种的根和叶中 ROS 浓度显著升高,但其分子机制尚待阐明[77]。在信号转导层面,缺钾胁迫可特异性激活 ROS 代谢相关酶活性,其中Ⅲ型过氧化物酶 *RCI3* 基因的表达量上升,促进 ROS 积累,而积累的 ROS 作为重要的信号分子,激活了下游信号通路,最终诱导了 *HAK5* 基因的表达[78]。进一步研究发现,ROS 通过激活质膜上的离子转运系统,提高了植物对钾离子缺乏的适应性[79]。综上,ROS 动态变化是植物响应钾离子缺乏的重要指标,而 *RCI3* 和 *NADPH* 作为 ROS 信号通路的关键组分,与 HAK5 共同构成核心调控模块,协同介导植物对低钾胁迫的适应性反应。

2.4 转录调控

如图 3 所示,转录因子在植物响应缺钾胁迫过程中发挥重要调控作用,通过特异性调控钾转运相关基因的表达建立适应性响应网络。WRKY6 转录因子突变体表现出对低钾胁迫的高度敏感性,而 WRKY6 转录因子过表达株系则表现出对低钾胁迫较强的耐受力,进一步的研究结果表明,低钾胁迫可快速诱导 *WRKY6* 基因的表达,WRKY6 蛋白能直接调控钾离子通道基因 *AKT1* 的转录水平[80]。拟南芥中 MYB77 转录因子通过正向调控 *HAK5* 基因表达促进高亲和性钾吸收[55],而生长素响应因子 ARF2 则通过低钾诱导的磷酸化修饰解除对 HAK5 的转录

抑制,从而增强钾吸收能力[81]。AP2/ERF 家族成员 RAP2.11 在乙烯-ROS 信号级联作用下被激活,通过特异性结合 *HAK5* 启动子 GCC-box 元件正向调控其基因表达[82]。MYB59 转录因子通过调控硝酸盐转运蛋白 NRT1.5/NPF7.3 基因的表达影响 K^+/NO_3^- 转运平衡,在缺钾条件下,该调控机制有助于维持根系钾稳态和活性[83]。NRT1.5/NPF7.3 还通过调控 PIN2 蛋白介导的生长素分布,参与低钾胁迫下的根系发育过程[84]。在水稻中,OsNAC25 通过结合 *OsSLAH3* 启动子抑制其转录,从而增强耐低钾能力[85]。在褪黑素诱导下,小麦 TaNAC71 与 *TaHAK1* 启动子结合以增强其表达,进而提升钾吸收效率[86]。



WRKY6:WRKY 家族转录因子 6;MYB77:MYB 家族转录因子 77;ARF2:生长素响应因子 2;RAP2.11:AP2/ERF 家族转录因子;MYB59:MYB 家族转录因子 59;NAC25:NAC 家族转录因子 25;AKT1:拟南芥钾转运体 1;HAK5:高亲和性钾转运蛋白 5;NRT1.5/NPF7.3:硝酸盐转运蛋白 1.5/硝酸盐、肽转运蛋白 7.3;SLAH3:S 型阴离子通道 3。

图 3 植物缺钾响应的转录调控基础

Fig.3 Basis of transcriptional regulation in response to potassium deficiency in plants

2.5 microRNA 调控

缺钾胁迫触发植物 microRNA 介导的复杂转录后调控网络,通过多靶点协同调控离子稳态与代谢平衡[56]。小麦(*Triticum aestivum* L.)中 miR160 作为关键根系发育调控因子,与 miR169、miR166 和 miR165 共同参与低钾胁迫响应[87]。在番茄(*Solanum lycopersicum*)中,miR168a 通过靶向 *SlAGO1A* 基因激活细胞分裂素和脱落酸信号通路,显著提高植株低钾耐受性[88]。在缺钾条件下,水稻(*Oryza sativa*)OsmiR399 被特异性激活,进而影响钾转运蛋白 KT、磷转运蛋白 LTN1/OsPHO2 基因的表达[89]。SlmiR319b 通过靶向抑制 *SlTCP10* 的表达,进而解除 SlTCP10 转录因子对 *SlJA2* 基因表达的抑制,SlJA2 通过促进 ABA 的生物合成,调控钾离子的转运与吸收,最终提高番茄对低钾胁迫的耐受性[90]。

3 展望

本文系统阐述了植物响应缺钾胁迫的生理特征变化与分子调控机制。在生理层面,植物通过整合光合作用、水分代谢、酶活调控及激素信号网络等过程建立适应性响应策略。赤霉素、乙烯和茉莉酸等植物激素在缺钾响应中发挥关键作用,然而其信号感知机制、转导途径及与钾稳态调控之间的分子关联仍需深入解析。在分子层面,植物进化出多层次的调控网络,包括高亲和性钾转运/通道系统、Ca^{2+}-CBL-CIPK 信号通路、ROS 信号级联、转录调控及表观遗传修饰等机制,共同精细调控植物缺钾响应基因的表达。目前植物缺钾胁迫响应研究仍存在局限:其一,研究对象集中于拟南芥、水稻和玉米等模式作物或大田作物,对其他物种的研究较为匮乏;其二,分子机制研究集中于 *HAK5* 和 *AKT1* 等核心基因,对其他功能基因的研究不足;其三,甲基化、乙酰化和泛素化等表观调控机制在缺钾响应中的功能尚未得到系统解析。这些研究缺口严重制约了耐低钾栽培技术研发和分子育种进展。未来研究可以进一步拓展物种范围,系统解析不同植物的适应性机制;

加强对非经典调控基因的功能研究;深入探究表观遗传调控网络;基于功能基因组学发掘优异等位变异,为培育钾高效利用新种质提供基因资源,从而助力现代农业可持续发展。

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