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·专题论坛·

植物SnRKs家族在胁迫信号通路中的调节作用

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摘要 蔗糖非发酵1 (SNF1)相关蛋白激酶家族(SnRKs)是植物胁迫响应过程中的一类关键蛋白激酶。在响应生物胁迫时, SnRKs可通过参与活性氧和水杨酸介导的信号转导途径, 增强植物对生物侵害的耐受性。在响应非生物胁迫时, SnRKs通过脱落酸(ABA)介导的信号通路, 增强植株对干旱、盐碱和高温等的耐受性; 且通过独立于ABA的信号通路, SnRKs可调控胞内能量状态, 维持离子平衡。该文总结了SnRKs蛋白激酶作为胁迫信号通路中的主要调节因子的最新研究进展, 并展望了未来的研究方向。

关键词 植物, SnRKs, 非生物胁迫, 生物胁迫

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植物在其生活史中一直面对各种生物与非生物胁迫。为了生存, 植物必须感知周围环境的变化, 并且针对这些变化产生防御机制以平衡生存, 完成生长和繁殖等生命过程。为此, 植物进化出了复杂且精密的逆境响应机制。其中, 植物蔗糖非发酵1 (SNF1)相关蛋白激酶(sucrose nonfermenting-1-related protein kinases, SnRKs)家族作为植物体内响应胁迫的一类关键蛋白激酶家族, 通过级联放大胁迫信号调控细胞内多条信号通路, 启动胁迫应答。SnRKs根据其不同的蛋白质三级结构可以细分为3个亚家族——SnRK1、SnRK2和SnRK3 (Coello et al., 2011)。其中, SnRK1参与植物生命活动的氮、蔗糖和脂质代谢、器官发生以及衰老等过程, 且在逆境条件下通过调节能量和胁迫信号响应生物与非生物胁迫, 从而调节植物的生长发育(Baena-Gonzalez and Sheen, 2008; Confraria et al., 2013; Emanuelle et al., 2015); SnRK2亚家族成员因其C-末端具有2个独特的子结构域而与其它家族区分开(Yoshida et al., 2006)。而对SnRK2的定量磷酸化蛋白质组学分析表明, SnRK2的底物蛋白有84种, 其参与生长发育, 如花期调控、RNA和DNA的结合、miRNA与表观遗传调控、信号转导、叶绿体功能以及离子运输等多种逆境响应过程(Wang et al., 2013)。SnRK3蛋白激酶因有丰富的作用底物, 可以分别与CBL (calcineurin-B-like CBL calcium sensors)、SOS3 (salt overly sensitive)和ScaBP (SOS3-like calcium binding protein)相互作用(Dong et al., 2012), 因此, 又分别命名为CIPKs (CBL interacting protein kinases)、SOS2类蛋白激酶(SOS2-like kinases)或者与SOS2相关蛋白激酶PKSs (protein kinases related to SOS2), 它可与分布在质膜、液泡膜、细胞质基质以及细胞核中的10种CBL相互作用, 感受细胞内盐碱及干旱等胁迫信号, 启动细胞发生胁迫响应, 被认为位于植物能量代谢和胁迫信号连接的中心(Batistič et al., 2010; Thoday-Kennedy et al., 2015)。因此, 深入了解SnRKs的作用机制将有助于植物耐性改良。虽然植物SnRKs家族发现较早, 但是由于其蛋白种类繁多、来源广泛且功能多样, 因此人们对其作用机制了解得还比较分散。本文通过总结植物SnRKs家族参与逆境信号转导的研究进展, 旨在拓展对SnRKs在植物抗逆中作用的认识, 为植物抗逆机理的研究提供新思路。

1 植物SnRKs蛋白激酶家族

丝氨酸/苏氨酸蛋白激酶是一个相对较大的蛋白激酶

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家族, 其中, AMPK (AMP-activated kinase)/SnRKs是高度保守的丝氨酸/苏氨酸蛋白激酶家族的重要成员(Emanuelle et al., 2015)。 SnRKs可以细分成3个亚家族, 即SnRK1、SnRK2和SnRK3 (Coello et al., 2011)。酵母SNF1 (sucrose nonfermenting1)、蛔虫AAK (AMP-activated kinase)、昆虫AMPK、哺乳动物的AMPK以及植物的SnRK1都属于SnRK1蛋白激酶亚家族, 而SnRK2和SnRK3则为植物特有的亚家族(Ghillebert et al., 2011; Emanuelle et al., 2015)。

1.1 植物SnRK1蛋白激酶亚家族

在真核生物中, AMPK/SNF1/SnRK1蛋白激酶是进化上高度保守的代谢感知因子(Emanuelle et al., 2015)。SnRK1与SNF1和AMPK的氨基酸序列一致性均为48%, 其中催化域的氨基酸序列约有62%一致(Halford et al., 2004)。SnRK1通常是由具催化功能的α亚基、起调节作用的β亚基以及有活化功能的γ亚基组成的多聚体蛋白激酶(Emanuelle et al., 2015)。而拟南芥中SnRK1则是由α亚基、非典型的β_3亚基以及植物中特有的βγ亚基组成(Ramon et al., 2013; Emanuelle et al., 2015)。

1.2 植物SnRK2蛋白激酶亚家族

植物SnRK2亚家族成员含有2个典型结构域: N端催化结构域和C末端调节结构域(Halford and Hardie, 1998)。C末端结构域又包括2个子结构域, 即结构域1和结构域2。其中, 结构域1被称为ABA结合框, 因其富含负电荷, 可与PP2C (phosphatase 2C)结合; 结构域2主要是SnRK2的2和3亚组成员的特有结构, 富含天冬氨酸(Yoshida et al., 2006)。对SnRK2亚家族进行全基因组分析, 发现其在拟南芥(*Arabidopsis thaliana*)和水稻(*Oryza sativa*)中各有10个成员, 但是染色体定位在二者之间有差异(Wang et al., 2015a)。如在拟南芥所有染色体上都有SnRK2亚家族基因, 而在水稻中其分别位于1、4、7、10和12号染色体, 在小麦(*Triticum aestivum*)中则定位于1A、1B及1D染色体上, 暗示其生理功能可能因物种的不同而异(Tian et al., 2013; Saha et al., 2014)。

1.3 植物SnRK3蛋白激酶亚家族

植物SnRK3蛋白激酶具有丰富的作用底物, 使其在细胞胁迫信号通路与碳、氮代谢通路中均发挥重要作用, 它包括1个N末端激酶催化结构域、1个连接结构域和1个高度可变的C末端调节结构域(Sanyal et al., 2015)。其中, 催化结构域包括1个活化环, 调节结构域还可分为NAF (designated according to the prominent amino acids N, A and F)结构域和PPI结构域(protein phosphatase interaction) (Batistič and Kudla, 2009), 推测其保守的钙依赖性NAF结构域不仅是调控SnRK3的基础, 也是上游激酶可变调节的基础(Chaves-Sanjuan et al., 2014), 介导了SnRK3与CBL之间的相互作用(Kleist et al., 2014)。PPI是SnRK3与ABI相互作用时的结合位点(Lan et al., 2011)。同时, SnRK3激酶的激活需要活性环从活化位点释放以及NAF结构域从核苷酸结合位点释放, 从而基于NAF自抑制结构域与CBL的相互作用体现其生物活性(Hashimoto et al., 2012)。例如, SnRK3.23的活化环可折叠成有序的结构, 阻碍活性位点接触底物。而SnRK3.11的活性环能够产生活性位点, 并可进行催化反应(Kleist et al., 2014)。

2 植物SnRKs参与生物胁迫应答

2.1 植物SnRKs参与活性氧途径

植物面对生物胁迫时, 可以通过激活由模式触发免疫(pattern-triggered immunity, PTI)和效应触发免疫(effector-triggered immunity, ETI)组成的免疫反应, 启动对生物胁迫的抵御(Jones and Dang, 2006)。而SnRKs主要参与ETI信号通路。来自SnRK3亚家族的成员, 如SnRK3.14和CBL10在质膜上与RbohB (respiratory burst oxidase homologs B)相互作用, 产生活性氧(reactive oxygen species, ROS), 并抑制由丁香假单胞菌效应因子触发的细胞程序性死亡(programmed cell death, PCD), 增强植株防御生物胁迫的能力(Torre et al., 2013)。SnRK3.26也可与CBL1或CBL9强烈相互作用, 磷酸化Rboh, 正调控Rboh产生ROS (Drerup et al., 2013)。而在Kimura等(2013)的研究中发现, SnRK3.26还可作为Rboh的调控因子, 与其N末端结构域直接相互作用, 负调控ROS的产生。因此, SnRK3在响应生物胁迫时, 可以通过与不同底物的相互作用参与非生物胁迫中的ROS信号通路, 通过上调或下调ROS的产生, 协助

机体抵抗生物胁迫。

2.2 植物SnRKs参与水杨酸介导的信号通路

水杨酸(salicylic acid, SA)在植物中调控防御基因的表达及宿主细胞死亡, 从而在宿主获得系统性免疫(systemic acquired resistance, SAR)中发挥重要作用, 而NPR1 (nonexpressor of pathogenesis-related gene1)是SA介导信号通路中的主要调节因子(Vlot et al., 2009; Yan and Dong, 2014)。SA介导通路中的信号分子可以激活编码SnRK2.8蛋白激酶的基因, 上调该基因的表达(Lee et al., 2015)。同时, SnRK2.8也可磷酸化NPR1, 使其入核并调控相应的转录因子表达, 增强植株的SAR能力(Lee et al., 2015)。此外, 在拟南芥*SnRK3.22*基因缺失突变体中, 由于SnRK3.22磷酸化NPR1的缺失, 进入核内的NPR1减少, NPR1的靶基因*WRKY38* (sequence-specific DNA-binding proteins38 of the N-terminal sequence WRKYGQK)和*WRKY62*的表达下调, 减弱了植株对病虫害的抗性(Xie et al., 2010)。可见, 在植株受到生物胁迫时, SnRKs可通过磷酸化NPR1参与SA介导的信号通路, 从转录水平上迅速启动防御机制, 以增强植株抵抗生物胁迫的能力。

2.3 植物SnRKs参与生物逆境响应

在病毒或微生物入侵时, 植物SnRK1可以通过磷酸化帽结合蛋白、翻译起始因子或者病毒蛋白参与生物胁迫响应。拟南芥SnRK1也可以磷酸化双生病毒AL2蛋白, 抑制双生病毒的转录与翻译, 延迟白菜卷叶病毒的感染(Shen et al., 2014)。在小麦中, SnRK1与*TaFROG* (*Triticum aestivum fusarium resistance orphan gene*)编码的蛋白质相互作用, 增强小麦抵御禾谷镰刀菌的能力以及对呕吐毒素(deoxynivalenol, DON)的耐性(Perochon et al., 2015)。Kim等(2015)发现受到真菌和细菌病原体侵染的水稻*OSK35-D* (水稻SnRK1b的编码基因)激活型突变体能增强基因表达的重编辑, 从而提高对病原体的抗性。此外, 当双生病毒感染拟南芥时, SnRK1磷酸化AtREM4s (*Arabidopsis thaliana* remorin group 4s), 进而参与26S蛋白酶体途径, 降解甜菜严重曲顶病毒, 增强抗病性(Son et al., 2014)。

3 植物SnRKs参与非生物胁迫应答

3.1 植物SnRKs参与ABA信号介导的非生物胁迫应答

2009年, ABA受体的发现为研究SnRKs参与胁迫信号转导通路提供了重要的节点。其中, ABA作为胁迫的响应因子, 可分别与PYR/PYL/RCAR (pyrabactin resistant/PYR-like/regulatory component of ABA) ABA受体结合, 激活的ABA受体抑制包括ABI1 (abscisic acid-insenitive 1)、ABI2、HAB1 (homology to ABI1)、HAB2、AHG1 (ABA-hypersensitive germination 1)以及AHG3等在内的9个A组PP2C的活性。失去活性的A组PP2C无法抑制SnRK2s的活性, SnRK2s通过自身磷酸化激活。激活的SnRK2s磷酸化下游的靶标, 从而进一步放大胁迫信号, 增强植株对非生物胁迫的耐性(Yoshida et al., 2006; Hirayama and Umezawa, 2010; Danquah et al., 2014)。

植物SnRKs参与ABA信号介导的非生物胁迫应答大致可分为直接和间接2种途径。直接途径是SnRKs通过自身的磷酸化或被磷酸化, 直接参与ABA信号介导非生物胁迫过程, 增强植株对干旱或盐碱等非生物胁迫的耐性。例如, 在拟南芥中, SnRK2.2/SnRK2.3/SnRK2.6是ABA信号转导通路中的重要调节因子, 其中在保卫细胞中响应ABA的ABAR (abscisic acid receptor)可直接与SnRK2.6相互作用, 调控ABA信号转导, 进而调节气孔运动(Liang et al., 2015)。而在ABA信号通路中产生的NO, 还可使保卫细胞中的SnRK2.2/SnRK2.3/SnRK2.6的巯基亚硝基化, 抑制SnRK2.2/SnRK2.3/SnRK2.6的活性, 调节由ABA参与调控的种子萌发及幼苗生长(Wang et al., 2015b)。PP2Cs的3个成员(ABI1、AHG1和AHG3)与SnRK2.10强烈相互作用, 介导SnRK2.10参与ABA信号转导, 响应毛果杨(*Populus trichocarpa*)对干旱和盐碱胁迫的应答(Song et al., 2015)。

另外, 在ABA信号级联通路中, SnRKs可与ABA信号通路中的调节因子相互作用, 间接参与ABA信号转导途径, 调控植株对非生物胁迫的耐受性(Chen et al., 2012)。例如, ABI2可分别与SnRK3.11、SnRK3.13和SnRK3.15相互作用, 而ABI1能够与SnRK3.6发生强烈相互作用, 它们互作后都能通过调控相关基

因的表达增强植株对ABA信号的敏感性, 从而增强对盐胁迫的耐受性(Ohta et al., 2003)。在拟南芥中, SnRK3.22可以磷酸化ABI5, 间接参与ABA响应基因的表达, 从而调节种子发芽(Zhou et al., 2015b)。处于种子萌发期以及幼苗期的拟南芥, 在它们细胞核中, SnRK2.2、SnRK2.3和SnRK2.6通过磷酸化RAV1 (related to ABI3 (ABA-insensitive3)/VP1 transcription factor, RAV)负调控编码转录因子ABI3、ABI4和ABI5的表达, 参与ABA信号级联通路(Feng et al., 2014)。SnRK2.2、SnRK2.6和SnRK2.3 (SRK2D/E/I)还可激活调节胁迫应答基因表达AREB1 (ABRE (ABA-responsive element)-binding factors1) 、AREB2和AREB3, 从而调控植株响应渗透胁迫, 维持植物正常的营养生长(Yoshida et al., 2015)。最新研究发现, ABA信号通路的新成员植物U-Box类E3连接酶(PUB (plant U-box E3 ligases))、PUB12和PUB13可以通过与ABA的信号元件ABI1相互作用, 释放与ABI1结合的SnRK2.6 (也被称为OST1), 促进OST1对下游靶标的磷酸化。研究者基于此提出了依赖ABA作用的新模型(Xuan et al., 2015)。我们按照最新研究进展(Axel et al., 2016), 将SnRK2s参与的ABA途径总结为图1。

3.2 植物SnRKs参与不依赖ABA信号的非生物胁迫应答

3.2.1 植物SnRKs参与调节能量代谢

植物SnRK1作为细胞信号网络中的关键组分, 可以分别与细胞内代表能量状态的不同形式的糖作用, 参与调节能量状态转变的其它激酶活性, 并与调控能量状态的转录因子相互作用, 调节细胞内的能量平衡, 是能量和逆境信号的交汇点(Baena-González and Sheen, 2008)。例如, SnRK1可在黑暗、缺氧和盐碱条件下响应高浓度或低浓度的葡萄糖水平, 参与胁迫下植物能量代谢的调控(Polge and Thomas, 2007)。海藻糖-6-磷酸(T6P)、葡萄糖-6-磷酸(G6P)和葡萄糖-1-磷酸(G1P)可分别抑制SnRK1的活性(Nägele and Weckwerth, 2014)。在T6P/SnRK1信号通路中, 通过磷酸化过程抑制SnRK1活性, 加强细胞代谢活性, 加速生长(Lawlor and Paul, 2014)。绿色器官中, 核糖-5-磷酸在向核糖-1-磷酸转化过程中能量的消耗, 也可以间接抑制SnRK1的活性(Nunes et al., 2013)。SnRK1可与拟南芥中催化生成59-AMP的ADK (adenosine kinase)相互作用, 调节细胞中AMP与ATP之间的能量平衡(Mohannath et al., 2014)。此外, 拟南芥中的SnRK1可磷酸化转录因子bZIP63, 调节下游靶基因的表达, 从而调控体内能量平衡(Mair et al., 2015)。当细胞处于低能量状态时, AKIN10 (SnRK1的催化亚基)与拟南芥中的PTL (petal loss)相互作用, 抑制细胞分裂(O'Brien et al., 2015)。同时, AKIN10也可以磷酸化花期调控转录因子IDD8 (indeterminate domain-containing transcription factor8), 参与IDD8介导的代谢过程, 调控开花时间, 使花期延迟(Jeong et al., 2015)。

3.2.2 植物SnRKs参与调节离子平衡

植物SnRK3在参与调控细胞内离子平衡尤其在抵御盐碱胁迫时发挥重要作用。一方面, SnRK3蛋白家族成员参与最重要的胁迫信号转导通路(即SOS通路), SOS3和SCaBP8与SnRK3.11 (又被称为SOS2)相互作用, 激活SnRK3.11, 并在质膜上形成复合体活化Na^+/H^+泵, 维持细胞内Na^+/H^+平衡(Lin et al., 2014)。同时, Park等(2013)在研究拟南芥的开花启动时, 发现SnRK3.11可以与光周期和生物钟调控因子GI (gigantea)相互作用。在正常条件下, GI: SnRK3.11复合体可以抑制植物体内主要的Na^+/H^+泵(SOS1激酶)的活性, 介导对盐碱的适应性(Park et al., 2013)。在盐胁迫下, GI:SnRK3.11复合体崩解且释放SnRK3.11, 激活SOS1, 调控胞内Na^+/H^+平衡; 与此同时, SnRK3.11可以与多种蛋白相互作用, 调控植株不同生长发育时期的耐盐碱性(Park et al., 2013)。SnRK3还可以激活离子通道蛋白参与钾离子运输。例如, Wang等(2016)发现, CBL1/9-SnRK3.23复合体和AtKC1 (*Arabidopsis thaliana* K^+ rectifying channel 1) 可以共同调控AKT1 (Arabidopsis K^+ transporter), 平衡AKT1介导的低K^+响应中K^+的吸收与释放。此外, SnRK3-CBL复合体也可以激活VvK1.2 (*Vitis vinifera* K^+ channel 1.2)。激活的VvK1.2作为电压-门控, 当整流电压低于−100 mv, 且受到外界刺激时, 激活内向K^+通道, 介导K^+的快速运输(Cuéllar et al., 2013)。植物SnRK3通过与多种蛋白相互作用, 还可维持细胞内的Mg^{2+}平衡, 防御Mg^{2+}毒害。例如, 拟南芥SnRK3.17/SnRK3.12/SnRK3.23与液泡膜上的CBL

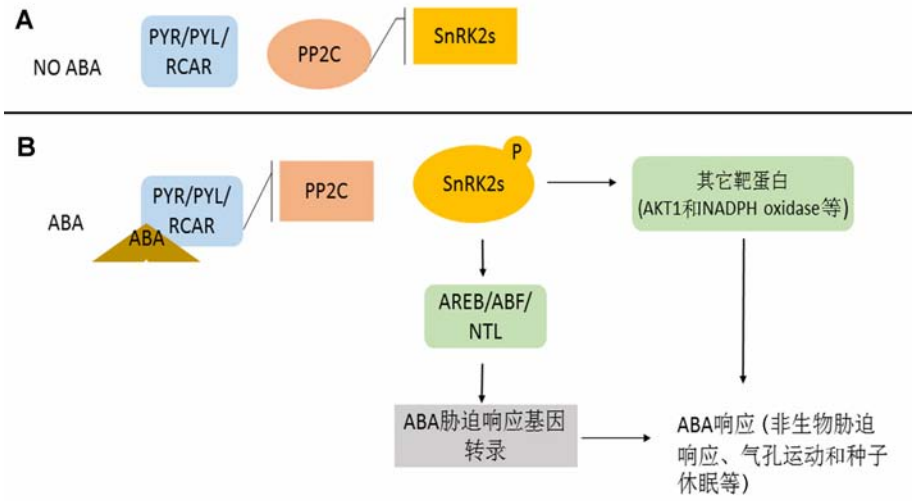

图1 植物SnRK2s参与的主要ABA途径(改自Axel et al., 2016)

(A) 无ABA时，ABA受体失活，PP2C抑制SnRK2s的活性；**(B)** 有ABA时，ABA受体与ABA结合，抑制PP2C活性，SnRK2s通过自身磷酸化激活，激活的SnRK2s磷酸化下游转录因子，磷酸化的转录因子调控ABA胁迫响应基因的表达。同时，SnRK2s也可以磷酸化其它蛋白，如AKT1及NADPH氧化酶等。这些事件共同引起植株体内ABA响应。

Figure 1 The plant SnRK2s is involved in the main ABA pathway (motified from Axel et al., 2016)

(A) In the absence of ABA, the ABA receptors are inactivated and PP2C (Protein Phosphatase 2C) proteins inhibit SnRK2s; **(B)** In the presence of ABA, the ABA receptors PYR/PIL/RCAR proteins bind to ABA and in turn inhibit PP2C activity, allowing the activation of SnRK2s through autophosphorylation. Activated SnRK2s then phosphorylate their downstream transcription factors, which modulate ABA responding gene expression. They can also phosphorylate other proteins, such as AKT1 and NADPH oxidases. Together, these events lead to the establishment of the ABA response.

2/3相互作用，共同螯合液泡中Mg^{2+} (Tang et al., 2015)。同时，拟南芥中的SnRK3.17/SnRK3.12/SnRK3.23 (又称CIPK3/9/23)还可与SnRK2D/E/I相互作用，通过感受细胞外高浓度Mg^{2+}，并通过与SnRK2D/E/I相互作用，传递胁迫信号，共同维持细胞内Mg^{2+}的动态平衡(Mogami et al., 2015)。

植物SnRK3还可与CBL相互作用，而CBL对于细胞内外的Ca^{2+}浓度变化敏感。因此，SnRK3与各种底物蛋白相互作用后，最终也会引起细胞内Ca^{2+}的浓度变化，参与Ca^{2+}信号通路，调控胞内Ca^{2+}浓度，维持细胞的正常生长发育，或者参与细胞内钙依赖的盐碱干旱胁迫响应。例如，*SnRK3.9*的过度表达诱导细胞产生严重液泡化表型，且*SnRK3.9*基因缺失突变体极性生长受损(Steinhorst et al., 2015)。这是由于液泡内的Ca^{2+}可以直接激活CBL2/3-SnRK3.9复合体，使得花粉管迅速生长(Steinhorst et al., 2015)。Zhou等(2015a)则发现*SnRK3.5*过表达可引起极性缺失，通过尖端膨胀引起胞内Ca^{2+}浓度升高。因此，SnRK3.5也可参与平衡Ca^{2+}流，避免Ca^{2+}毒害(Zhou, 2015a)。此外，SnRK3也可间接参与平衡胞内Ca^{2+}。如SnRK3.22一方面能够通过磷酸化调控质膜上的质子泵(ATP酶)活性，引起质膜内外pH值变化；另一方面，当其与SCaBP1结合时，质膜外高pH值可以触发增加胞浆内Ca^{2+}浓度。由于胞浆内质子浓度与盐胁迫相关，因此，SnRK3.22间接参与Ca^{2+}信号通路，这也是植物细胞迅速启动盐胁迫应答的一种重要方式(Fuglsang et al., 2007)。

3.2.3 植物SnRKs参与调控非生物胁迫下植株的生长发育

植物SnRKs可以通过磷酸化与生长发育过程中的各种调节因子相互作用，直接参与调控非生物胁迫下植株的生长发育，增强植株对胁迫的耐性。烟草(*Nicotiana tabacum*) SnRK1可以与作为种子萌发负调控因子的HSPRO (Ortholog of sugar beet Hs1pro-1)相互作用，调控幼苗的发育(Schuck et al., 2013)。马铃

薯(*Solanum tuberosum*)中细胞程序性死亡抑制因子Adi3 (AvrPto-dependent Pto-interacting protein3)可以磷酸化SnRK1的β亚基，磷酸化的SnRK1不能与底物特异性相互作用，抑制SnRK1活性，调节生长发育过程(Avila et al., 2012)。AKIN10可通过磷酸化IDD8延迟花期(Jeong et al., 2015)。Kim等(2012)则在研究拟南芥响应干旱胁迫时，发现SnRK2.8磷酸化转录因子NTL6 (NAC (nam/ataf1/2/cuc2) with transmembrane motif1-like6)，延缓植株在响应干旱胁迫时的休眠进程。此外，在拟南芥响应盐胁迫时，SnRK2.4和SnRK2.10可以与磷脂酸(phosphatidic acid, PA)结合，参与不依赖ABA的非生物胁迫信号通路，其中SnRK2.4有磷脂酸结合区域(PA-binding domain, PABD)，此结构域影响拟南芥的根系发育(Wang et al., 2015b)。

综上所述，我们总结了植物不同SnRKs家族基因参与细胞内不同的生命过程(表1)。

4 植物SnRKs参与不同胁迫信号通路途径间的可能联系

4.1 植物体内SnRKs的平衡调节机制

当植物受到胁迫时，SnRKs迅速感受刺激并产生应激反应。此时，SnRK1主要通过调整代谢来改变细胞内的能量胁迫状态(Halford and Hey, 2009)。但是，当应激反应强度过大且时间持久时，活化的SnRK1则触发自身的小型泛素化修饰(small ubiquitin-like modifier, SUMO)过程，通过蛋白酶体途径进行降解，进而抑制细胞的正常生理活动(Nägele and Weckwerth, 2014; Crozet et al., 2016)。此外，SnRK1的降解是严格依赖其自身活性的(Crozet et al., 2016)。例如，无活性的SnRK1在植物体内是非常稳定的，但是，当SnRK1被激活时，同时启动了SUMO化过程(Crozet et al., 2016)。此外，Rodrigues等(2013)发现，A组PP2C作为ABA介导的胁迫信号通路中的抑制因子，可以与SnRK1的催化亚基相互作用使其去磷酸化并失活。同时，SnRK1靶基因表达分析进一步证明，由于ABA抑制PP2C使SnRK1激活，在能量匮乏时，SnRK1激活下游的靶标(Rodrigues et al., 2013)。可见，SnRK1的失活与激活可能通过自身的严格控制而达到平衡。

4.2 植物SnRK1、SnRK2和SnRK3间的交互作用

植物SnRKs基因家族种类庞大，虽然对其生理功能的研究都是独立进行的，但随着研究的深入，一些最新证据也表明该家族不同亚基基因之间也存在互作。例如，在拟南芥中，SnRK3.15可以与SnRK1.1和SnRK1.2相互作用，调节SnRK1.1和SnRK1.2活性，参与调节糖代谢(Yan et al., 2014)。Mogami等(2015)发现，SnRK3.17/SnRK3.12/SnRK3.23可以与SnRK2D/E/I相互作用，且SnRK3.17/SnRK3.12/SnRK3.23与CBL相互作用后，将Mg^{2+}胁迫信号转导并参与由SnRK2D/E/I介导的信号通路中，通过磷酸化过程共同调控细胞内Mg^{2+}平衡。因此，植物SnRKs不同亚家族成员间的交互作用，强化了植株响应胁迫的能力。可见，研究植物SnRK1、SnRK2和SnRK3三者间的交互作用，将有助于理解细胞内以SnRKs为中心枢纽的细胞信号网络。

5 展望

植物SnRKs蛋白激酶家族是一个庞大的家族，其功能包括细胞能量的正常运转以及植株响应外界胁迫两大方面。SnRK1主要是在调节植株的生长发育、维持能量平衡以及响应生物逆境方面发挥重要作用; SnRK2则在响应干旱盐碱等非生物胁迫中发挥重要作用; SnRK3比较特殊，由于其更多地参与胞内离子浓度的平衡，通过调节离子浓度，迅速启动植株响应非生物胁迫。随着组学技术的广泛应用，尤其是磷酸化、泛素化以及甲基化等蛋白质组学方法的普及，今后有望进一步利用磷酸化蛋白质组分析不同SnRKs家族与底物互作的作用机制，并利用代谢组分析SnRKs参与的不同代谢过程，从而构建SnRKs在细胞内与底物之间的作用机制网络，以便高效解析种类多样的SnRKs家族基因的功能。其次，鉴于SnRKs激酶家族成员在生物胁迫与非生物胁迫响应信号通路的功能，深入揭示不同信号通路之间的交互作用也将是未来研究的重要方向。第三，虽然现在对SnRKs已有较多的了解(Zulawski et al., 2013)，但是不同的*SnRKs*基因在感受胁迫时如何调节细胞内的能量状态，尤其在生存、生长和繁殖等不同生命过程中的实

表1 拟南芥*SnRKs*基因家族成员的功能分类

Table 1 Different functions of *SnRKs* gene family of Arabidopsis

功能	途径	SnRKs亚家族基因名称	基因座位号	基因证号	参考文献
信号	糖	*SnRK1.1* (KIN10)	At3G01090	821259	Polge and Thomas, 2007
		SnRK1.2 (KIN11)	At3G29160	822566	Polge and Thomas, 2007
		SnRK1.3	At5G39440	833940	Mair et al., 2015
	钙离子	*SnRK3.16* (CIPK1)	At3G17510	821016	D'Angelo et al., 2006
		SnRK3.5 (CIPK19)	At5G45810	834621	Zhou et al., 2015a
	硝酸盐	*SnRK3.13* (CIPK8, PKS11)	At4G24400	828542	Hu et al., 2009
激素	脱落酸	*SnRK2.2*	At3G50500	824214	Feng et al., 2014
信号		*SnRK2.3*	AT5G66880	9305254	Wang et al., 2015a
		SnRK2.6 (OST1)	At4G33950	829541	Yoshida et al., 2015
		SnRK3.14 (CIPK6)	At4G30960	829221	Chen et al., 2013
		SnRK3.17 (CIPK3, PKS12)	At2G26980	817240	Kim et al., 2003
		SnRK3.22 (CIPK11, PKS5)	At2G30360	817586	Zhou et al., 2015b
	水杨酸	*SnRK2.8*	At1G78290	844164	Lee et al., 2015
		SnRK3.22 (CIPK11, PKS5)	At2G30360	817586	Xie et al., 2010
生物	活性氧	*SnRK3.26* (CIPK26)	At5G21326	832246	Drerup et al., 2013
胁迫		*SnRK3.14* (CIPK6)	At4G30960	829221	Chen et al., 2013
非生物	非生物胁迫	*SnRK3.1* (CIPK15, PKS3)	At5G01810	830556	Kanwar et al., 2014
胁迫		*SnRK3.2* (CIPK2)	At5G07070	830598	
		SnRK3.6 (CIPK20, PKS18)	At5G45820	834622	
		SnRK3.7 (CIPK13)	At2G34180	817979	
		SnRK3.8 (CIPK10)	At5G58380	835951	
	寒冷	*SnRK3.10* (CIPK7, PKS7)	At3G23000	821874	Huang et al., 2011
		SnRK3.17 (CIPK3, PKS12)	At2G26980	817240	Kim et al., 2003
	缺氧	*SnRK3.25* (CIPK25)	At5G25110	832582	Meena et al., 2015
	渗透胁迫	*SnRK3.18* (CIPK16, PKS15)	At2G25090	817047	Amarasinghe et al., 2016
		SnRK3.4 (CIPK21)	At5G57630	835868	Pandey et al., 2015
		SnRK2.7 (SRK2F)	At4G40010	830162	Fujii et al., 2011
		SnRK2.9	At2G23030	816833	Fujii et al., 2011
		SnRK2.1 (SRK2G)	At5G08590	830760	Fujii et al., 2011
	盐碱	*SnRK3.9* (CIPK12)	At4G18700	827604	Steinhorst et al., 2015
		SnRK3.11 (SOS2, CIPK24)	At5G35410	833502	Park et al., 2013
		SnRK3.12 (CIPK9, PKS6)	At1G01140	839349	Mogami et al., 2015
		SnRK3.15 (PKS24, CIPK14)	At5G01820	831765	Lin et al., 2014
		SnRK3.22 (CIPK11, PKS5)	At2G30360	817586	Zhou et al., 2015b
		SnRK3.18 (CIPK16, PKS15)	At2G25090	817047	Amarasinghe et al., 2016
		SnRK3.23 (CIPK23, PKS17)	At1G30270	839907	Wang et al., 2016
生长	根生长	*SnRK2.10*	At1G60940	842385	McLoughlin et al., 2012
发育		*SnRK2.4* (ASK1, SRK2A)	At1G10940	837637	McLoughlin et al., 2012
		SnRK3.25 (CIPK25)	At5G25110	832582	Meena et al., 2015
	种子发芽	*SnRK3.22* (CIPK11, PKS5)	At2G30360	817586	Zhou et al., 2015b
	幼苗生长	*SnRK1.1* (KIN10)	At3G01090	821259	O'Brien et al., 2015
	开花	*SnRK1.1* (KIN10)	At3G01090	821259	Jeong et al., 2015

数据来源包括基因库http://www.ncbi.nlm.nih.gov/。Sources include GenBank http://www.ncbi.nlm.nih.gov/.

时转换机制尚属未知。随着对其研究和认识的深入，今后有望在调节植物抗逆和高产之间的矛盾中找到一个共同的节点，或可提供一个新视角。这将是一个重要的研究课题，将为改良作物和获得抗逆性状指明

一个新方向。

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The Function of Sucrose Nonfermenting-1 Related Protein Kinases in Stress Signaling

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Abstract SnRKs (sucrose nonfermenting-1-related protein kinases) are key protein kinases in stress responses. In response to biotic stresses, they are involved in reactive oxygen species and salicylic acid-mediated signaling transduction pathways to enhance the plant tolerance to biological stress. In response to abiotic stresses, SnRKs enhance the plant tolerance to drought, salinity and high temperatures by intracellular signaling mediated by abscisic acid (ABA), regulate cellular energy homeostasis and maintain ion balance by ABA-independent signaling. SnRKs are the main regulators in stress signaling in recent research and we give a brief outlook for future study.

Key words plant, SnRKs, abiotic stresses, biotic stresses

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