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植物气孔发育的分子遗传调控

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摘要 气孔广泛存在于植物地上组织和器官的表皮, 是植物与外界环境进行气体交换的主要门户, 调节光合作用和蒸腾作用等生理活动. 原表皮细胞经过一系列固定的分裂和分化, 最终产生成熟气孔. 在气孔发育过程中, bHLH转录因子调控气孔细胞的起始、扩增和分化, 受体-配体、MAPK信号级联介导的细胞间通讯确保正确的气孔发育图式的形成, 极性蛋白调节气孔细胞不均等分裂的方向. 此外, 植物激素和环境因子也影响气孔发育. 这些因子共同构建出植物气孔发育的分子遗传调控网络. 本文综述了该网络及其最新研究进展.

关键词 气孔发育, 图式形成, 不均等分裂, 细胞分化

距今约4亿年前, 伴随着从水生进化为陆生, 植物表皮产生了气孔结构. 气孔的产生早于维管、根、叶、种子和花等组织器官, 表明气孔在植物基础生理活动中扮演着极其关键的角色[1]. 气孔由一对保卫细胞构成, 通过膨压高低变化调节开闭, 使植物在相对干旱的环境中, 既能有效防止水分散失, 又能充分保证与外界进行气体交换, 以便光合和呼吸作用能够正常进行. 同时, 气孔调节的蒸腾作用既促进了水分和矿物质在植物体内的运输, 又可以有效降低叶片表面的温度. 气孔在植物表皮均匀分布, 发育图式(pattern)几乎遵循“一个细胞间隔”(one cell spacing)的规则, 即两个气孔之间至少间隔一个非气孔表皮细胞, 使气孔与周围细胞能够进行高效的水分和离子交换, 确保气孔很好地行使功能. 因此, 气孔图式发育必然受到严格调控.

双子叶植物气孔一般由一对肾形保卫细胞构成, 发育起始于部分散分布的原表皮细胞. 这些细胞获得了拟分生组织母细胞(meristemoid mother cell, MMC)的命运并进行不均等起始分裂(asymmetric entry division), 产生两个大小形态各异的细胞. 小的三角形细胞称为拟分生组织细胞(meristemoid), 大的姊妹细胞称为气孔系基础细胞(stomatal lineage ground cell, SLGC). 拟分生组织细胞能够进行0~3次不均等扩增分裂(asymmetric amplifying division), 产生新的拟分生组织细胞和更多的SLGCs. 拟分生组织细胞最终转变为保卫母细胞(guard mother cell, GMC). GMC进行一次均等分裂(symmetric division)产生一对保卫细胞(guard cell, GC), 形成气孔. SLGC可以直接分化为扁平细胞, 也能获得MMC命运, 在远离气孔及气孔前体细胞(拟分生组织细胞和GMC)的位置进行不均等间隔分裂(asymmetric spacing division), 产生卫星拟分生组织细胞并最终发育为气孔. 不均等间隔分裂使气孔发育遵循“一个细

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胞间隔”的原则(图1A)[2~5].

单子叶植物, 如水稻(*Oryza sativa*)、玉米(*Zea mays*)和二穗短柄草(*Brachypodium distachyon*), 气孔由一对哑铃状的保卫细胞及两侧的一对副卫细胞(subsidiary cell, SC)构成, 气孔发育起始于气孔列原表皮细胞. 这些原表皮细胞具有MMC特性, 朝同一方向垂直于叶片轴向进行不均等分裂, 产生一个大的扁平细胞和一个小的拟分生组织细胞. 后者直接转变为GMC. 随着发育的进行, GMC可以诱导两侧的副卫母细胞(subsidiary mother cell, SMC)发生极化, 在SMC与GMC的接触面形成微丝斑, 同时使SMC的细胞核朝向GMC迁移. 随后, SMC平行于叶片轴向进行不均等分裂, 产生SC和扁平细胞. 最后, GMC平行于叶片轴向均等分裂产生一对GC(图1B)[6,7], 最终形成由一对哑铃状GC及其两侧的一对SC构成的四细胞结构. 因此, 单子叶气孔发育图式也遵循“一个细胞间隔”的原则.

近年来, 大量的分子遗传学研究揭示, 气孔的分化及“一个细胞间隔”发育图式的形成受到细胞内外信号、植物激素以及环境因子的共同调控, 并由此构建出气孔发育的分子遗传调控网络. 本文将着重综述该分子遗传调控网络的最新研究进展.

1　双子叶植物气孔发育机制

目前, 对气孔发育分子机制的理解主要来源于双子叶模式植物拟南芥(*Arabidopsis thaliana*)气孔发育的研究. 通过广泛的遗传筛选, 众多调控气孔分化和发育图式的关键因子被发现, 主要包括转录因子、受体、配体、MAPK信号级联模块以及植物激素和环境因子, 它们共同构建出双子叶植物气孔发育的遗传调控网络(图2)[2,3,8~10].

1.1　转录因子调控气孔分化

植物原表皮层(L1)的分化是气孔发育起始的先决条件. 同源异形盒转录因子*ML1*(meristem layer 1)和*PDF2*(protodermal factor 2)调控表皮层的建成和维持, 二者功能同时缺失导致表皮层分化缺陷[11]. 超表达*ML1*和*HDG2*(homeodomain glabrous 2)(与*ML1*和*PDF2*同一家族的另外一个同源异形盒转录因子, 在拟分生组织细胞中表达)诱导叶片内部形成表皮层和气孔(图2)[12,13].

3个紧密相关的bHLH(basic helix-loop-helix)转录因子*SPCH*(speechless), *MUTE*和*FAMA*依次在特异气孔系细胞表达, 分别调控MMC向拟分生组织细胞、拟分生组织细胞向GMC以及GMC向GC的细胞命运转变(图2)[14~16]. 尽管原表皮细胞获得MMC命运的分子机制还不清楚, 但该过程需要*SPCH*. *SPCH*在表皮细胞广泛表达, 而SPCH蛋白仅被限定在MMC和拟分生组织细胞, 表明SPCH蛋白受到严格的翻译后调控. *spch*突变体不能起始气孔发育, 叶表皮全部由扁平细胞构成. 相反, 超表达*SPCH*促进气孔发育起始, 叶表皮细胞过度分裂产生大量的气孔系细胞. 同时, *SPCH*还能维持

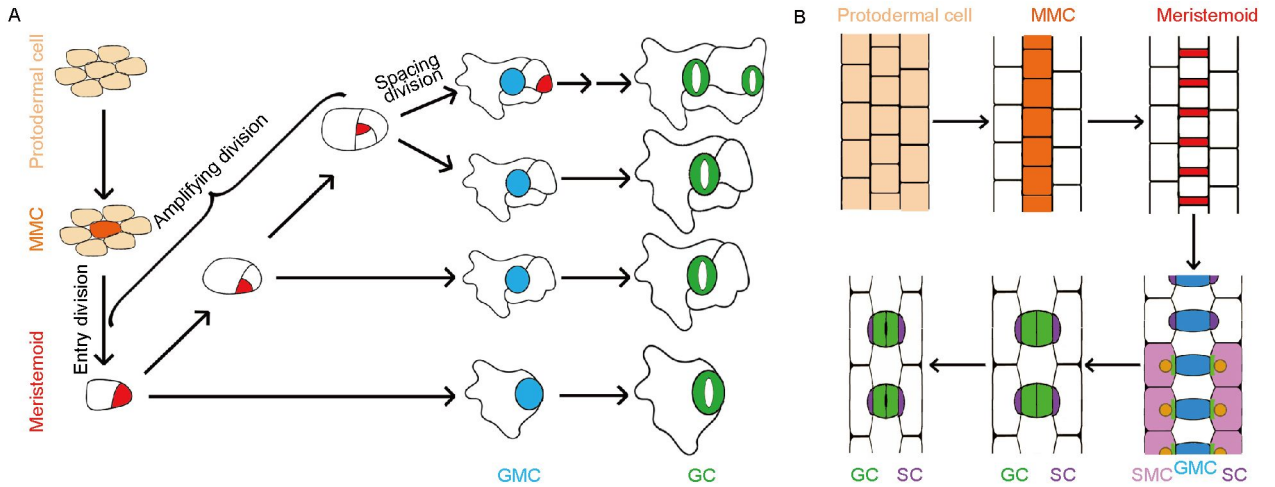

图 1　双子叶和单子叶植物气孔发育模式图

A: 双子叶植物气孔发育模式图; B: 单子叶植物气孔发育模式图

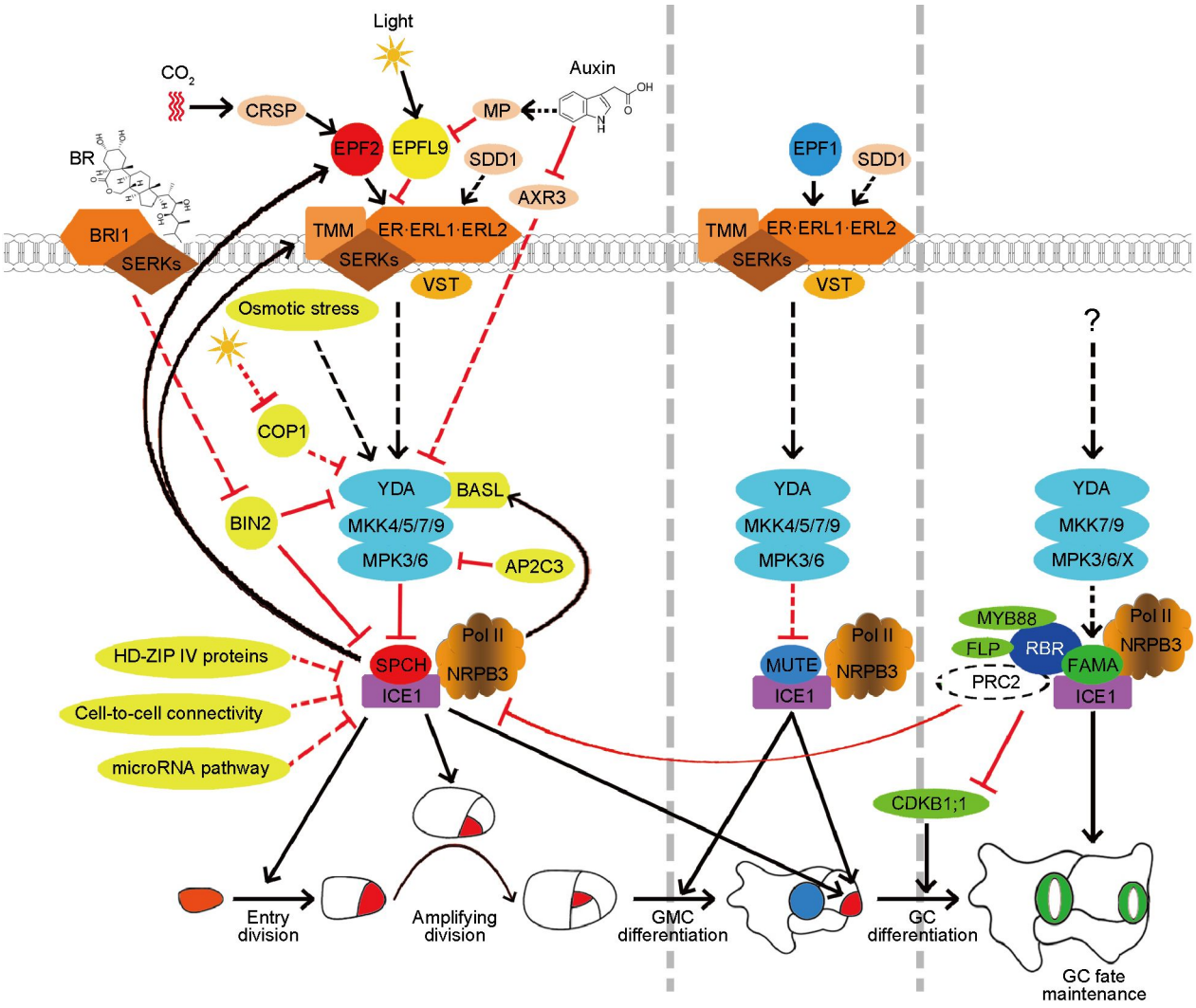

图 2　拟南芥气孔发育的分子遗传调控网络示意图

MAPK信号级联介导的植物激素信号和环境信号也调控拟分生组织细胞向GMC以及GMC向GC的分化, 图中没有展示

拟分生组织细胞的干细胞活性, 促进其分裂[15~17]. *MUTE*在晚期拟分生组织细胞表达. *MUTE*功能缺失导致拟分生组织细胞过度不均等分裂而不分化, 产生花环状结构, 而异位表达*MUTE*导致表皮细胞全部转变为气孔[15,16]. 因此, *MUTE*与*SPCH*在气孔发育中功能相反, *SPCH*促进MMC和拟分生组织细胞进行不均等分裂, 而*MUTE*终止拟分生组织细胞的干细胞活性. 与此相一致的是, SPCH蛋白与MUTE蛋白具有重叠但不同的表达模式(SPCH在晚期拟分生组织细胞中逐渐消失的同时MUTE逐渐出现)[18]. *FAMA*在GMC和GC表达. *FAMA*功能缺失突变体中GMC进行多次均等分裂产生一列细长的细胞, 呈毛毛虫状. 但是, 这些细胞不具有GC特性. 超表达*FAMA*导致在不同组织中产生未成对的保卫细胞[14]. 此外, 两个与*FAMA*功能冗余的同源R2R3 MYB转录因子*FLP*和*MYB88*也调控GMC向GC的分化(图2). *flp*突变体中GMC进行至少一次均等分裂, 通常产生两个平行排列的气孔. *myb88*突变体气孔发育正常, 而*flp myb88*双突变体中GMC多次分裂产生类似*fama*的表型, 但这些细胞具有GC特性. 这3个转录因子可能通过抑制共同的靶基因如*CDKB1;1*(cyclin dependent kinase b1;1)等协同限制保卫母细胞的分裂[19~21].

另外, 两个在气孔系细胞中广泛表达的同源bHLH亮氨酸拉链(bHLH-LZ)转录因子ICE1(inducer of CBF expression 1, 也被称作SCRM(scream))和SCRM2也促进气孔分化(图2). *ice1*产生类似*fama*的表型, 叶表皮形成毛毛虫样的结构; *ice1 SCRM2*+/–产生类似*mute*的表型, 叶表皮形成花环状的结构; *ice1 scrm2*产生类似*spch*的表型, 叶表皮无气孔产生. SCRMs能够与SPCH, MUTE和FAMA分别形成异源二聚体调控整个气孔分化过程中细胞命运的转变[22]. 最新研究发现, FAMA和ICE1能够与RNA聚合酶Ⅱ第三大亚基NRPB3(the third largest subunit of RNA polymerase Ⅱ)互作, 将气孔发育信号传递到RNA聚合酶Ⅱ这一关键的转录装置来执行特异的转录调控(图2)[23].

1.2 胞间信号调控气孔“一个细胞间隔”发育图式的形成

受体-配体介导的细胞间通信对于动植物器官、组织的图式发育至关重要. 目前发现的调控气孔发育的受体包括质膜上的富亮氨酸重复类受体蛋白TMM(too many mouth)、类受体激酶ER(erecta)家族成员(ER, ERL1(erecta like 1)和ERL2), 以及SERK(somatic embryogenesis receptor kinase)家族成员(SERK1–SERK4)(图2). 这些受体在气孔系细胞中广泛表达, 其中TMM, ERL1, ERL2在分裂活性较高的气孔系细胞表达较强. *tmm*, *er erl1 erl2*以及*serk1 serk2 serk3*突变体叶表皮均产生气孔簇[24~27]. 这些受体形成复合物, 接收EPFL(epidermal patterning factor like)家族的分泌性小肽信号, 抑制或促进特异组织或器官表皮的气孔发育. 第一个被发现的小肽基因*EPF1*在晚期拟分生组织细胞、GMCs和GCs中表达, 主要依赖受体*ERL1*抑制拟分生组织细胞的分化和促进正确的不均等间隔分裂. *EPF1*功能缺失突变体形成气孔簇, 而超表达植株无气孔产生但存在SLGCs[26,28]. *EPF2*在MMCs及早期拟分生组织细胞中表达, 编码的小肽能够结合受体ER并激活下游MAPK信号来抑制气孔发育起始. *EPF2*功能缺失导致气孔发育起始增加, 产生大量气孔系细胞; 超表达*EPF2*导致表皮无气孔发育起始, 仅由扁平细胞构成[26,29~31]. 与*EPF1*和*EPF2*不同, *STOMAGEN*(*EPFL9*)在叶肉组织中表达并促进气孔发育. STOMAGEN与EPF2竞争性结合ER但不能激活下游MAPK信号, 从而阻断了EPF2对气孔发育的抑制(图2). 沉默*STOMAGEN*可以抑制气孔形成, 而超表达*STOMAGEN*产生气孔簇[31~34]. *tmm*下胚轴和茎表皮无气孔形成, 然而EPFL家族成员*CHAL*(challah)/*EPFL6*的功能缺失能使*tmm*下胚轴和茎表皮产生气孔. *CHAL*在茎和下胚轴维管组织周围的细胞表达, 主要依赖*ER*家族受体负调控气孔发育, 而受体*TMM*抑制该信号[35,36]. *STOMAGEN*和*CHAL*都在表皮下组织表达而调控气孔发育, 表明EPFL小肽介导的胞间通讯不仅存在于表皮层, 而且存在于不同的组织层之间, 从而将气孔发育调控扩展到三维空间[37]. 此外, 枯草杆菌蛋白酶类的丝氨酸蛋白酶*SDD1*(stomatal density and distribution 1) 也作用于*TMM*上游调控气孔发育(图2). *SDD1*在拟分生组织细胞和GMCs中表达. *SDD1*功能缺失突变体气孔密度升高2~4倍, 并成簇; 超表达*SDD1*抑制气孔分化, 气孔密度下降. *SDD1*独立于*EPF1*和*EPF2*负调控气孔发育, 推测SDD1能够剪切加工未知小肽前体, 产生被受体识别的抑制气孔发育的配体[38,39].

气孔发育受体-配体信号的下游是一个MAPK(mitogen activated protein kinase)信号级联模块, 由MAPKKK(YDA), MKK4/5/7/9和MPK3/6构成(图2). 这些激酶的功能缺失导致表皮气孔剧烈成簇, 而其持续激活导致表皮无气孔产生. 利用*SPCH*, *MUTE*和*FAMA*启动子, 分别在特异气孔系细胞中表达显性负效应的YDA或持续激活的YDA, MKKs, 发现YDA-MKK4/5/7/9-MPK3/6信号级联模块抑制MMCs向拟分生组织细胞, 以及拟分生组织细胞向GMC的分化; YDA-MKK7/9-MPK3/6/X(代表未知MPK)信号级联促进GMC向GC的分化[40,41]. MAPK信号的活性水平对于气孔发育至关重要. 磷酸酶AP2C3能够去磷酸化MPK3/6, 从而抑制MPK3/6的活性, 促进气孔发育. *AP2C3*超表达诱导表皮细胞几乎全部分化为气孔, 该表型类似于*mpk3 mpk6*双突变体[42]. 最近研究发现, VST(vap-related suppressors of tmm)蛋白能与ER家族类受体激酶互作, 促进内质网与质膜接触, 增强受体介导的气孔发育信号向下游MAPK信号模块转导[43]. MAPK信号能够通过MPK3/6磷酸化抑制SPCH, 将信号传递到bHLH转录因子(图2)[44].

1.3 极性蛋白调控MMC和拟分生组织细胞的不均等分裂

动物中, 保守的PAR(partitioning defective)蛋白复

合物在母细胞中极性表达, 诱导纺锤体极性定位, 在细胞不均等分裂的过程中将细胞命运决定因子有差别地分配到两个子细胞, 使其获得不同的命运. 植物中没有PAR蛋白的同源物, 但极性蛋白BASL(breaking of asymmetry in the stomatal lineage)和POLAR(polar localization during asymmetric division and redistribution)作用模式类似PAR蛋白[45,46]. *BASL*调控MMCs和拟分生组织细胞进行不均等分裂. *basl*突变体气孔系细胞几乎均等分裂, 产生大小均等和命运相同的姊妹细胞, 形成气孔簇. BASL表现出动态的细胞边缘和细胞核的双重定位, 该动态定位与细胞命运密切相关: BASL仅定位于细胞边缘时, 细胞分化为扁平细胞; 仅定位于细胞核时, 细胞分化为气孔; 同时定位于细胞边缘和细胞核时, 细胞进行不均等分裂[45]. MPK3/6介导的磷酸化对于BASL的极性定位是必需的. 磷酸化的BASL能够募集YDA和MPK3/6, 使MAPK级联信号在细胞边缘增强. 活化的MPK3/6一方面通过磷酸化BASL进一步增强细胞边缘的MAPK级联信号, 另一方面通过磷酸化降解SPCH, 使SLGCs退出气孔分化(图2)[47]. POLAR极性定位于MMCs和拟分生组织细胞中远离不均等分裂的位置. POLAR的极性定位依赖于BASL, 表明POLAR可能是BASL介导的信号通路的一个组分[46].

1.4 正负反馈回路调控气孔发育

发育图式的形成需要一个局部自我增强的反应偶联一个长距的拮抗反应[48]. 气孔图式发育也不例外, SPCH作为分子开关位于气孔发育信号的最上游, 直接结合气孔发育基因的启动子并激活其表达, 这些基因包括*SCRMs*, *TMM*, *EPF2*, *ERL2*, *BASL*和*POLAR*. 其中, SCRMs能够结合自身启动子并进一步激活*SCRMs*表达, 形成正反馈环; 同时, SCRMs也能直接激活*EPF2*和*TMM*的表达, 反过来通过MAPK信号级联磷酸化抑制SPCH的活性, 进而抑制*SCRMs*表达, 形成一个长距的负反馈环(图2). 以此为基础构建的数学模型能模拟所有已知气孔突变体的表型[49].

1.5 保卫细胞特化命运的维持

植物形成类型和功能各异的细胞, 那么这些细胞是如何维持各自特化命运的呢? 气孔保卫细胞为回答这一问题提供了理想的研究模型. $FAMA_{pro}$::*FAMA-GFP*(*FAMA*^{trans}) 和 FLP_{pro}::*FLP-GFP* (*FLP*^{trans})转基因植株中, 特化的保卫细胞失去终末分化命运, 获得MMC命运并起始气孔发育, 在保卫细胞中产生新的气孔, 形成SIS(stoma-in-stoma)气孔表型[50,51]. 沉默*RBR*(retinoblastoma related)基因也产生类似的气孔表型. RBR能与FAMA, FLP/MYB88互作[51,52]. 进一步研究发现, FAMA含有与RBR特异互作的基序LxCxE, 该基序突变导致二者不能互作, 产生SIS表型. 因此, 推测*FAMA*^{trans}和*FLP*^{trans}可能通过干扰FAMA与RBR的互作, 产生SIS表型. FAMA与RBR都能结合气孔发育基因*SPCH*, *EPF1*和*FAMA*的启动子[52]. SIS表型与*SPCH*, *MUTE*启动子的H3K27me3水平相关. 超表达PRC2复合体(polycomb repressive complex 2)蛋白CLF(curly leaf)能使SIS表型植株中*SPCH*, *MUTE*启动子的H3K27me3从较低水平恢复到较高水平, 并抑制SIS表型[50]. RBR能够与PRC2复合物组分互作. 因此, 在GC中, FAMA, FLP/MYB88与RBR互作, RBR招募染色质修饰蛋白如PRC2复合体, 进而抑制气孔发育基因表达, 实现GC终末分化命运的维持(图2)[52].

1.6 植物激素和环境因子调控气孔发育

目前发现, 植物激素如BR, ABA、生长素以及环境因子如CO_2、光和渗透胁迫等调控气孔发育(图2). BR信号通路组分BIN2(BR insensitive 2)在子叶中能够磷酸化抑制YDA, 而在下胚轴中能够直接磷酸化抑制SPCH. 因此, BR通过失活BIN2, 既能促进下胚轴表皮气孔发育, 又能通过增强YDA的活性负调控子叶表皮气孔发育(图2)[53,54]. BR的合成前体甾醇的早期合成通路缺陷导致叶表皮产生小细胞团和气孔簇, 该通路独立于已知气孔发育信号途径来调控气孔系细胞命运的定向和维持[55]. ABA在调节气孔开闭中起着重要作用, 同时ABA合成缺陷导致气孔密度升高, 而外源施加ABA或内源ABA积累导致气孔数目减少, 表明ABA抑制气孔发育[56]. 生长素参与调控气孔发育过程中不均等分裂向均等分裂的转变, 同时生长素的转运也调控气孔图式发育[57]; 在暗下, 生长素抑制气孔发育, 而生长素抗性突变体*axr3-1*促进气孔发育, *axr3-1*介导的气孔发育信号位于bHLH转录因子和MAPK信号模块的上游, 部分依赖于ER家族受体(图2)[58]; 此外, 更为深入的研究表明, 生长素通过转录因子MP/ARF5直接抑制*STOMAGEN*在叶肉细胞的表达来负调控气孔发

育, 建立了生长素信号和配体信号在气孔发育中的交互作用(图2)[59].

一般情况下, 高浓度CO_2抑制气孔发育, 但拟南芥*hic*(high carbon dioxide)突变体对CO_2浓度不敏感, 表明HIC可能通过感受CO_2浓度的变化负调控气孔发育[60]. 此外, CO_2结合蛋白βCA1(β-carbonic anhydrases 1)和βCA4也参与CO_2浓度介导的气孔发育, 双突变体产生类似*hic*的表型[61]. 最新研究发现, CO_2诱导表达胞外蛋白酶CRSP(CO_2 response secreted protease)来剪切EPF2前体, 产生有活性的EPF2来抑制气孔发育(图2)[62]. 与植物生长一样, 气孔发育也受光强、光质的影响. 高光强度下, 植物气孔数目升高但发育图式不受影响, 研究发现光照强度能通过调节*STOMAGEN*的表达来促进气孔形成[63]. 不同波长的光也影响气孔发育, 例如, 高强度红光促进气孔发育, 而远红光抑制气孔发育[64,65]. *phyB*突变体不能响应红光诱导的气孔数目增加, 表明红光受体phyB(phytochrome B)在红光诱导的气孔发育中起关键作用. phyB互作因子PIF4(phytochrome-interacting factor 4)功能缺失产生类似*phyB*的气孔发育表型[65]. 此外, 红光/远红光受体phyA(phytochrome A)和蓝光受体CRY1(cryptochrome1), CRY2在高光强下也能促进气孔产生. 光信号和气孔发育信号可能通过E3泛素连接酶COP1(constitutive photomorphogenic 1)连接起来. *cop1*突变体气孔密度增加、气孔成簇. COP1在遗传上位于YDA的上游而平行于TMM(图2)[66]. 另外, 渗透胁迫通过降解SPCH, 从而减少MMCs来抑制气孔发育, 该过程依赖于MAPK信号级联(图2)[67].

1.7 影响气孔发育的其他因子

细胞壁的完整性和通透性以及microRNA信号也调控气孔图式发育. 类葡聚糖合成酶GSL8(glucan synthase-like 8)以及类糖基转移酶蛋白KOBITO1的突变导致细胞壁完整性产生缺陷或胞间连丝通透性增大, 使共质体大分子, 包括气孔命运决定因子, 如SPCH, 在细胞间的扩散增加, 产生气孔簇(图2)[68,69]. *miR824*识别并降解MADS box家族成员*AGL16*(Agamous-like16)的mRNA, 调控卫星拟分生组织细胞的形成[70]. 此外, miRNA通路组分*AGO1* (ARGONAUTE1)促进SLGC连续进行不均等间隔分裂. *AGO1*遗传上位于受体*TMM*下游, 负调控*SPCH*转录(图2)[71].

2 单子叶植物气孔发育机制

单子叶植物气孔分化机制的研究非常有限. 最近发现, 二穗短柄草*BdSPCHs*和*BdICE1*调控气孔发育起始; *BdSPCH2*是GMC命运决定因子, 超表达*BdSPCH2*能够使叶毛细胞分化为气孔; *BdSCRM2*调控气孔复合物的形成, 其功能缺失导致GC不能成熟, 产生停止发育的四细胞复合物(图3)[72]. 单子叶植物副卫细胞极化调控方面的研究相对比较丰富. 研究发现, LRR受体激酶PAN1(pan-gloss1)和PAN2可能作为受体识别来源于GMC的未知信号, 调控SMC的极性分裂; 二者功能缺失均产生分裂异常的SMCs. PAN1和PAN2都极性定位于SMC与GMC接触面的SMC细胞膜上, 且PAN1的极性定位依赖于PAN2. PAN1与PAN2不能互作, 而PAN2能够形成同源二聚体[73,74]. 另外, ROP2, ROP9以及SCAR/WAVE复合体也极性定位于SMC和GMC的接触面. ROP2/9与PAN1互作调控SMC的极性分裂[75]. SCAR/WAVE复合体的极性定位早于PAN1和PAN2, 可能通过未知蛋白间接和PAN2互作, 促使PAN2极性定位(图3)[76].

3 展望

人们对气孔分化和图式发育的分子机制有了更深入的了解, 然而, 还存在许多问题亟待解决. 例如, *SPCH*, *MUTE*和*FAMA*的表达如何被调控? 受体-配体信号如何传递到MAPK信号模块? *BASL-POLAR*调控细胞极性和不均等分裂的机制是什么? MAPK信号级联如何调控MUTE和FAMA? 此外, 虽然目前发现表观遗传调控*EPF2*和*ER*的转录[77,78], 但是表观遗传在气孔发育中扮演的角色还有待深入研究. 另外, 单子叶植物气孔发育过程不同于双子叶, 暗示可能存在新的分子遗传调控机制. 随着新的生物学技术的发展, 将为回答以上问题提供有力的解决方案.

植物气孔发育过程易于追踪和观察, 是研究众多基础生物学问题的理想模型, 包括发育图式形成、植物与环境互作、细胞间信号转导、细胞增殖与分化、细胞极性与不均等分裂、细胞特化命运维持以及植物进化等[4]. 气孔发育及其运动调节的研究将在提高农业生产和用水效率方面发挥重要作用, 为粮食和用水安全带来长远利益, 以应对多变的全球气候变化[79].

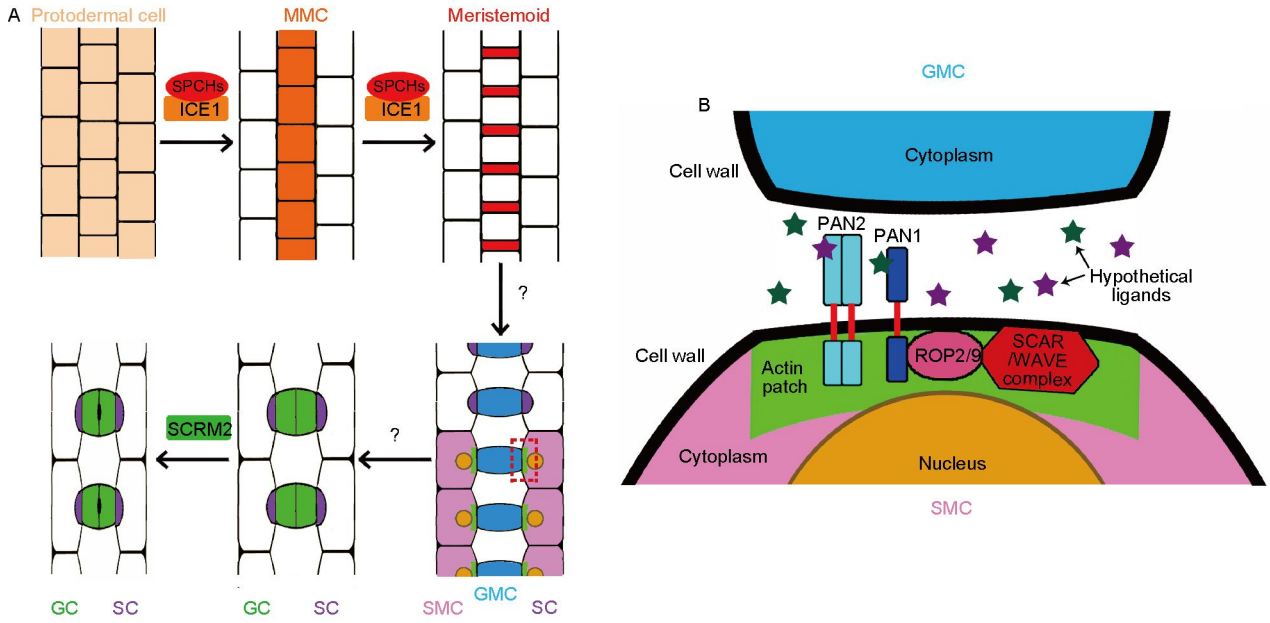

图 3　单子叶植物气孔发育的分子遗传调控示意图

A: 单子叶植物气孔分化的分子遗传调控示意图; B: 单子叶植物副卫细胞极化的分子遗传调控示意图(B图是A图中红色虚线框部分的放大): 在SMC发育的早期, 可能来源于GMC的信号诱导SCAR/WAVE复合物首先极性定位于SMC与GMC的接触面, 导致PAN2, PAN1和ROP依次极性定位; PAN1能够与ROP互作并激活ROP, 活化的ROP进而激活SCAR/WAVE复合物, 最终导致微丝斑形成和细胞核迁移

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Molecular genetic control of plant stomatal development

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Stomata widely exist in the epidermis of plant shoot. They are the main passageway for gas exchange between plants and the environment. Stomata regulate many physiological processes, such as photosynthesis and transpiration. The protodermal cells undergo a series of stereotypical cell divisions and differentiation, and finally produce stomata. Several transcription factors control the initiation, proliferation and differentiation of stomatal cells in different developmental stages. The cell-cell communication, which is mediated by the ligand-receptor and MAPK cascade, ensures correct stoamtal patterning. A few polarity proteins have been reported to direct the orientation of asymmetric cell divisions. Additionally, plant hormones and environmental factors also influence stomatal development. All these factors together build the molecular genetic network of stomatal development. In this review, we summarize the recent advances of the network.

stomatal development, patterning, asymmetric cell division, cell differentiation

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