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Transcriptional control of cell fate in the stomatal lineage

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The Arabidopsis stomatal lineage is a microcosm of development; it undergoes selection of precursor cells, asymmetric and stem cell-like divisions, cell commitment and finally, acquisition of terminal cell fates. Recent transcriptomic approaches revealed major shifts in gene expression accompanying each fate transition, and mechanistic analysis of key bHLH transcription factors, along with mathematical modeling, has begun to unravel how these major shifts are coordinated. In addition, stomatal initiation is proving to be a tractable model for defining the genetic and epigenetic basis of stable cell identities and for understanding the integration of environmental responses into developmental programs.

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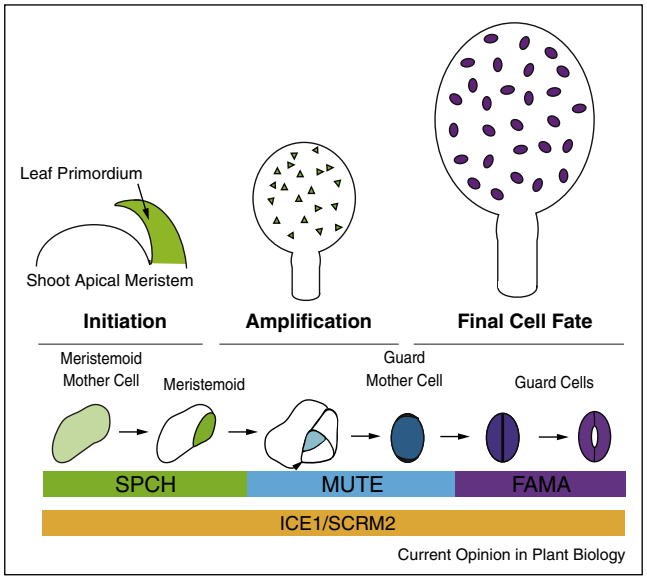
Life on land requires plants to balance acquisition of carbon from the atmosphere with loss of water from internal tissues. More than four hundred million years ago, plants solved this problem by generating stomata in their aerial organs; stomata are small epidermal pores surrounded by guard cells that open and close in response to homeostatic cues and signals from the environment. Today, stomata are present and essential in nearly all land plants, but their structure and distribution display clade-specific patterns and often require multi-step developmental programs. Because of their essential nature, but flexible pattern, stomata are a useful model for understanding a myriad of developmental processes including asymmetric and stem-cell like divisions, cell fate acquisition (and its connection to gene regulation and epigenetics), cell–cell communication, and responses to hormones and environmental cues. A mechanistic understanding of stomatal development has arisen primarily from studies in the dicot *Arabidopsis thaliana*, and this review will focus on this species, but recent work in Maize has shed light on how cell polarity is established in a multicellular context [1•] and careful morphological assessments in diverse species suggest ways in which control over the orientation of early divisions in the stomatal lineage lead to different leaf patterns [2,3].

Stomatal development in *A. thaliana* proceeds through a stereotyped sequence of cell divisions and cell fate transitions in the epidermis (Figure 1). Stomatal lineage cell types can be defined morphologically and by expression of fate markers, the latter used with great success in combination with extended time-lapse imaging to define the trajectory of individual cells [4–7]. Such studies, along with classical lineage tracing, show that in the young leaf, a subset of protodermal cells in the epidermis, known as meristemoid mother cells, will divide asymmetrically to produce a small, usually triangular, meristemoid and a larger daughter cell known as an SLGC (stomatal lineage ground cell). The SLGC may differentiate directly into a pavement cell, or divide asymmetrically to generate a satellite meristemoid. Each meristemoid continues to divide asymmetrically, typically twice more, before it undergoes a fate and morphological transition into a round guard mother cell (GMC). The GMC is the first committed stage in the stomatal lineage; until they become GMCs, cells are developmentally plastic and may take on other epidermal fates. GMCs will divide a final time, symmetrically, to produce the two guard cells (GCs) of a stoma. Divisions of the meristemoid and SLGC are oriented through cell–cell communication to ensure that two stomata do not form in direct contact with one another.

Each stage in the stomatal lineage requires precise transcriptional control over cell identity and behavior. Five basic helix–loop–helix (bHLH) transcription factors lie at the core of this regulation. Stomatal initiation and subsequent meristemoid self-renewal requires the first of these factors, SPEECHLESS (SPCH). *SPCH* RNA is broadly expressed in the young leaf, but SPCH protein is restricted to meristemoids and enables their continued asymmetric division [4,8,9•]. When meristemoids exit this 'SPCH' stage, they begin expressing the transcriptional factor MUTE and exhibit substantial changes in global gene expression [10,11••]. MUTE is required for GMC fate, as loss of function mutants get stuck in a continuous self-renewing stage. When overexpressed, MUTE converts all epidermal cells into stomatal precursors [10]. One final

Figure 1



Overview of stomatal development in the context of the developing leaf. Leaf primordia develop from the shoot apical meristem and the epidermis is already specified at this time. During the initiation phase, meristemoid mother cells (MMCs) are created; these cells in the developing leaf will begin to express SPCH protein and divide asymmetrically to form meristemoids (green). The amplification stage is dominated by cell divisions of MMCs and meristemoids that generate the epidermis and establish proper stomatal patterning. In later phases of leaf development (final cell fate stage) meristemoids transition to guard mother cells (blue) and then to differentiated guard cells (purple) while other cells expand to drive the increase in leaf size. The expression patterns of key transcriptional regulators discussed in this review are indicated at the bottom.

transition from GMC to GC requires a symmetric division and is preceded by expression of FAMA [12]. FAMA is necessary for the acquisition of GC identity, but also for continued maintenance of this identity and this latter function is mediated by FAMA in conjunction with RBR (RETINOBLASTOMA RELATED) [13••,14]. At each of their specific expression stages, SPCH, MUTE and FAMA act as obligate heterodimers with one of two more broadly expressed, but still stomatal lineage enriched, bHLHs, ICE1/SCREAM (SCRM) or SCRM2 [15]. Loss of both *ICE1* and *SCRM2* eliminates the stomatal lineage, resembling loss of *SPCH*, whereas stabilizing mutations (*scrm-D*) convert all epidermal cells into stomatal guard cells [15]. Because SPCH, MUTE and FAMA expression and activities nicely parallel the different stomatal cell types and transitions, we will use these factors as organizing nodes for the rest of this review.

Limiting stomatal lineage competence to the epidermis

True leaves develop from the shoot apical meristem where tissue layers are already established; the epidermis is derived from anticlinical divisions of the L1 layer, whereas the underlying mesophyll and vascular tissues are derived from deeper L2 and L3 layers. Several HOMEODOMAIN LEUCINE ZIPPER CLASS IV (HD-ZIP IV) proteins are required for epidermal identity beginning in the embryo and loss of two such factors; MERISTEM LAYER 1 (*ML1*) and PROTODERMAL FACTOR 2 (*PDF2*), results in plants that lack an epidermis [16]. A third HD-ZIP IV family member, HOMEODOMAIN GLABROUS 2 (HDG2) is expressed in meristemoids, but surprisingly, Peterson *et al.* (2013) found that ectopic overexpression of HDG2 (or ML1) can induce stomata to form within the mesophyll. Overexpression of MUTE with the same promoter, however, was not capable of inducing internal guard cells [15] suggesting that epidermal identity is a prerequisite for stomatal lineage fates; consistent with this finding, overexpression of ML1 and HDG2 leads to the appearance of a SPCHp:GUS reporter in mesophyll cells suggesting a transformation to the earliest stomatal lineage fate [17].

SPCH as master regulator and point of integration for various signals

SPCH promotes asymmetric divisions that initiate, amplify and space future stomata in the epidermis and therefore where and how often it is expressed will define stomatal pattern and density [8]. In addition to generating stomata, the stomatal lineage builds most of the epidermis, including pavement cells [18] and possibly trichomes [11••]. Although protodermal cells that do not express SPCH may divide symmetrically to produce pavement cells, the bulk of the epidermis is derived from cells that have expressed SPCH at some point. Therefore, SPCH represents the logical point for numerous signaling cascades to converge to regulate leaf size, stomatal patterning and stomatal density in response to a variety of signals (Figure 2).

Regulating the pattern of stomata: control over initiation and direction

SPCH protein has been shown to be regulated through phosphorylation by MITOGEN ACTIVATED PROTEIN KINASE (MAPKs), GLYCOGEN SYNTHASE 3 KINASE (GSK3) and CYCLIN DEPENDENT KINASE (CDK) families [19–21]. Phosphorylation by MPKs or the GSK3 BIN2 targets SPCH for degradation, and non-phosphorylatable forms of SPCH have increased expression, leading to increased entry divisions and ultimately larger leaves and a higher stomatal density [19,21,22]. MAPKs, GSKs and CDKs upstream of SPCH are broadly expressed in the leaf, so additional information is required to activate these kinase pathways in specific cells to produce the normal pattern of SPCH activity. This information comes, in part, from cell-type specific expression of receptor TOO MANY MOUTHs, some members of the ERECTA (ER) family of receptor kinases and ligands in the EPIDERMAL PATTERNING FACTOR (EPF)

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