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Systemic signaling during plant defense

Aardra Kachroo and Guillaume P Robin

Systemic acquired resistance (SAR) is a type of pathogen-induced broad-spectrum resistance in plants. During SAR, primary infection-induced rapid generation and transportation of mobile signal(s) 'prepare' the rest of the plant for subsequent infections. Several, seemingly unrelated, mobile chemical inducers of SAR have been identified, at least two of which function in a feed-back regulatory loop with a lipid transfer-like protein. Signal(s) perception in the systemic tissues relies on the presence of an intact cuticle, the waxy layer covering all aerial parts of the plant. SAR results in chromatin modifications, which prime systemic tissues for enhanced and rapid signaling derived from salicylic acid, which along with its signaling components is key for SAR induction. This review summarizes recent findings related to SAR signal generation, movement, and perception.

Addresses
Department of Plant Pathology, University of Kentucky, Lexington, KY 40546, United States

Corresponding author: Kachroo, Aardra (apkach2@uky.edu)

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Introduction

Systemic acquired resistance (SAR) is a highly desirable form of resistance that provides long-lasting (up to several months) and broad-spectrum resistance in plants [1,2]. SAR involves the generation of a mobile signal(s) in the primary infected tissues (within 4–6 hours), its rapid translocation to systemic uninfected tissues, and preparation of the systemic tissue for subsequent infections from related/unrelated pathogens. In addition to the individual plant, SAR also has transgenerational benefits, where immune 'memory' is passed on to the next generation [3••,4••]. The onset of SAR involves key events including, first, SAR signal(s) generation in the primary infected site; second, systemic translocation of the SAR signal(s); third, signal(s) perception in the systemic tissue; and fourth induction of a 'defense-ready' status. Many molecular components of SAR have been identified and discussed extensively in several recent reviews [5–8]. However, not much is known about how they all function together to eventually orchestrate SAR. This review focuses on recently identified SAR regulators that affect the four main events listed above.

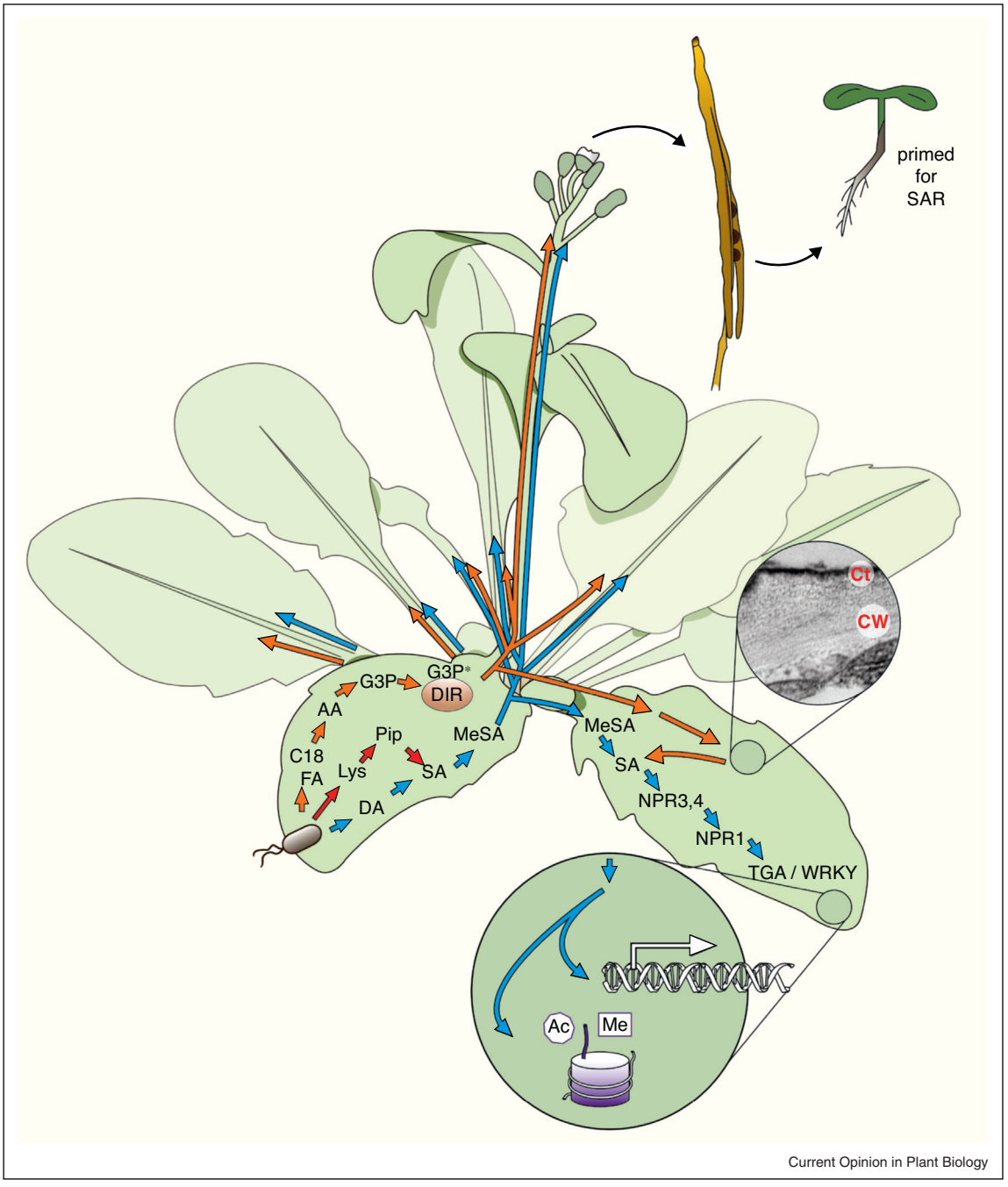
Signal(s) generation

SAR is induced upon the onset of effector-triggered immunity in the primary tissues infected with avirulent pathogen. Several studies now show that SAR can also be triggered by virulent pathogens [3••,9,10•,11•], although an early study showed that unlike avirulent pathogen, induction of SAR required high doses of virulent pathogen [12]. Except for a link between changes in amino acid homeostasis and the initial onset of defense signaling and SAR, not much is known about step(s) leading to initiation of SAR [13]. Characterization of the SAR deficient *ald1* mutant had led to the proposition that an amino acid derivative generated from the activity of the ALD1 aminotransferase might contribute to the initial SAR signal [14]. A recent study linked the SAR defect of the *ald1* mutant to its inability to accumulate the lysine derived non-proteinacious amino acid, pipecolic acid (Pip) [10•]. Pip accumulates soon (8 hours) after pathogen infection and can induce foliar resistance when supplied to the plant via roots. Pip induces significant accumulation of the defense phytohormone salicylic acid (SA) and primes for SA signaling. This suggests that Pip might serve as an early SAR signal upstream of SA (Figure 1). However, the mobile SAR inducer status of Pip is pending direct evidence of its systemic movement, and demonstration of its ability to induce systemic resistance when applied in a localized manner.

Mobile signal(s)

Three main characteristics determine a defense activator as a mobile SAR inducer: first, pathogen-responsive accumulation in the time-frame of signal generation (4–6 hours); second, translocation of the signal or a product thereof to systemic tissues within the time-frame of signal movement; and third, ability to rapidly induce resistance in distal (untreated) tissues. Recent studies focused on identifying such SAR inducers have generated several excellent candidates, not all of which satisfy every characteristic (Table 1). These include the methylated SA derivative, methyl SA (MeSA), a dicarboxylic acid, azelaic acid (AzA), an abietane diterpenoid, dihydroabetinal (DA), and a phosphorylated sugar derivative, glycerol-3-phosphate (G3P). All these chemicals induce systemic resistance when applied locally. AzA, DA, and G3P translocate within the time frame (6–12 hours post primary infection) of SAR signal generation [15••,16•,17•,18•]. The precise timing of MeSA movement is not known since its systemic translocation was implied based on grafting experiments [19•]. Moreover, MeSA is volatile,

Figure 1



Model depicting the events underlying SAR activation based on current findings. Pathogen infection induces the release of free carbon 18 fatty acids (C18 FA) from membrane lipids, these results in azelaic acid (AzA) production, which in turn induces glycerol-3-phosphate (G3P) biosynthesis. G3P is derivatized to an unknown compound (G3P*), which together with DIR1 moves systemically to induce resistance. Pathogen infection also induces accumulation of the amino acid (Lysine, Lys) derivative pipecolic acid (Pip) and dihydroabetinal amine (DA), which function in SAR via salicylic acid (SA). DA and a portion of AzA also move systemically, where AzA is present as a derivatized product in the distal tissues. SA is converted to methyl SA (MeSA), which is presumed to move systemically, where it is converted back to SA. Systemic defense activation requires an intact cuticle, depicted by a transmission electron micrograph in the inset, Ct = cuticle, CW = cell wall. SA is perceived by its receptors and signaling components NPR1, 3 and 4, which activate downstream defense signaling via the TGA, WRKY and other transcription factors. SAR induces chromatin modifications, which prime systemic tissues for SA responses. SAR can also be transmitted to the next generation progeny.