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Review

Express yourself: Transcriptional regulation of plant innate immunity

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ABSTRACT

The plant immune system is a complex network of components that function together to sense the presence and activity of potential biotic threats, and integrate these signals into an appropriate output, namely the transcription of genes that activate an immune response that is commensurate with the perceived threat. Given the variety of biotic threats a plant must face the immune response must be plastic, but because an immune response is costly to the plant in terms of energy expenditure and development it must also be under tight control. To meet these needs transcriptional control is exercised at multiple levels. In this article we will review some of the latest developments in understanding how the plant immune response is regulated at the level of transcription. New roles are being discovered for the long-studied WRKY and TGA transcription factor families, while additional critical defense functions are being attributed to TCPs and other transcription factors. Dynamically controlling access to DNA through post-translational modification of histones is emerging as an essential component of priming, maintaining, attenuating, and repressing transcription in response to biotic stress. Unsurprisingly, the plant's transcriptional response is targeted by pathogen effectors, and in turn resistance proteins stand guard over and participate in transcriptional regulation. Together, these multiple layers lead to the observed complexity of the plant transcriptional immune response, with different transcription factors or chromatin components playing a prominent role depending on the plant-pathogen interaction being studied.

Contents

Abbreviations: TF, transcription factor; HR, hypersensitive response; NB-LRR, nucleotide-binding leucine-rich repeat; TIR, toll/interleukin-1 receptor; CC, coiled coil; ChIP, chromatin immunoprecipitation; M/PAMP, microbe/pathogen-associated molecular pattern; PTI, PAMP-triggered immunity; ETI, effector-triggered immunity; HAT, histone acetyltransferase; HDAC, histone deacetylase; HKMT, histone lysine methyltransferase; HKDM, histone lysine demethylase; SA, salicylic acid; JA, jasmonic acid; ET, ethylene.

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1. Introduction

Plants are regularly exposed to agents of biotic stress, both in their natural environments and in agricultural settings. These pests and pathogens include viruses and representatives from virtually every kingdom of life, including parasitic plants [1]. In order to defend themselves against pathogens, plants have evolved a tightly regulated and multifaceted innate immune system. As an initial layer of defense, plasma membrane-associated extracellular receptors are utilized to recognize the presence of 'non-self' factors such as conserved molecular signals called microbe or pathogen-associated molecular patterns (M/PAMPS) [2]. PAMP recognition induces a signal transduction cascade to activate components of the plant innate immune system, ultimately affecting plant physiology with the goal of minimizing pathogen virulence [3]. While these defenses may be sufficient to protect against the vast majority of potential invaders, some microbial pathogens are not so easily inhibited. As part of an ongoing evolutionary arms race, some pathogens have developed mechanisms to suppress PAMP-triggered immunity (PTI) via the activity of effector proteins secreted into the host cell. Though deployment of effector proteins largely results in promoting virulence by suppression of PTI or influencing host physiology, detection of an effector by one or more resistance proteins will alert the host cell to the pathogen's presence. Direct or indirect recognition of effectors by resistance proteins activates a much stronger defense response called effector-triggered immunity (ETI), which may include localized cell death known as the hypersensitive response (HR) in order to inhibit the spread of pathogens [4,5]. Between the molecular components of PTI and ETI, plants are genetically equipped with an effective suite of tools to combat infection.

Strictly speaking, this description of a two-tiered plant innate immune system is most relevant for plant interactions with biotrophic microbial pathogens. Viruses for example require a living host cell and can therefore be classified as biotrophic pathogens, but do not possess PAMPs in the original sense. Plants respond to viruses with ETI and a specialized viral defense involving RNA interference [6]. PAMP or damage-triggered immunity is relevant for combatting microbial necrotrophs, but ETI can be manipulated by necrotrophs to hasten host cell death for the benefit of the pathogen [7,8]. Instead, a combination of PTI and response to wounding, host-specific toxins and oxidative stresses appears to characterize plant responses to necrotrophs [8]. For more information on these general features of the plant innate immune system we refer to reviews that summarize this large body of work [1–4,6,8–11].

Regardless of the nature of the biotic stress, plants must be able to induce broad changes in transcriptional profiles in a temporal and environmentally contextual manner to effectively defend themselves [12]. In the case of biotrophic microbial pathogens, extensive overlap but an accelerated response during ETI was observed when gene expression changes occurring during PTI and ETI were compared [13,14]. This overlap highlights the central importance of transcriptional reprogramming as a core immune response, and suggests that activated resistance proteins feed into an underlying defense network to accelerate responses [15,16]. Intermingled with the plant defense response are gene expression changes induced by pathogen effectors to promote virulence [17,18]. It is becoming apparent that the pathogen response machinery, including the regulators of transcriptional responses, constitutes a highly interconnected and complex network [19], with different regulators identified as important or rate-limiting depending on which plant-pathogen system is studied.

In this review, we provide an overview over transcriptional modules that have a prominent role in plant-pathogen interactions, with an emphasis on responses to biotrophic microbial pathogens (Fig. 1). As with other facets of the plant immune system, principles derived with this class of pathogens are likely to be relevant to a general view of plant responses to biotic stress [11].

2. Primary transcription factors regulating innate immunity

Perception of pathogen-associated stimuli triggers both immediate and prolonged activation and repression of genes to tailor an appropriate immune response to the pathogen(s) in question. The massive transcriptional changes associated with PTI and ETI are accomplished through interacting networks of transcriptional regulators from multiple protein families [20]. The *Arabidopsis thaliana* genome is thought to contain over 1600 transcription factor (TF) genes (6–10% of predicted total gene number), a large number relative to other eukaryotes [21]. Sessile organisms like plants may utilize a diverse array of regulatory proteins to fine-tune physiological responses to changing environmental conditions. It is increasingly apparent that these activities are regulated additionally through post-translational modification as a mechanism to modulate a flexible plant defense response in an efficient and precise manner [22]. In recent years, the roles of several key families of TFs in the plant innate immune response have begun to be elucidated, as well as the molecular mechanisms behind their regulatory activities.

2.1. TGA/bZIP family

Proteins of the basic leucine-zipper subfamily TGA were among the first TFs identified as plant defense regulators, originally studied for their binding affinity for the activating sequence-1 element (*as*-1) [23]. *as*-1 was also identified in the promoter of pathogenesis-related marker gene *PR1* [24], expression of which is enhanced during systemic acquired resistance (SAR), a lasting defense mechanism induced by mobile hormonal signals such as salicylic acid (SA) in uninfected tissue to prevent secondary infection [25]. Accordingly, TGAs have largely been described as positive regulators of SA-mediated gene expression and defense against biotrophic pathogens, although regulatory roles in other defense signaling pathways are now becoming apparent. In the *Arabidopsis* genome, 10 members of the TGA TF family have been identified and grouped according to sequence homology, 6 of which are implicated in defense signaling by mutant analysis. The class I TGAs TGA1 and TGA4 are positive regulators of basal resistance to the virulent bacterial pathogen *Pseudomonas syringae* pv. *maculicola*. Neither SAR nor resistance to an avirulent oomycete was compromised in the *tga1 tga4* double mutant, and this function was independent