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Phytocytokines and its Role in Plant Immunity

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SUMMARY

Plant plasma membrane-resident immune receptors regulate plant immunity by recognizing microbe-associated molecular patterns (MAMPs), damage-associated molecular patterns (DAMPs) and phytocytokines. Phytocytokines are plant endogenous peptides, which are usually produced in the cytosol and released into the apoplast when plant encounters pathogen infections. Phytocytokines regulate plant immunity through activating an overlapping signaling pathway with MAMPs/DAMPs with some unique features.

INTRODUCTION

Phytocytokines is that they are perceived by specific cell surface receptors. Phytocytokines are usually perceived by cell surface-resident receptor-like kinases (RLKs), which contain an extracellular domain, a transmembrane region, and a cytoplasmic kinase domain resembling the animal receptor tyrosine kinases. Phytocytokines are usually derived from precursor proteins with the following characteristics: an amino (N)-terminal signal peptide (only for secreted peptides), a carboxyl (C)-terminal region conserved in the same family of peptides, and a variable region (also named prodomain) between signal peptide and conserved region (Fig. 1).

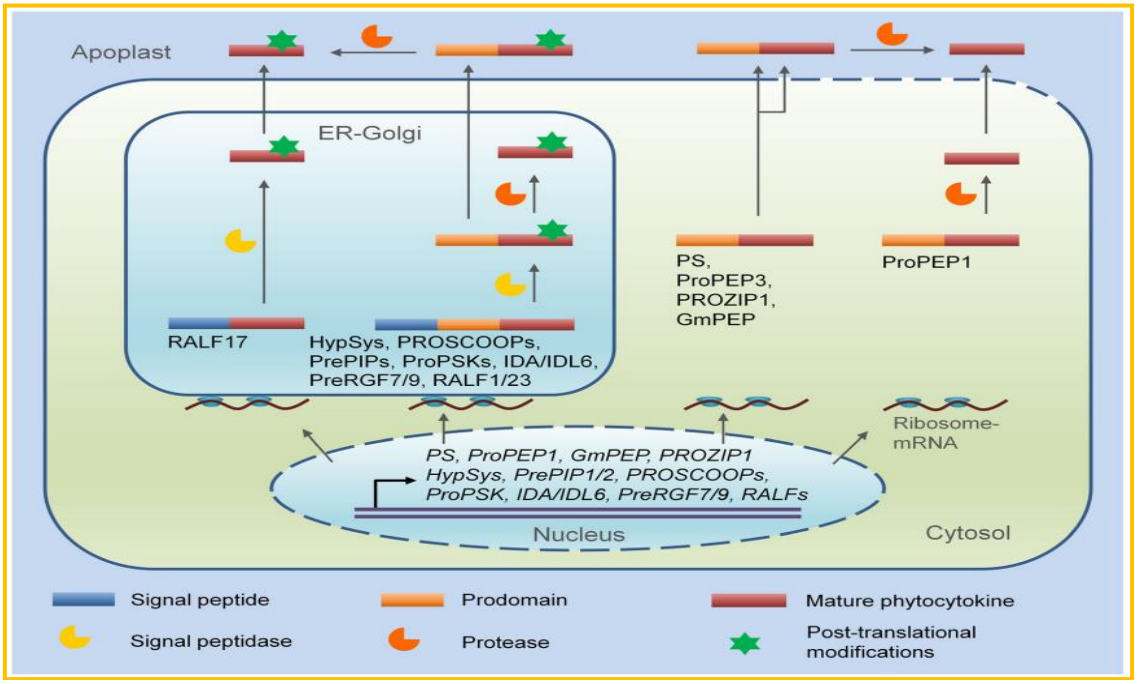

Fig. 1: Regulation of phytocytokine maturation and release

Once translated, the phytocytokine precursors enter the secretory pathway with the guide of signal peptide and are finally secreted into the extracellular compartment (apoplast) of plant cells as biologically active, mature phytocytokines. In the secretory pathway of endoplasmic reticulum (ER) and Golgi or the apoplast, proteolytic cleavages of signal peptide and prodomain, and post-translational modifications, such as tyrosine sulfation, proline hydroxylation, hydroxyprolinearabinosylation, and intramolecular disulfide bond formation are required for the phytocytokine maturation(Olsson *etal.*, 2019).

For the phytocytokine precursors without a signal peptide (non-secreted peptides), they do not enter the canonical ER-Golgi secretory pathway and undergo post-translational modifications and are proposed to be released into the extracellular compartment via an unconventional secretory pathway or during cellular damage. The processing of the phytocytokine precursors to remove their prodomains that happens in the cytosol or the apoplast is also essential for their maturation (Fig. 1).

Phytocytokine-mediated regulation of plant immunity:

Upon perception by cognate PRRs, MAMPs trigger convergent PTI responses, including phosphorylation of the receptor-like cytoplasmic kinases (RLCKs), the elevation of cytosolic Ca^{2+} concentration, transient apoplastic ROS burst, the activation of mitogen-activated protein kinases (MAPKs) and calcium-dependent protein kinases (CDPKs), reprogramming of defense gene expression, callose deposition, production of immune-related hormones and antimicrobial components, and plant growth inhibition. Like MAMPs, some phytocytokines also activate canonical PTI responses (Fig. 2). For example, Pep1, PIP1, and SCOOP12 all trigger MAPK activation, ROS production, callose deposition, and induce the expression of some PTI marker genes. Since the expression of these phytocytokines and cognate receptors are induced by MAMPs, these phytocytokines were thought to amplify MAMP responses (Hou*et al*., 2021).

Pathogen infections swiftly activate or inhibit the expression of phytocytokine precursor genes, or promote phytocytokine maturation. When released to the apoplast, phytocytokines are perceived by their corresponding receptor- and co-receptors. These perceptions activate ROS burst, Ca^{2+} influx, and phosphorylation of MAP kinase kinase kinases (MAPKKKs) mediated by BIK1 and/or related RLCKs. Ca^{2+} may promote phytocytokine maturation. Activated MAPKs may phosphorylate transcription factors (TFs), which further up- or down-regulate the expression of phytocytokine precursor genes and PRR genes, and SA- and/or JA-responsive genes, thus amplifying or attenuating immunity. Phytocytokine signaling may also modulate PRR complex stability and assembly. For example, the complex formation between FLS2 and RGI induced by flg22 increases FLS2 abundance, and the association between FER, FLS2, and BAK1 is promoted or inhibited by RALF17 or RALF23, thus positively or negatively regulates PTI, respectively. Due to limited space, only several of well-studied phytocytokines and their receptors are shown in this Fig. 2(Stegmann*et al*., 2017).

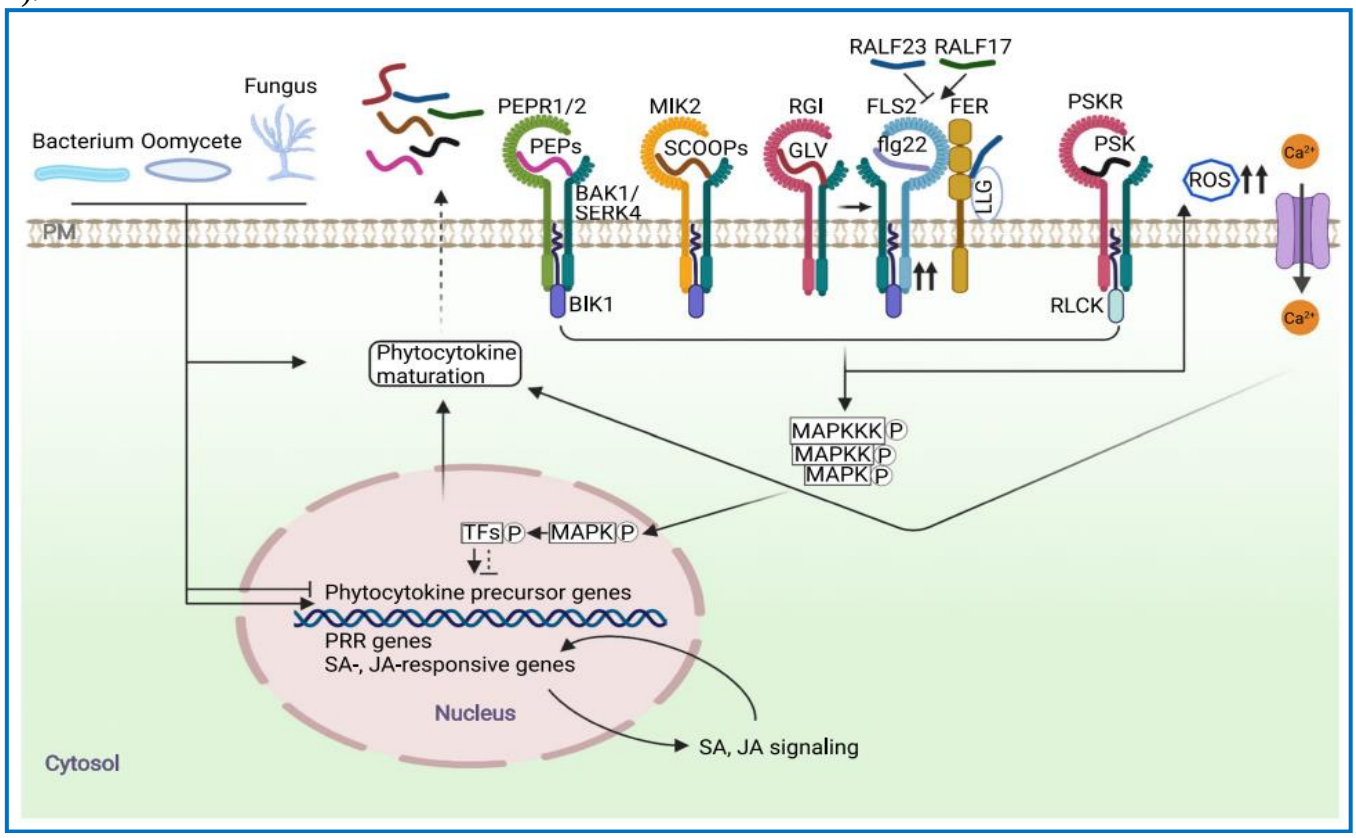

Fig. 2:A model of phytocytokine-mediated regulation of plant immunity

CONCLUSION

Some peptide hormones initially identified as regulators of plant development, reproduction or abiotic stress response have been shown to be involved in plant immunity. Similarly, some immunological peptides also play roles in other physiological processes. Plant endogenous peptide signaling are involved in the regulation of plant immunity and these immunomodulatory peptides were recently defined as phytocytokines. Thus,phytocytokines have dual roles in plant immunity, development, growth, reproduction or stress adaptation, similar to cytokines in animal physiology.

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