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GDSL lipase 1 regulates ethylene signaling and ethylene-associated systemic immunity in *Arabidopsis*

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ABSTRACT

Arabidopsis GDSL lipase 1 (GLIP1) has been shown to modulate systemic immunity through the regulation of ethylene signaling components. Here we demonstrate that the constitutive triple response mutant *ctr1-1* requires GLIP1 for the ethylene response, gene expression, and pathogen resistance. The *glip1-1* mutant was defective in induced resistance following primary inoculation of necrotrophic pathogens, whereas *GLIP1*-overexpressing plants showed resistance to multiple pathogens. Necrotrophic infection triggered the downregulation of EIN3 and the activation of *ERF1* and *SID2* in a GLIP1-dependent manner. These results suggest that GLIP1 positively and negatively regulates ethylene signaling, resulting in an ethylene-associated, necrotroph-induced immune response.

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1. Introduction

Plants utilize multilayered immune systems to counteract pathogen attacks [1]. The basal resistance to pathogens is triggered by the recognition of pathogen-associated molecular patterns (PAMPs) by cell surface receptors, a mechanism referred to as PAMP-triggered immunity (PTI) [2]. Some pathogens successfully combat PTI by directly secreting effectors into the cytosol of host cells. Plants have further evolved effector-triggered immunity (ETI), wherein plant resistance (R) proteins specifically recognize pathogen effectors [3]. These local immune responses lead to a broad-spectrum resistance, designated as systemic resistance, in yet uninfected tissues. Systemic acquired resistance (SAR) and induced systemic resistance (ISR) are two main types of systemic immune responses [4–8]. In general, plants utilize various defense mechanisms, depending on the lifestyle of pathogens [6,9,10]. SAR is associated with the accumulation of salicylic acid (SA) and resistance to biotrophic pathogens, whereas ISR requires jasmonic acid (JA) and ethylene signaling, and confers resistance to necrotrophic pathogens [9–13].

The signal transduction pathway of one of the defense hormones, ethylene, has been elucidated using genetic and molecular analyses [14,15]. The effect of ethylene on dark-grown seedlings, the so-called triple response, is characterized by the inhibition of hypocotyl and root growth and exaggerated curvature of the apical hook. Ethylene signaling components have been isolated based on the altered triple response phenotypes of corresponding mutant seedlings [14–16]. In the ethylene response pathway, ethylene is recognized by His kinase receptors such as ETHYLENE RECEPTOR 1 (ETR1) [17]. In the absence of ethylene, ethylene receptors activate a Raf-like serine/threonine kinase CONSTITUTIVE TRIPLE RESPONSE 1 (CTR1) that functions as a repressor of ethylene responses [18]. ETHYLENE INSENSITIVE 2 (EIN2) and EIN3 are positive regulators of ethylene responses and act downstream of CTR1 [19]. In the absence of ethylene, two classes of F-box proteins, EIN2 TARGETING PROTEIN 1 (ETP1)/ETP2 and EIN3-BINDING F-BOX PROTEIN 1 (EBF1)/EBF2, target EIN2 and EIN3 for proteosomal degradation, respectively [20–23]. Upon ethylene perception, CTR1 is repressed and EIN2 is subjected to proteolytic cleavage [24]. The resulting carboxyl-terminal fragment of EIN2 is translocated to the nucleus, which results in the stabilization of EIN3 and consequently triggers ethylene responses. Transcription factors EIN3

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저널명	저널명약어	ISSN	저널분류	IF	IF 5년	ES	분야	전체상위%	분야IF상위%	분야 등수	분야IF5상위%	SJR
FEBS LETTERS	FEBS LETT	0014-5793	SCI;SCIE;SCOPUS;PUBMED	3.263	3.26	0.15154	BIOCHEMISTRY & MOLECULAR BIOLOGY	14.7	36.7	101/275	35.2	1.695
FEBS LETTERS	FEBS LETT	0014-5793	SCI;SCIE;SCOPUS;PUBMED	3.263	3.26	0.15154	BIOPHYSICS	14.7	25.3	18/71	30.9	1.695
FEBS LETTERS	FEBS LETT	0014-5793	SCI;SCIE;SCOPUS;PUBMED	3.263	3.26	0.15154	CELL BIOLOGY	14.7	48.4	76/157	46.4	1.695
2009년도의 저널명이 FEBS letters인 저널의 등급은 M1급입니다. 바로 위 표를 참조하세요.												

저널명	저널명약어	ISSN	저널분류	IF	IF 5년	ES	분야	전체상위%	분야IF상위%	분야 등수	분야IF5상위%	SJR
FEBS LETTERS	FEBS LETT	0014-5793	SCI;SCIE;SCOPUS;PUBMED	3.54	3.319	0.13125	BIOCHEMISTRY & MOLECULAR BIOLOGY	12.4	32.8	93/283	34.6	1.691
FEBS LETTERS	FEBS LETT	0014-5793	SCI;SCIE;SCOPUS;PUBMED	3.54	3.319	0.13125	BIOPHYSICS	12.4	27	20/74	33.7	1.691
FEBS LETTERS	FEBS LETT	0014-5793	SCI;SCIE;SCOPUS;PUBMED	3.54	3.319	0.13125	CELL BIOLOGY	12.4	46.2	75/162	48.7	1.691
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FEBS LETTERS	FEBS LETT	0014-5793	SCI;SCIE;SCOPUS;PUBMED	3.601	3.398	0.11102	BIOCHEMISTRY & MOLECULAR BIOLOGY	12.4	35.6	102/286	36.7	1.752
FEBS LETTERS	FEBS LETT	0014-5793	SCI;SCIE;SCOPUS;PUBMED	3.601	3.398	0.11102	BIOPHYSICS	12.4	32.8	24/73	34.2	1.752
FEBS LETTERS	FEBS LETT	0014-5793	SCI;SCIE;SCOPUS;PUBMED	3.601	3.398	0.11102	CELL BIOLOGY	12.4	44.9	80/178	48.8	1.752
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FEBS LETTERS	FEBS LETT	0014-5793	SCI;SCIE;SCOPUS;PUBMED	3.538	3.329	0.09533	BIOCHEMISTRY & MOLECULAR BIOLOGY	12.8	35.1	102/290	37.5	1.802

FEBS LETTERS	FEBS LETT	0014-5793	SCI;SCIE;SCOPUS;PUBMED	3.538	3.329	0.09533	BIOPHYSICS	12.8	28.3	21/74	35.1	1.802
FEBS LETTERS	FEBS LETT	0014-5793	SCI;SCIE;SCOPUS;PUBMED	3.538	3.329	0.09533	CELL BIOLOGY	12.8	45.3	82/181	49.7	1.802
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FEBS LETTERS	FEBS LETT	0014-5793	SCI;SCIE;SCOPUS;PUBMED	3.582	3.477	0.08376	BIOCHEMISTRY & MOLECULAR BIOLOGY	12.6	33.4	97/290	36.2	1.848
FEBS LETTERS	FEBS LETT	0014-5793	SCI;SCIE;SCOPUS;PUBMED	3.582	3.477	0.08376	BIOPHYSICS	12.6	25	18/72	34.7	1.848
FEBS LETTERS	FEBS LETT	0014-5793	SCI;SCIE;SCOPUS;PUBMED	3.582	3.477	0.08376	CELL BIOLOGY	12.6	43.2	80/185	47.5	1.848
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