[Grant-in-Aid for Scientific Research (S)]

Molecular basis of host adaptation and enhanced virulence in plant pathogens and new disease resistance technologies

CONTRACT OF STREET	Principal Investigator	Kyoto University, Graduate School of Agriculture, ProfessorTAKANO YoshitakaResearcher Number : 80293918	
	Project Information	Project Number : 25H00431 Keywords : plant pathogen, effector, host	Project Period (FY) : 2025-2029

Purpose and Background of the Research

• Outline of the Research

Most damage in plant diseases is caused by plant pathogenic fungi (hereafter referred to as plant pathogens). It is plausible that virulence-related secreted proteins, called effectors, play crucial roles in host infection by many plant pathogens, but its molecular basis is still unknown. In addition, plant pathogens often appear as a hypervirulent form, but the molecular background for this aspect is also unknown. In this study, we will elucidate the mechanism of host specificity establishment of plant pathogens via effectors, starting from the analysis of EPC effectors that are necessary for host infection in *Colletotrichum orbiculare*. We will analyze the function and structure of each EPC effector, identify and analyze the target plant molecules, and analyze the structure of the complex between the effector and the target plant molecule. Furthermore, by conducting multi-omics and molecular genetic analyses of hypervirulent and normal virulent pathogens, we will elucidate the molecular mechanism of enhanced virulence in plant pathogen (Fig. 1). Based on the findings, we will attempt to develop a novel disease resistance technology that disables the function of critical pathogen effectors using gene editing technology (Fig. 1).



Expected Research Achievements

Starting from the discovery of the EPC effector, this research aims to elucidate the mechanism by which plant pathogen effectors establish host specificity. Furthermore, we aim to elucidate the mechanism for enhanced virulence in plant pathogens, and to pioneer the dynamism of pathogens from host adaptation to enhanced virulence. To achieve this goal, we will clarify the details of the plant immune suppression ability of the EPC effectors and its localization in plant cells (Fig. 2). We will also determine the three-dimensional structure of the EPC effectors and identify the plant factors that are targeted by the effectors (Figs. 3 and 4). Furthermore, by detailed comparative analysis of normal and hypervirulent strains, we will identify the pathogen factors that cause enhanced virulence (Fig. 5). The knowledge gained from this research will also pave the way for the development of new disease resistance technologies that disable the effector function. Disease resistance achieved by conventional resistance genes is often overcome by pathogen mutations within about 10 years. In this study, we will create a modified target protein that is resistant to EPC effectors (Fig. 6). To achieve this, we will determine the complex structure of the effector and the target protein, and then perform mutation analyses based on the revealed structure to identify mutations that can change the target to be a resistant type. We will then introduce this mutation into a cucurbit crop using the versatile genome editing technology called iPB method, and attempt to create a cucurbit crop that is resistant to cucurbit anthracnose (Fig. 7). It is assumed that this effector-resistant disease resistance will be almost unbreakable within about 10 years. If we are successful in creating such disease-resistant cucurbit crops, it is expected that this technology will be applicable to other diseases and other crops.

EPC3-GFP BIP5-mCherry Merged

ER-localized BIP5 is a target of EPC3 Figure 2. Localization analysis of EPC3



https://www.plant-pathology.kais.kyoto-u.ac.jp/en Homepage Address, etc.

Activation

of immunity



Figure 3. NMR-based structural analysis of a pathogen effector



Figure 5. Virulent and hypervirulent strains

