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Mini Review

Image-based Analysis to Study Plant Infection with Human Pathogens

Marek Schikora ^a, Adam Schikora ^{b,*}

^a Fraunhofer Institute for Communication, Information Processing and Ergonomics FKIE, Fraunhoferstrasse 20, 53343 Wachtberg, Germany

^b Institute for Phytopathology and Applied Zoology, IFZ, JLU Giessen, 35392 Giessen, Germany

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ABSTRACT

Our growing awareness that contaminated plants, fresh fruits and vegetables are responsible for a significant proportion of food poisoning with pathogenic microorganisms indorses the demand to understand the interactions between plants and human pathogens. Today we understand that those pathogens do not merely survive on or within plants, they actively infect plant organisms by suppressing their immune system. Studies on the infection process and disease development used mainly physiological, genetic, and molecular approaches, and image-based analysis provides yet another method for this toolbox. Employed as an observational tool, it bears the potential for objective and high throughput approaches, and together with other methods it will be very likely a part of data fusion approaches in the near future.

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1. Phytopathometry in Use

Identification and quantification of plant diseases are required for the adequate plant protection, the determination of crop losses, and the design of breeding strategies in agriculture [1]. The use of images in disease control and survey has a long tradition. Already 90 years ago aerial pictures made from airplanes were used to study crop diseases on fields in the USA [2,3]. Since then image-based detection of disease symptoms constantly improved. Today, not only detection, but also very sophisticated and informative analysis is possible. Those can, and use, the full spectra of electro-magnetic radiation. However,

the vast majority uses images based on the UV, visual and infrared spectra. Image-based analysis is also a powerful tool in studies of plant physiology, especially the responses to pathogen attack at the organism or tissue levels.

The different aspects of image-based detection and measurement of disease symptoms in plants are under constant development and were reviewed in several recent publications [1,4,5]. In 1966, E. C. Large introduced the general term phytopathometry to describe the quantification of plant disease [6]. Few decades later, Nutter and coworkers together with the American Phytopathological Society defined several other terms related to measurable symptoms of plant diseases [7,8]. Among them they defined: “disease severity” as the proportion or percentage of sample unit (fruit, plant or field) showing the symptoms. “Disease incidence” as the proportion of individual plants or plant organs within

* Corresponding author.
E-mail address: adam.schikora@agrar.uni-giessen.de (A. Schikora).

the total number of assessed individuals and “disease prevalence” as the proportion of fields, areas or countries in which the disease was detected. Also the term “disease intensity” related to the amount of disease in the host population was introduced. Hence, many studies concentrate on disease severity, describing the distribution of symptoms caused by pathogens on plant organs (leaves, stems, roots, etc.) or in plant populations at field, forest or grassland scale.

The visible symptoms, observed on plants and caused by the propagation of a pathogen might be based on different physiological phenomena. For instance, fruit or leaf soft rot diseases caused by diverse bacteria from the *Erwinia*, *Pseudomonas*, *Bacillus*, or *Clostridium* groups [9], are the result of disintegration of plant tissues by bacterial enzymes. Those, the enzymes, are secreted to the surroundings and cause destruction of the middle lamella followed by maceration of cell walls and the cellular content. Many fungal pathogens exhibiting necrotrophic lifestyle (*Botrytis* spp., *Alternaria* spp., or *Rhizoctonia* spp.) also rely on an active degradation of host tissues, causing in consequence, well visible disease symptoms [10]. Such symptoms are often referred to as necrosis. On the other hand, biotrophic or hemi-biotrophic pathogens may trigger intense activation of defense mechanism known as hypersensitive response (HR). HR occurs within few hours or days after inoculation and results in localized cell death. Very often HR is the consequence of the so-called effector-triggered immunity (ETI), which occurs when the plant recognizes the effector proteins injected by the pathogen into the plant cell [11]. The function of this rapid cell death, or HR, is to counteract the systemic spreading of the pathogen. Although both necrosis and HR originate from different mechanisms, their result is a change of leaf or other tissue appearances. Those morphological differences can be easily visualized using the visible part of the electromagnetic spectrum by analog or digital photography [1]. Necrosis and HR are however, not the only possible outcome of a pathogen attack. Upon recognition of a pathogen plants may close their stomata and therefore restrict the access to mesophyll tissue [12–14]. Because of the physiological functions of stomata, which are gas exchange and the control of inner surface evaporation, stomatal closure results in an increase of leaf temperature [15]. Those differences can be assessed using for example infrared imaging. In the same manner, pathogens affecting plant metabolism can influence the content of plants chlorophyll and other pigments, which in turn changes the plants' autofluorescence and can be visualized using the near-UV spectrum imaging. Taking together, plant physiology and their reactions to pathogen attack offer multiple possibilities for an image-based assessment of changes and hence the detection and measurement of disease symptoms.

2. Plants as Source for Human Pathogens

Numerous pathogenic bacteria seem to have a fairly broad spectrum of host organisms. Among these, *Salmonella* spp., *Pseudomonas* spp., *Klebsiella* spp., *Escherichia coli* EHEC, and others efficiently proliferate in animal and plant organisms [16–18]. *Salmonella enterica* is one of the main causes of food-borne poisonings today. Salmonellosis is unfortunately a constant threat to human health not only in developing but also in developed countries. A large study conducted in 2007 showed that in the UK, the Netherlands, Germany and Ireland 0.1 to 2.3% of pre-cut products were contaminated with *Salmonella* bacteria [19]. Another European study from 2009 revealed that 2.5% of fresh produce were contaminated with *Salmonella* [19]. In the USA, one out of six citizens is estimated to infect himself by eating contaminated food [20]. *Salmonella* infections have not declined in the last 15 years, making the non-typhoidal strains the leading cause of food poisoning. In cases related to domestic food poisoning in the USA, salmonellosis was responsible for 35% of the hospitalizations and 28% of deaths [21]. Poultry and eggs are commonly associated with *Salmonella* outbreaks; however, 20% of infections from 2004 to 2008 were linked to other sources including: sprouts, leafy greens, roots, grain-beans, fruits

and nuts [20]. The assumption that *Salmonella* passively survives on plants after occasional contaminations changed in the last few years. Research on the interaction between plants and these bacteria suggests an active infection process [22–31].

In order to deploy the host immune system *S. enterica* uses diverse effector proteins, those proteins interact with the host immune system and inhibit or abolish its action. Effectors are usually injected into host cytoplasm by Type III Secretion Systems (T3SSs), those secretion apparatuses function as molecular needle and allow the translocation of bacterial proteins (e.g. effectors) into the host cytoplasm [32]. *Salmonella* has two T3SSs, which secrete different yet overlapping sets of effector proteins that function at different stages of the infection. Giving the importance for human health, the suppression of the animal immune system by *Salmonella* is very intensely studied. We know already 44 effectors, which are injected into animal host cells, and for many of them we know the function and the target proteins [33]. Interestingly, bacterial effectors often target signaling cascades, which are important regulators of the immune response in animals and plants. For instance, the SpvC effector from *Salmonella* spp. encodes a phosphothreonine lyase that dephosphorylates and therefore deactivates the ERK1/2 kinases, key regulators of animal immune system [34–36]. Another effector protein, the integral membrane protein SseF [37] together with SseG, is responsible for the formation of *Salmonella*-induced filaments, an elongated tubular structure within which the bacteria reside in animal cells. In plant cells, SseF is recognized and triggers the above-discussed HR [38].

Although several *Salmonella* effectors have homologues in phytopathogenic bacteria: e.g.: HopAI1 is a homologue of SpvC in *Pseudomonas* spp. [39] and HopAO1 is a functional homologue of SptP [40], the function of *Salmonella* proteins during the inactivation of the plant immune system remains elusive. Nonetheless, it is very tempting to speculate that biochemical features of those effectors are conserved between animal and plant hosts. This would provide *Salmonella* and other pathogenic bacteria with an efficient toolbox for suppression of plant immune system [18]. Such suppression was already reported. Recent study on the interaction between tobacco plants and *Salmonella* Typhimurium showed that in contrast to living bacteria, dead bacteria elicited an oxidative burst and pH changes in tobacco cells [31]. Similar response was provoked by the *invA* mutant, which lacks one of the T3SS [31]. Those results suggest that *Salmonella* depends on the secretion of effectors to actively suppress tobacco immune responses. Two transcriptome analyses performed after inoculation of *Arabidopsis* plants with the wild type *S. Typhimurium* strain 14028s and the *prgH*, a T3SS mutant, revealed a similar scenario [30,41]. The *prgH* mutant, similar to *invA* lacking one of the T3SS, induced the expression of more genes than the wild type bacteria, and the majority of which were related to defense responses, suggesting that the wild type bacteria are able to suppress the expression of a set of defense related genes. Moreover, mutants impaired in their T3SSs were less virulent towards *Arabidopsis* plants than wild type bacteria [30,42].

Taking together, recently published results indicate that *Salmonella* uses plants as alternative hosts and that these bacteria could, similarly to the infection in animals, actively suppresses the plant defense mechanisms. Whether these bacteria use the same or different effectors in order to achieve this goal is not yet clear, it seems however to be acceptable to conclude that *Salmonella* requires T3SSs during interaction with plants.

3. How to Use Image-Based Analysis to Study Infection with Human Pathogens

Visible symptoms caused by *Salmonella* on plant leaves depend on several mechanisms: i) The recognition of bacterial effectors, as in the case of SseF, which triggers the HR as a part of the ETI response [38]; ii) the suppression of ETI and therefore the HR as indicated by the inability to do so by mutants in T3SS [42,43]; iii) the serotype of the

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