‘Meltdown’ of Celery Research Update

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Michigan ranks second in the U.S. for celery production with nearly 2,000 acres planted generating about $19 million in revenue. Celery is primarily grown on muck soil and Michigan growers produce for both the fresh market and the processing market to be used in products such as soup or juice. Around 2018, growers began reporting a disease they termed ‘meltdown’ which was affecting the most widely grown cultivar, CR-1, and causing alarming losses.

Symptoms of ‘meltdown’ of Celery. Symptoms associated with ‘meltdown’ are generally consistent with Fusarium yellows, although growers report observations that they describe as unlike previous epidemics involving *Fusarium oxysporum* f. sp. *apii* race 2. Field symptoms are more spotty than previous outbreaks which tended to be more consistent and expansive. However, the characteristic symptoms of Fusarium yellows are present and include stunted plants, yellowing, wilting, and plant death (Fig. 1A). Yellowing and wilting do not typically appear until the disease is more advanced, making it seem rarely observed compared to previous epidemics (Fig. 1B). When symptomatic plants are cut open, the heart exhibits darkening in the vascular tissue and even complete rotting (Fig. 1C). One of the most characteristic symptoms of Fusarium yellows is browning of the vascular tissue in the petiole (Fig. 1D). Collectively, these symptoms are consistent with Fusarium yellows, even if they do not present exactly as they did in previous epidemics.

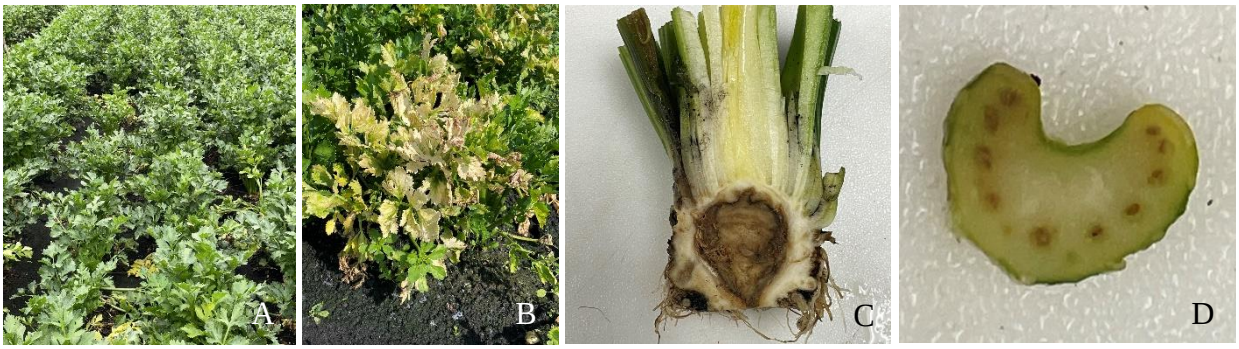

Figure 1. Symptoms of ‘meltdown’ of celery, aka. Fusarium yellows. A) Field level view of symptoms. B) Celery plant displaying characteristic yellowing of foliage. C) Internal rotting of the celery heart typical of advanced stages of the disease. D) Browning of the vascular tissue in a cross section of a celery stalk.

History of Fusarium Yellows. Fusarium yellows was first reported in 1914 in Michigan on the cultivar Golden Self-Blanching. The disease quickly spread to other growing regions in the U.S. and by the mid-1930s it had become a serious problem for celery producers throughout the country. The causal agent was determined to be *Fusarium oxysporum* f. sp. *apii* (*Foa*) and by the early 1950s resistant cultivars, specifically green types such as Tall Utah 52-70, were being used to manage the disease. There were few reports of Fusarium yellows until the late 1970s when a new strain of the pathogen was able to cause disease on the cultivars that were based on the Tall Utah 52-70 resistance. Once again, the disease spread across the country and threatened celery production. Strains of a pathogen that cause disease on different cultivars of a crop are referred to as ‘races’, where the original strains affecting Self-Blanching cultivars are referred to as ‘race 1’ and strains affecting Tall Utah cultivars are referred to as ‘race 2’.

In the mid-1980s, a major resistance gene was identified in celeriac and incorporated into commercial celery cultivars such as Challenger and Command. This resistance was the basis of Fusarium yellows management for the next 3 decades until new strains emerged to once again threaten celery production. In California, researchers identified a highly aggressive strain, known as *Foa* race 4, that caused severe disease on all modern celery cultivars, even those previously resistant to *Foa* race 2. In

Michigan, reports of 'meltdown' of celery prompted the Hausbeck lab to investigate the cause of this disease and to determine if the outbreak was related to Fusarium yellows and *Foa* race 4 from California.

Current Knowledge. We have examined hundreds of celery samples and have a collection of over 300 *F. oxysporum* isolates. Pathogenicity testing revealed several isolates that could cause disease on cultivar CR-1. Therefore, a subset of 95 *F. oxysporum* isolates was selected for expanded genetic evaluation using multiple genetic markers, including those previously published for the identification of race 2 and race 4. Fortunately, results indicate that there is no evidence of *Foa* race 4 in Michigan celery fields. Similarly, *Foa* race 2 is rare and represents just 3% of the isolates screened. However, we have identified a group of isolates that share genetic similarity with *Foa* race 1 and represent about 33% of the isolates screened (Fig 2). This group contains the strains that are currently causing disease on 'CR-1'.

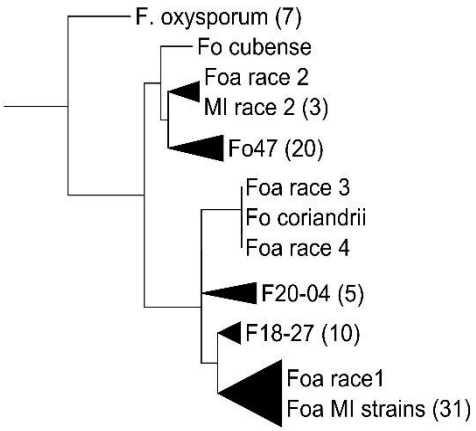

Figure 2. Genetic relationship of *Fusarium oxysporum* isolates recovered from Michigan compared to defined *Foa* races.

Despite the similarities between this group of Michigan strains (*Foa* MI) and *Foa* race 1, there are some important differences. First, *Foa* race 1 is not expected to cause disease on the cultivars Tall Utah or CR-1, but *Foa* MI strains can cause disease on both of those cultivars. Second, there is some genetic material in the *Foa* MI strains that is not present in the *Foa* race 1 isolates. These observations can be explained by the presence of a two-part genome that is common in *F. oxysporum* isolates.

This two-part genome of *F. oxysporum* consists of a set of core chromosomes that contain the genes necessary for essential functions such as growth, reproduction, and metabolism and another set of chromosomes known as accessory or dispensable chromosomes. These accessory chromosomes often contain host-specific genes that allow the fungus to exploit resources available in a specific host, such as a particular celery cultivar. Through a process that is not yet well understood, these accessory chromosomes can be gained or lost, and this can result in strains with a different response to different hosts. In addition, these accessory chromosomes contain large numbers of distinct genetic elements, called transposons, that can reorganize the genome through a cut-and-paste type process. In this way, it appears that strains of *Foa* race 1 have been surviving in the background, virtually unnoticed, until they were able to hit on a genetic combination that has allowed them to reemerge as a pathogen on celery cultivars that were previously resistant to Fusarium yellows.

Moving Forward. We continue to study the genetics involved in this group of isolates to identify the specific factors involved in loss of host resistance. There is much that is still unknown about the source of novel genetic material and how genomes evolve in this system, and we expect this research will provide valuable insight. Considering this outbreak would be the fourth time in the last century that host resistance was overcome in celery, it seems likely that any resistance identified now to manage this outbreak will be overcome again in the next couple of decades. A better understanding of how fungal genomes evolve may improve our ability to monitor, predict and respond to future outbreaks.

Fusarium yellows are notoriously challenging to manage as chemical controls are generally ineffective. Host resistance is therefore the most effective strategy for managing the disease. Now that we have a grasp on which strains are causing disease, we can begin more intensive screening of cultivars for resistance to these Michigan strains. Meanwhile, we will be testing some chemical treatments, primarily aimed at improving plant health, to provide growers with some support until resistant or tolerant cultivars can be identified.

Acknowledgments. This research was supported by funding from Project GREEEN, Celery Research Inc., and by a Michigan Specialty Crop Block Grant administered by Celery Research Inc.