EQUIPMENT/SUPPLIES & POTATO EXPO COVERAGE ISSUE

INTERVIEW: ZACH MYKISEN
Big Iron Equipment

A new Spudnik 6640 Harvester, available from Big Iron Equipment, is used to dig red potatoes.

YOUNG GROWERS
Spread Their Wings

BREEDING FOR LATE
Blight Resistance

ALL BETS WERE ON
At Potato Expo 2020

EXECUTIVE DIRECTOR
Gives Annual Report
Identifying disease-resistant genes is integral to improved potato production

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Disease management is an important and complex component of potato production.

Figure 1 shows the United States Department of Agriculture (USDA) statistics for fungicide use in Wisconsin, in 2016, which is the most recent year available.

The percent of acreage treated with different fungicides is shown by the gray bars on the left axis, and the average number of applications is shown via the blue line on the right axis.

Nearly the entire Wisconsin potato

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Above: Dr. Jeffrey Endelman provides updates on the Wisconsin potato variety development program for attendees of the 2019 Hancock Agricultural Research Station Field Day.

Right: Figure 1. USDA statistics indicate 2016 fungicide usage in Wisconsin.
crop was treated with chlorothalonil (e.g., Bravo, Echo, Initiate) to control late blight, and on average, nine applications were made.

Before fungicides were widely available, U.S. and Canadian potato production was more dependent on breeding to manage late blight and other diseases.

Through trial and error, breeders in the late 19th and early 20th centuries identified domesticated landraces and wild relatives of potato from Latin America that, upon cross-pollination with commercial varieties, produced resistant offspring.

By the 1950's, scientists began to identify different “R” genes based on how resistance to different pathogen isolates was inherited.

**GENE-FOR-GENE MODEL**
This pattern of disease resistance, known as the “gene-for-gene” model, has been observed in many different plant-pathogen systems.

![Resistant (Payette)](image1)

![Susceptible](image2)

**Figure 2.** Payette Russet (left) is highly resistant to the US-23 strain of late blight. This greenhouse experiment was conducted inside the Biotron facility at the University of Wisconsin-Madison.

The first set of potato late blight R genes, which were derived from the
Mexican species *Solanum demissum*, were simply named R1, R2, etc.

Eventually, a more informative naming scheme was developed in which both the pathogen and plant host are included.

For example, three late blight R genes have been identified in the wild relative *Solanum bulbocastanum*, which are named Rpi-blb1, Rpi-blb2 and Rpi-blb3, because “pi” are the initials of the pathogen (*Phytophthora infestans*) and “blb” is an abbreviation for the wild species.

In 2016, the DNA sequence of the R8 gene was published by a research group in the Netherlands who also showed that Missaukee and Jacqueline Lee—two resistant varieties from Michigan State University—contained R8.

University of Wisconsin-Madison researchers had already been using the two varieties for crossing to introduce resistance, and from the R8 sequence, we now had the possibility of developing a DNA marker to track R8 in breeding populations.

As part of a research project funded by the Wisconsin Potato & Vegetable Growers Association, we designed a marker and confirmed that it was a reliable predictor of R8.

When it came time to confirm the resistance phenotype, however, none of the clones with R8 had much resistance in our greenhouse assay against US-23, which is the main strain of late blight in Wisconsin and other states.

**A SILVER LINING**

Fortunately, there was a silver lining to this experiment. In 2015, the Pacific Northwest breeding program released a new fry processing variety called Payette, which was reported to have resistance to several strains of late blight.

We had an opportunity to observe Payette for several years in the National Fry Processing Trial and used it as a parent for crossing in 2015.

It was largely forgotten then until the R8 experiment in 2018, where it was used as a resistant control. Even as plants were dying all around it, Payette showed almost no signs of disease (Figure 2).

At the time, no one knew which R gene or genes were in Payette. To figure that out, in early 2019, we challenged nearly 100 offspring of Payette (crossed to a susceptible parent) with US-23.

We used a detached leaf assay, which uses less space and can be repeated more quickly than the whole plant assay shown in Figure 2.

There was a very clear difference between resistant and susceptible offspring (Figure 3), which were present in about equal numbers, indicating the presence of a single R gene that is designated “Rpi-pay.”

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Breeding for Late Blight Resistance...
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Each offspring was also genotyped using a microarray chip containing 21,000 DNA markers.

MEMBER OF THE FAMILY
When the marker data were combined with the resistance data, we discovered that Rpi-pay was located in the same genomic region as a family of previously studied R genes, and further research confirmed that Rpi-pay is a member of this family.

We also serendipitously discovered that one of the markers on the microarray is diagnostic for Rpi-pay, and this marker is being adapted for routine breeding use in 2020.

Just as pathogen populations can evolve resistance to fungicides when a single mode of action is used too heavily, a similar phenomenon can occur with plant R genes.

Good stewardship of R genes means that varieties with a single R gene should not be grown on large acreage without any chemical protection.

However, it should be possible to safely reduce the number of fungicide applications, and this topic needs additional research.

Another strategy to mitigate the risk of resistance breakdown is to focus on R genes that recognize essential pathogen genes.

For example, Rpi-blb1 (also known as RB) recognizes a gene present in every strain of *P. infestans* that we have tested.

GENE STACKING
Eventually, our goal is to stack multiple R genes in a single potato variety, each of which interacts with a different gene in the pathogen.

This makes it more difficult for the pathogen to evolve resistance (similar to using multiple fungicides with different modes of action).

DNA markers are essential to stacking R genes because the presence of a second or third R gene may not be possible to detect phenotypically.

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