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Zebra Chip: A New Disease of Potato

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Introduction

A new disease of potatoes called "zebra chip" (ZC), so named for the characteristic symptoms that develop in potato tubers from infected plants, has been documented by the research group at North Dakota State University (NDSU) to occur in commercial potato production fields. The disease was first documented in potato fields around Saltillo, Mexico, in 1994 (GA Secor, unpublished), and was first identified in the USA in the year 2000 in commercial potato fields in the Pearsall, TX, production area. Since that time ZC has spread to a number of other states, including NE, CO, KS, NM and CA. ZC was sporadically important economically until the 2004 and 2005 growing seasons when it caused millions of dollars in losses to both potato producers and processors in numerous locations. It also causes serious losses to the fresh market, tablestock and export potato industry, often causing the abandonment of an entire field.

ZC continues to be economically important in the fresh and processing potato producing areas of Mexico, particularly in the states of Coahuila and Nuevo Leon in northeastern Mexico, where it is called "papa manchada" (stained potato). In 2004, ZC incidence in this region was as high as 80% in some fields. This area borders the winter potato production area in TX where ZC was found in the USA. The disease is also serious in potato production areas of Guatemala, where it is named "papa rayada" (striped potato), and causes a serious problem of market potatoes, subsis-

Figure 1: Foliar symptoms of "zebra chip" in potato. Scorching of leaves of an individual plant (top) and an area in a field with collapsing plants that die early (bottom).

tence gardens, and processed potatoes. A complete survey to determine the geographical extent of the disease has not been conducted but there is anecdotal evidence that a disease of potato with similar symptoms exists in

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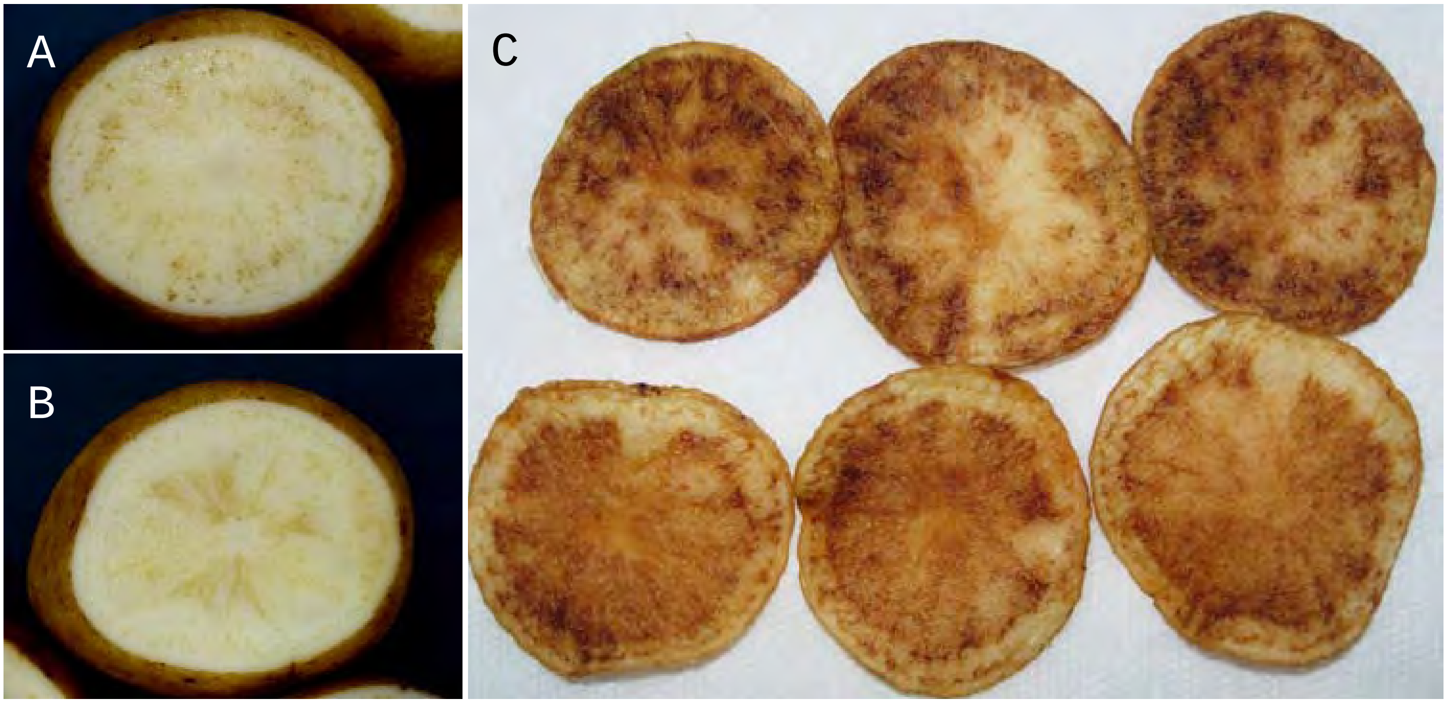

Figure 2: Potato tuber symptoms showing necrotic flecking (top left) and necrotic streaking of medullary ray tissue (bottom left) resulting in a "zebra chip" appearance and defect in the finished processed chip product (right).

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Eastern Europe and Southern Russia.

ZC-affected potato plants exhibit a range of foliar symptoms that resemble those of the purple top wilt syndrome caused by the clover proliferation phytoplasma recently found in WA and OR. Foliar symptoms include stunting, chlorosis, swollen nodes causing a "zig-zag" appearance of the upper growth, proliferated axillary buds, aerial tubers, browning of the vascular system in below ground portions of stems, and leaf scorching (Figure 1A). ZC-affected plants are usually scattered throughout the field. In some instances plants affected by ZC appear to coalesce in areas of the field with clusters of collapsing plants that die early (Figure 1B).

Below-ground plant symptoms include enlarged lenticels of the underground stem, collapsed stolons, and brown discoloration of the vascular ring and necrotic flecking of internal tuber tissues (Figure 2A) and occasionally streaking of the medullary ray tissues (Figure 2B). These symptoms are similar to, but distinct from, symptoms of net necrosis in potato tubers caused by the phloem-restricted potato leaf roll virus. These symptoms affect the entire tuber from the stem end to the bud end. It is these tuber symptoms that differentiate ZC from all previously described potato diseases. Potato chips made from tubers of affected plants have a severe dark brown streaking defect, hence the name 'zebra chip' (Figure 2C). This defect has become a major defect of processing potatoes produced in the areas where ZC occurs.

Tubers affected with ZC generally do not sprout, or if they do, produce hair sprouts or weak plants. In some instances, tubers with ZC symptoms produce nearly healthy appearing plants that produce progeny tubers with or without ZC symptoms. We do not know if this secondary 'seed' spread of ZC is due to a late infection by the pathogen or some other factor, but the role of seed borne infections of ZC needs to be investigated further. Symptoms of ZC have been observed in many cultivars, none appear to be resistant.

ZC Research

One of the first studies performed by the research group at NDSU was to determine if the cause

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of ZC was graft transmissible under glasshouse conditions. Apical tissue was removed from ZC affected plants and grafted onto healthy plants. Symptoms of ZC in tubers were evident within 28 days post-grafting (Figure 3). Graft transmission of the ZC phenomenon has been repeated many times over several years since those initial attempts, utilizing both primary and secondary infected plants arising from graft transmissions. We interpret these data to mean that the cause of ZC is a pathogen and not an abiotic cause such as heat stress (heat necrosis), psyllid toxin (psyllid yellows), nutrient deficiency or toxicity or a combination of these factors. We believe that these graft transmission studies clearly indicate that a systemic or vascular plant pathogen was involved, most likely a virus or prokaryote (bacterium or bacterial-like). However, we did not dismiss the possibility of a fungal pathogen as the cause of ZC.

The ZC tuber symptoms are characteristic of a phloem-restricted pathogen. The phloem of potato plants moves carbohydrates produced by the leaves down to the roots and tubers. When the phloem of potato plants is affected by a pathogen, carbohydrate metabolism in the tuber is affected, hence the darkening evident in the chips after cooking (Figure 2C). A number of potato pathogens are phloem-restricted, including potato leaf roll virus and all phytoplasmas.

Extensive testing ensued to determine the cause, source, and spread of ZC. A number of potential causes of ZC were initially investigated, including purple top phytoplasma, potato leaf roll virus, and beet western yellows virus. Subsequent to this work, laboratory testing of ZC plants has eliminated a number of other potential candidate pathogens including Ralstonia solanacearum, Serratia marcescens (the cause of cucurbit yellow vine disease), and many viruses including tomato spotted wilt, PVY strains, geminiviruses, tobacco rattle, beet western yellows, potato leaf roll and 19 other virus families.

Two pathogens appear to be associated with the zebra chip complex. The first is a new phytoplasma called the "stolbur-like" phytoplasma. Phytoplasmas are phloem-restricted organisms without cell walls that are transmitted primarily by leafhoppers during their entire life span. The stolbur-like phytoplasma was identified using PCR and other molecular analyses and determined to be related to, but distinct from, the stolbur phytoplasma, sharing 96.6% DNA sequences in common. The stolbur phytoplasma is prevalent in Eastern Europe, where it has caused significant crop losses, particularly in grape, tomatoes and potatoes. The stolbur-like phytoplasma in the U.S. is potentially very important to other crops. We do not yet know the vector of this new phytoplasma. The stolbur-like phytoplasma appears to be the primary cause of ZC in NE in 2004 and 2006. It is important to note, perhaps, that ZC has never been as economically damaging in NE as it has been in TX, which is illustrated by the fact that no ZC occurred in NE in 2005.

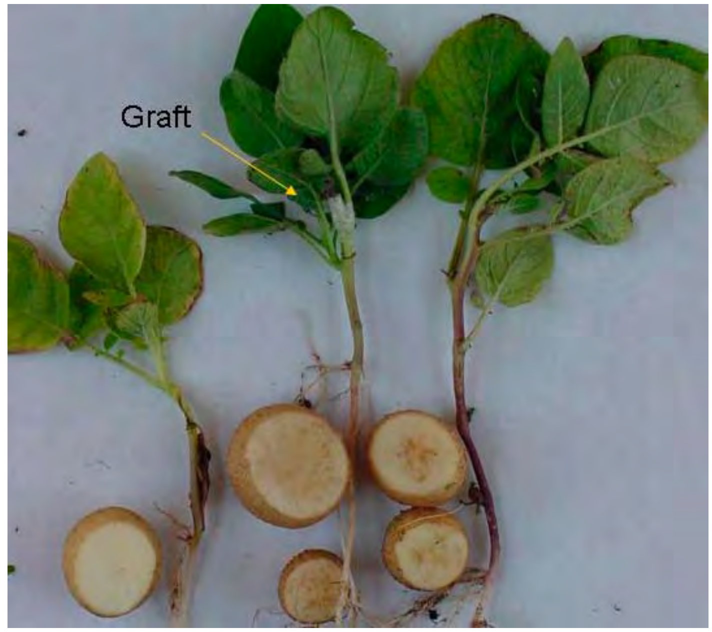

Figure 3: Graft transmission of zebra chip (ZC) pathogen into a healthy plant and concomitant development of ZC symptoms in progeny tubers.

The stolbur-like phytoplasma does not appear to be the primary cause of ZC in TX; however, approximately 5-10% of the plants with this disease test positive for this pathogen in the laboratory. Although the foliar symptoms of ZC in TX and NE are quite similar, the tuber symptoms can be quite different. ZC tuber symptoms in NE are more reminiscent of those depicted in Figure 2B while the ZC tuber symptoms in Figure 2A are more characteristic of the symptoms observed

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in TX. Furthermore, preliminary data suggests that the potato psyllid insect is the likely vector for the ZC agent in TX. This is important information. We hypothesize that the NE ZC agent may have a leafhopper vector and if so, it may permit this pathogen to spread into other potato production areas via movement of the insect. If the TX ZC agent is transmitted only by the potato psyllid, then this pathogen will likely be restricted to those areas with an environment conducive to psyllid reproduction and survival, which is generally in the Southern USA.

Summary and Future Directions

Based on our findings, and those of others, we believe that ZC is likely a disease complex involving at least two distinct pathogens and no less than two insect vectors. There are a number of plant diseases in which similar symptoms are caused by two distinctly different pathogens, such as "spraing" of potato in the United Kingdom and marginal necrosis of strawberry and 'bassess richessess' in sugar beet in France. So, while this type of disease complex is unusual, it is not unique to ZC.

Future studies on ZC will concentrate on determining the cause of ZC in Texas. We are currently investigating three potential pathogens as the cause, of all of which will represent newly described plants pathogens. Once the primary cause of TX ZC can be determined, we will be able to design laboratory diagnostic tests that will allow us to study the biology and the ecology of the pathogen in the field. We are in the process of doing this for the stolbur-like phytoplasma responsible for ZC in NE. Using molecular diagnostic tests, we hope to determine the insect vector or vectors responsible for transmitting this ZC agent and the potential of reservoir hosts of the pathogen being present in NE. We believe that ZC disease management will involve an integrated approach incorporating clean seed, insect and reservoir host management and varietal resistance.

The Nebraska Potato Eyes is on the World Wide Web at: www.panhandle.unl.edu/peyes.htm

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