Zebra chip and purple top: An overview on two emerging diseases of potato

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Abstract

Potatoes rich in vitamins, minerals, antioxidants were a life-saving food. But it’s yield suffers a huge loss due to disease-pest infestation. Emerging diseases pose significant threats to potato cultivation and its production. Two such diseases are zebra chip and purple top wilt of potato highlight the complexity of emerging disease challenges in potato production. Zebra chip, caused by the bacterium Candidatus Liberibacter solanacearum, leads to severe economic losses globally. It is transmitted by the potato psyllid insect. Infected plants develop unsightly dark streaks in their tubers when fried, rendering them unmarketable. Management relies on integrated pest management strategies, resistant cultivars, and pathogen surveillance. Purple top, caused by phytoplasma, is transmitted by the beet leafhopper. This disease causes discoloration and deformation of leaves, stunting plant growth and reducing yields. Infected potato tubers show purple discoloration and decreased market quality. Disease management encompasses vector control, crop rotation, and utilization of disease-free seed material. Early detection, vigilant monitoring, robust quarantine measures, and development of resistant varieties are crucial components of safeguarding potato crops from these threats. Additionally, continued research to understand the biology and epidemiology of these diseases is essential to formulate effective control strategies in the ever-evolving field of agriculture.

Keywords: Potato, emerging diseases, zebra chip, purple top

Introduction

Potatoes (Solanum tuberosum L.) have been a fundamental part of the global diet for centuries, with a total worldwide production of approximately 368.2 million tons in 2018. Among the top potato-producing nations, the United States ranks fifth, following China, India, Russia, and Ukraine (Faostat 2020) [9]. The potato industry in the United States is valued at approximately $3.5 billion (USDA, 2019; Faostat, 2020) [10, 11]. Approximately one-third of potatoes grown in the United States are designated for processing, with 63-83% allocated to frying, chipping, and various packaged products, while the remainder is distributed to the fresh market, livestock feed, or utilized as seed potatoes (USDA, 2019) [12]. The domestication of potatoes led to the development of cultivars with reduced glycoalkaloid content in their tubers, rendering them more palatable and resulting in larger tuber sizes, improved carbon fixation, and enhanced transport (Spooner et al. 2014; Machida-Hirano 2015) [13, 20]. Over nearly a century, a few resilient wild potato species were intentionally crossed with their cultivated counterparts to enhance resistance to diseases, increase yield, and improve overall quality (Jansky et al. 2013) [14]. These efforts yielded highly marketable advancements, including improved processing quality for chipping and frying, as well as resistance to certain viruses and nematodes (Douches et al. 1996; Hirsch et al. 2013) [8, 14]. However, the limited genetic diversity resulting from these breeding practices rendered potato crops vulnerable to pests and diseases, leading to significant inbreeding depression.

Emerging diseases of potato

Zebra chip disease: Zebra chip (ZC) disease was initially documented in Saltillo, Mexico in 1994, and later in South Texas, United States in 2000 (Munyaneza et al. 2007, 2009) [22-23].
The probable causal agent for this disease was identified as the finicky phloem-limited bacterium known as *Candidatus Liberibacter solanacearum* (CLso). The transmission of CLso to plants is facilitated by the potato-tomato psyllid *Bactericera cockerelli* Sulc (Munyaneza et al. 2007; Hansen et al. 2008; Liefding et al. 2009) [22, 13]. Plants affected by ZC disease exhibit various symptoms, including leaf chlorosis, discoloration, curling or upward rolling, aerial tubers, axillary bud proliferation, stunted growth, and, eventually, premature plant death. Potato tubers infected with CLso frequently display deformities and are of subpar quality, characterized by collapsed stomons, vascular ring browning, and brown flecks (Fig.1). When these tubers are fried for chips, the brown discoloration intensifies, resulting in a bitter taste and rendering the chips unsuitable for the market (Secor and Rivera-Varas 2004) [31].

Unfortunately, overreliance on pesticides has led to instances of neonicotinoid resistance in areas such as South Western United States, South Texas, and Northern Mexico (Prager et al. 2013; Chávez et al. 2015; Szczepaniec et al. 2019) [28, 4, 34], making this approach both economically and environmentally unsustainable. Various cultural methods for psyllid control have also been explored. These include using certified clean seed and incorporating non-host plants in crop rotations to maintain psyllid-free planting areas (Vereijssen et al. 2018) [36]. In warmer climates like the Southern United States, altering planting dates can delay exposure to potato psyllids (Guenthner et al. 2012) [122]. Some organic farmers have reported success in lowering psyllid infestations by employing physical barriers like mesh covers (Merfield et al. 2015) [21].

The first documented instance of potato psyllid infestation in peppers in Colorado, United States, was in 1909 when Sulc (1909) described it as a potential pest. However, it wasn’t until 1927 that the full extent of the detrimental effects of psyllids was realized. Vast outbreaks of what was then referred to as psyllid yellows (PY) disease occurred, leading to a significant reduction in potato yields in the Rocky Mountain States of the United States, particularly in Utah. Currently, the primary approach to managing ZC involves controlling the psyllid vector populations. This includes implementing various components of integrated pest management (IPM), such as chemical, cultural, and biocontrol strategies, which have been widely adopted worldwide (Vereijssen et al. 2018) [36]. Extensive monitoring and detection methods are also utilized to track psyllid populations and their movements (Butler and Trumble 2012) [3].

Data collected from monitoring psyllids in sweep nets are then correlated with psyllid-vectored diseases in tomato fields (Pletsch 1947; Cranshaw 1994) [27, 5]. Typically, psyllid infestations begin at the field’s perimeter and progress towards the center as their population grows (Wallis 1955; Cranshaw 1994) [39, 5]. Although leaf examination is a method to identify psyllid infestations, it is labor-intensive and time-consuming (Pletsch 1947; Goolsby et al. 2007) [27, 11]. Alternatively, sticky traps have proven effective for monitoring psyllid populations, even at low densities (Goolsby et al. 2007) [11].

In some regions, pesticide use has been the primary approach to psyllid control. This involves applying neonicotinoids like imidaclorpid and thiamethoxam as seed treatments at planting, followed by foliar applications to control adult and nymph psyllids (Prager et al. 2013; Vereijssen et al. 2015; Nuñez et al. 2019) [28, 37, 24].

**Fig 1:** Symptoms of Zebra Chip disease of potato (A) chlorosis and (B) upward curling of leaves (C) necrotic browning of tuber/ chips

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triggers in response to pests or pathogens (Dicke and Van Poecke 2002; War et al. 2012) [7–40]. Host resistance mechanisms to pests can be categorized as either antixenosis or antibiosis. Antixenosis refers to the plant’s ability to deter insect behavior, while antibiosis affects the insect’s life cycle and reproduction (Painter 1951; Kogan and Ortman 1978; Smith 2005) [29, 30].

In the context of ZC disease, researchers have identified several potato varieties and hybrids with varying degrees of tolerance to the disease. In some varieties, this tolerance is attributed to the deterrent effects of glandular trichomes (Butler et al. 2012; Diaz-Montano et al. 2014; Rubio-Covarrubias et al. 2017) [3, 6, 30]. Furthermore, a few varieties appear to possess a genetic basis for tolerance to CLSo, which also affects psyllid behavior (Rashidi et al. 2017; Fife et al. 2020) [29, 30]. Recently, certain wild relatives of tomatoes, such as S. pennelli and S. cornelianum, were found to possess resistance to B. cockerellii (Avila et al. 2019) [2], and several quantitative trait loci (QTL) associated with insect mortality and reduced fecundity were identified in S. habrochaites. These QTL found in wild species could serve as valuable sources for breeding resistance in cultivars. However, their complex inheritance, modes of action, and interactions among pathogens, vectors, and hosts necessitate further characterization.

**Purple top disease**

In North America, this pest is known to cause potato haywire disease and is classified as an AI quarantine pest by EPPO (OEPP/EPPO 1984) [25]. With the exception of the first one, all of these pests are categorized under the ‘aster yellows’ group, characterized by symptoms such as yellow or purple leaf discoloration. Typically, they are not transmitted through tubers. Leafhopper vectors responsible for their transmission include *Macrosteles* and *Hyalostethes* spp. In Europe, numerous phytoplasmas of the aster yellows type are found, infecting various hosts. However, only the potato stolbur phytoplasma is detected in potatoes, and it is generally recognized as a distinct phytoplasma strain. In Australia, several Asian countries, and the USA, the tomato big bud phytoplasma causes a disease that closely resembles stolbur. Additionally, three phytoplasmas in India are identified as belonging to the Old World aster yellows type and are differentiated from the potato stolbur phytoplasma. Potato purple-top wilt phytoplasma shares a close relationship with the aster yellows phytoplasma complex, which exhibits a broad spectrum of hosts. It can infect approximately 350 species spanning at least 54 plant families. Apical leaves exhibit symptoms of pinching, curling, and the development of yellowish-purple pigments. This leads to a loss of apical dominance in infected plants, causing a proliferation of axillary buds. The affected plants may prematurely wilt and die, often producing hair sprouts. According to Conners (1967), the main symptom is the occurrence of purple-top wilt, with the haywire stage attributed to secondary infections. In its natural habitat, potato purple-top wilt phytoplasma may spread through its leafhopper vectors. On a global scale, there is potential for it to be transported via potato plants or the insect vectors that are linked to them, although such transmission is rare in real-world scenarios.

**Conclusion**

ZC disease has now established itself in multiple potato-producing regions worldwide. The suspected causative agent, CLSo, has the potential to infect other economically significant Solanaceae crops, posing an even greater threat to the agricultural industry. Integrated pest management (IPM) strategies, including chemical, cultural, and biological controls, have been put into action to manage the psyllid vector population and curtail the spread of ZC disease. However, a need for long-term solutions remains evident. Recent advancements in potato genetic resources and crop improvement technologies offer promising opportunities for developing new potato cultivars with innate resistance to the psyllid and/or CLSo.

**References**