

Early Aphanomyces Resistance as a Key Element for Future Robust Sugarbeet Varieties

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Introduction

Aphanomyces cochlioides Drechs. is a soil-borne oomycete fungus causing both an acute seedling disease (damping-off) as well as a chronic rot of the mature root (black root) in sugarbeet. The pathogen can lead to serious damage in most sugarbeet growing areas, including North America, Europe and Asia. Chemicals like Hymexazol (Tachigaren®) have been instrumental in protecting the early developmental stages against seedling death and protecting the emerging plantlets from early infection, which contributes to heavy late root rot. While the late phase of Aphanomyces root rot has long been a focus of breeding, genotypic variation for early Aphanomyces infection was of lower priority in part due to highly effective chemical protectants.



Fig. 1. Aphanomyces disease nursery plots in field (Shakopee, MN). Traditional plots resistant variety (A) and susceptible variety (B). Early seedling evaluation at 2 weeks after planting dead (C) and alive (D). (Pictures: KWS)

Aim

To investigate genotypic variation for resistance to early Aphanomyces attack in KWS germplasm during different breeding stages (early breeding material, line development, experimental hybrids, and commercial hybrids) in order to improve this trait and contribute to the robustness of future sugarbeet varieties.



Fig. 2. Variation in sugarbeet reactions to early Aphanomyces attack in an Aphanomyces disease nursery (Shakopee, MN). (Picture: KWS)

Challenge

To develop a test system that exploits genotypic variation for early Aphanomyces infection, especially on early breeding material. This system would allow proper description of the final trait of interest: early Aphanomyces resistance. Early Aphanomyces attack happens during plant emergence and establishment. Hybrids and commercial varieties have consistently high germination rates (>90%), which allow plant reduction due to Aphanomyces to be calculated based on initial planting rate in routine field screenings (Fig. 1). Inbreeding effects and variable germination rates, especially under field conditions, require a more reliable germination baseline than the initial planting rate for inbred lines and younger breeding materials.

Approach

Different genotypic reactions were determined by first developing a baseline of expected plant stand under non-disease conditions, and later comparing that result with early plant reductions in Aphanomyces infected fields (Fig. 2). Germination assays were conducted under various conditions and in absence of disease. We used the baseline data to compare plant development rates with and without early Aphanomyces attack. Effect of Aphanomyces attack was assessed at different timepoints (2, 4, and 8 weeks) after planting (Fig. 3). Some genotypes had a paired Hymexazol treatment for comparison.

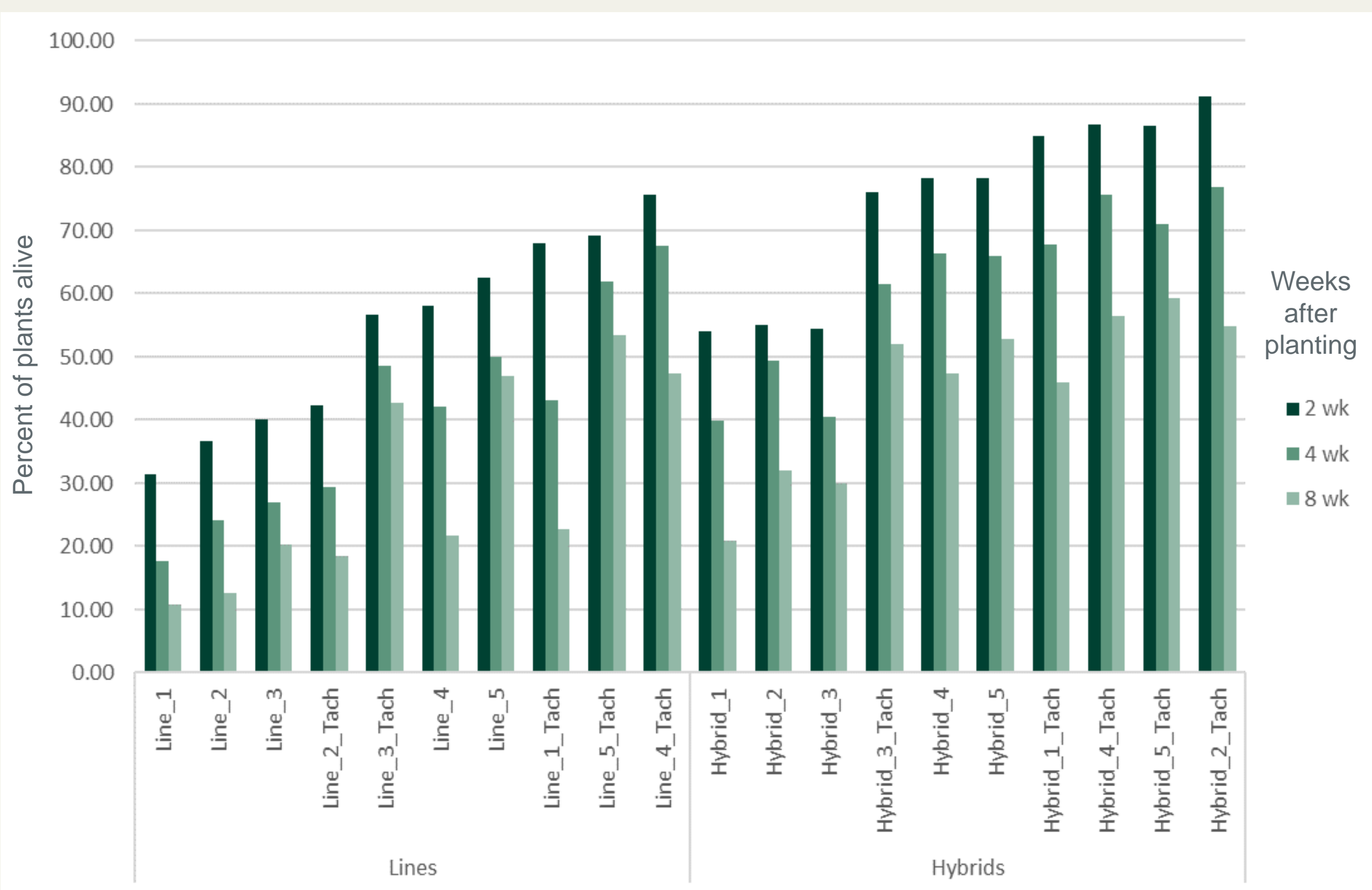


Fig. 3. There was variability between genotypes (LSD = 8.84) that was consistent over time (genotype by time interaction was NS). Hymexazol (Tach) treatment improved populations (LSD =12.56), even in more resistant genotypes.

Results

Resistance to early Aphanomyces attack was evident at 2 weeks after planting, and trends continued for evaluations at 4 and 8 weeks after planting. Even though Hymexazol treatments appear to improve survival, genetic resistance also appears to be a factor contributing to plant stand (Fig. 3). Heritable early Aphanomyces variation was found between different lines (Fig. 4) and hybrids (data not shown).

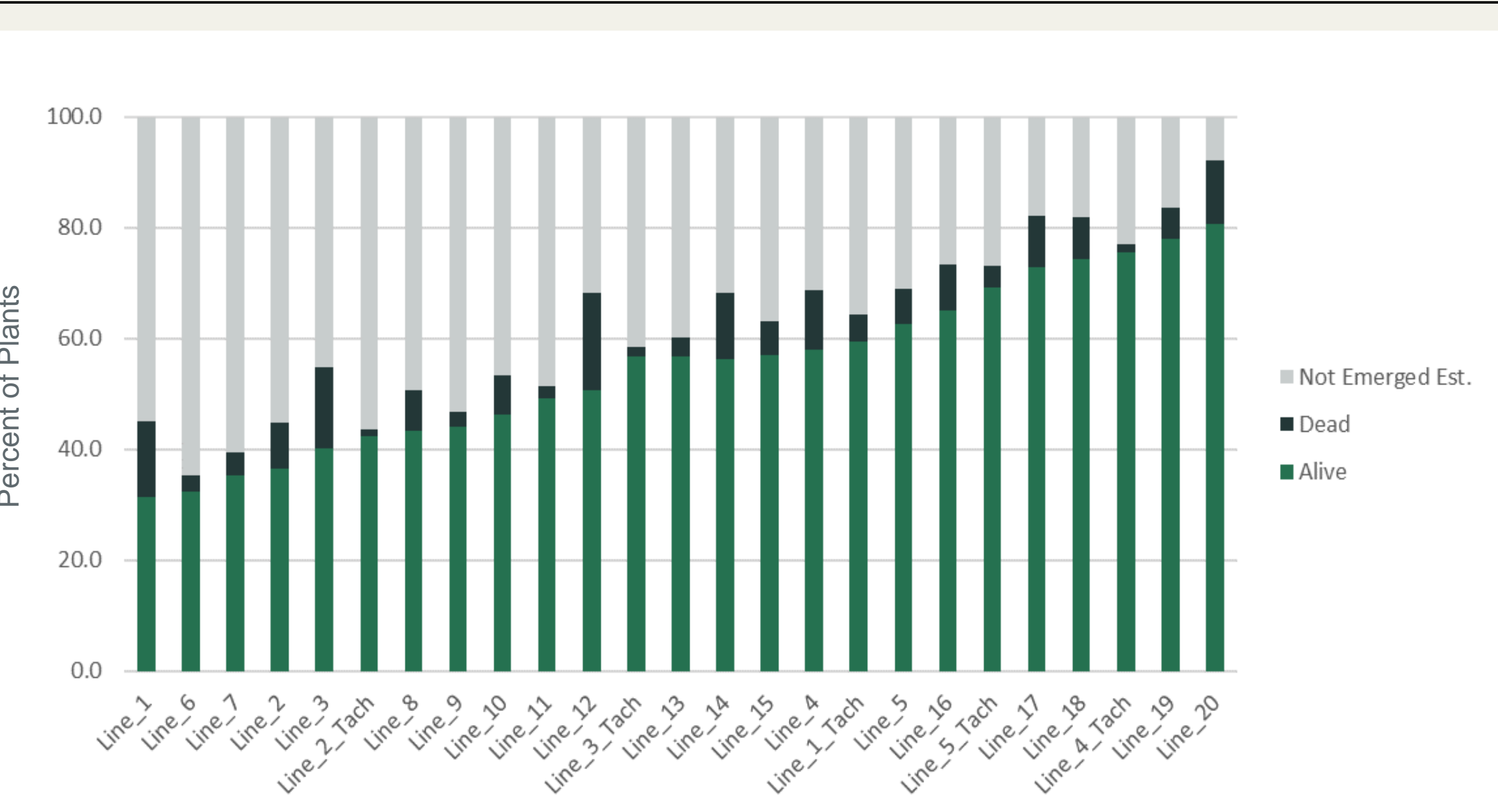


Fig. 4. Percent of plants surviving early Aphanomyces attack at 2 weeks after planting. Linear Mixed Model Analysis resulted in LSD of 19.27, CV of 0.233, repeatability of 0.882, and heritability of 0.957.

Conclusions and Outlook

Various sources of genotypic variability for early Aphanomyces have been reported, i.e., from wild beet resources or USDA germplasm. Within KWS, we have been working on early Aphanomyces resistance over many decades and are establishing routines for larger germplasm screenings. Within the different breeding materials, we can observe considerable and heritable genetic variation in response to early Aphanomyces attack. The relationship between early and late Aphanomyces resistance is a topic for future investigations. Current genetic variation is probably not strong enough to replace existing chemical protection, but improvements would provide complementary safeguarding. The goal of our breeding approach is to integrate early Aphanomyces resistance into variety development to further strengthen the line of defense against soil-borne diseases. Along with overall improved root and leaf health, and disease resistance packages, this trait shall serve as a key element for future robust sugarbeet varieties.

