PHENOTYPIC AND GENETIC EVALUATION OF LONREN GERMPLASM

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Abstract

The non-cultivated, African diploid species *Gossypium longicalyx* is virtually immune to reniform nematode (*Rotylenchulus reniformis*). By crossing *G. longicalyx* to bridging species and doubling the chromosomes of progenies with colchicine, Dr. A. Bell made two triple-species hybrids (2*n*=52), (*G. hirsutum x G. longicalyx*) x *G. armorianum* (HLA), and (*G. hirsutum x G. herbaceum*) x *G. longicalyx* (HHL). In 2005, Cotton Incorporated, Texas A&M University, and USDA-ARS began a program to develop agronomic *G. hirsutum* lines using the reniform resistance in the triple-species hybrids by means of a backcross (BC) breeding program. USDA-ARS also established cooperative research and development agreements with Bayer CropSciences and Delta and Pine Land Company (D&PL) to rapidly move the resistance into commercial cultivars. While development of lines for public release progressed, USDA-ARS made crosses of advanced lines from the respective companies with the backcross families. In the spring of 2007, two BC7 lines from HLA, LONREN-1 and LONREN-2, were publicly released. Registration of this release has not yet been submitted, because early-season stunting was observed in the summer of 2007 at certain sites managed by external cooperators where high populations for reniform nematode were present.

Subsequently, certain public breeders who received LONREN seed observed similar stunting. Following the 2008 field season, concordance across sites of the incidence of early-season plant growth reduction with the agronomic practices employed at the respective sites suggested that the stunting was associated with use of the photosystem II (PS II) inhibiting herbicides, fluometuron (Cotoran®) or prometryn (Caparol®). In January 2009, based on a growth chamber experiment, Dr. Bell presented a positive correlation of PSII herbicide exposure and stunting in the LONREN lines. However, in 2009, stunting was observed at a field site where PS II herbicides were not used and reniform nematode populations were high, but was not observed where reniform populations were low and no PS II herbicide was used. The instances of stunting have at least two possible causes: [1] the resistance mechanism itself causes, leads to, or otherwise entails injury or increased disease susceptibility; or [2] linkage drag, i.e., the inadvertent loss of beneficial Upland genes displaced by the alien segment, or gain of deleterious genes near the resistance gene. Comparative histology of reniform nematode attack on susceptible and LONREN lines demonstrates that the mechanism of resistance is lignification of the cells adjacent to the nematode head and collapse and necrosis of the syncytial walls; nonetheless, loss of cellular integrity is localized to the area surrounding the initial feeding cell. Accordingly, Cotton Incorporated, Texas AgriLife, and USDA-ARS believe that the resistance source is valuable, and are undertaking a renewed effort to find vigorous recombinants expressing the resistance. We further intend to register the original LONREN release for the scientific record, and hope to release new highly-resistant lines that are unencumbered by adverse characters.