Presence and Distribution of Suspected Palmer Amaranth Resistant to PPO-inhibiting

Herbicides in the North Carolina Coastal Plain. D.J. MAHONEY\*, D.L. JORDAN, A.T. HARE, , K.M. JENNINGS, R.G. LEON, and M.C. VANN, North Carolina State University, Raleigh, NC 27695; and N.R. BURGOS, University of Arkansas, Fayetteville, AR 72701.

In a survey conducted by the Weed Science Society of America, Palmer amaranth (*Amaranthus palmeri* S. Wats.) was named the most troublesome weed in the United States. Palmer amaranth is a highly competitive, obligate cross-pollinator whose pollen has been documented to travel great distances. Along with immense herbicide selection pressure, these characteristics have led to Palmer amaranth populations resistant to several modes of action with some populations expressing multiple resistance. Most recently, Palmer amaranth populations resistant to PPO-inhibiting herbicides have been confirmed in Arkansas, Illinois, and Tennessee. Evolved resistance was conferred by a glycine deletion (∆G210) and/or a glycine (R128G) or methionine (R128M) substitution for arginine within the PPX2 gene. While resistance in North Carolina (NC) has been suspected, it has yet to be confirmed in this species. Peanut producers in NC rely heavily on PPO-inhibiting herbicides for weed management; thus, rapid detection of resistance is critical to ensure management practices are adjusted to minimize wide-spread development of resistant populations. The objective of this research was to determine the presence and distribution of Palmer amaranth populations resistant to PPO-inhibiting herbicides in the NC Coastal Plain. In fall 2016, 125 Palmer amaranth populations were collected from fields predominantly in the NC Coastal Plain, the state’s primary peanut producing region. A known resistant population from Arkansas was included for comparison. Following inflorescences being dried, threshed, and cleaned, seeds were sown into cellular trays thinned to one plant cell-1. When plants reached the 2- to 4-leaf stage, they were treated with fomesafen (280 g a.i. ha-1) plus a nonionic surfactant (0.25% v v-1). Plant injury was estimated visually (0 to 100%) and mortality was recorded 3 wks after application. Plants surviving fomesafen were repotted to obtain tissue (100 mg) for genotyping via KASP assay based on the ∆G210, R128G, or R128M mutations. Three experimental runs were completed. Four populations from NC (35, 52, 53, and 56 from Edgecombe and Halifax counties) had survivors through the first two experimental runs, although percent survival was relatively low (1-10%). Therefore, a third experimental run was included using fewer populations to allow for an increase in individual plants to be screened. Four populations (6, 17, 32, and 107) were included and regarded as “susceptible” since no survivors were detected in the first two experimental runs. When pooled over experimental runs, percent survival of the Arkansas population (45%) was greatest. Percent survival from NC populations was as follows: population 56 (37%) > 52 (24%) > 17 (14%) = 32 (13%) > 35 (2%) = 53 (2%) = 6 (< 1%) = 107 (0%). Genotyping determined that all surviving plants from the Arkansas population possessed the ∆G210 mutation while the R128G and R128M mutations were not detected. No mutations were detected in surviving plants from NC suggesting resistance may be conferred by other mechanisms. Further tissue sampling was completed on surviving NC populations in order to sequence the PPX2 gene to determine if other mutations – which confer resistance – exist. Heritability work is ongoing to further characterize the mechanism of resistance in these populations. Whole-plant scale metabolic and tolerance assays with PPO-inhibiting herbicides will be conducted in all 125 collected populations.