Synergistic Impact of Bioactive Compounds and Pressure-Based Pasteurization against Wild-Type, Rifampicin-Resistant, and Pressure-Stressed O157 and Non-O157 Shiga Toxin-Producing *Escherichia coli*

Introduction: Various serogroups of Shiga toxin-producing *Escherichia coli* are important foodborne pathogens of public health concern in various commodities including apple cider. Adaption of high-pressure processing (HPP) continues to gain importance and momentum in the food industry.

Purpose: The current study investigated impact of malic acid, and citricidal for augmenting the pressure-based inactivation of O157 and non-O157 serogroups of wild-type, rifampicin-resistant, and pressure-stressed Shiga toxin-producing *E. coli* in apple cider.

Methods: A six-strain mixture of *E. coli* O157: H7 and six-strain mixture of non-O157 serogroups of the pathogen (O26, O45, O103, O111, O121, and O145) were used in this study. Additionally, three phenotypes of each strain mixture (wild-type, rifampicin-resistant, and pressure-stressed) were utilized for pressure-based (300 and 600 MPa) treatments with and without 3.5% malic acid and citricidal. Temperatures of trials were adjusted at 4.4 and 60.0 °C using a stainless-steel jacket surrounding Hub880 Barocycler chamber connected to a circulating water bath. Results were statistically analyzed using Tukey and Dunnett's-adjusted ANOVA.

Results: While 300 MPa treatments at 4.4 °C resulted in 0.45 log reduction ($P < 0.05$) after five minutes, same treatment resulted in 4.26 and 6.02 log reductions ($P < 0.05$) with addition of malic acid and citricidal, respectively. O157 and non-O157 serogroups and all three tested phenotypes illustrated similar sensitivity to elevated hydrostatic pressure, mild heat, and the two antimicrobials.

Significance: Results of our study illustrate that application of selected antimicrobials could to a great extent augment the decontamination capability of a pressure-based treatments against Shiga toxigenic *E. coli*. Additionally, we observed O157 and non-O157 serogroups of the pathogen have comparable sensitivity to the elevated hydrostatic pressure and the antimicrobials. Thus, a validated treatment plan against O157 serogroups is most probably equally effective against the non-O157 serogroups. A validated HPP plan against wild-type cells could similarly be efficacious against rifampicin-resistant and pressure-stressed phenotypes of the pathogen as well.