Understanding mechanisms of salt tolerance for reducing listeria monocytogenes growth in food

Human listeriosis is a serious foodborne disease caused by Listeria monocytogenes, a bacterium often called Listeria. The disease primarily affects older adults, pregnant women, newborns, and adults with weakened immune systems. After consumption of contaminated food, Listeria can enter the blood stream and can cause meningitis, encephalitis, miscarriage, or stillbirth. Approximately 90% of persons with bloodborne Listeria infection require hospitalization, and nearly 20% die. In some instances, Listeria causes gastrointestinal infection without spread to the blood, resulting in nausea, abdominal cramps, and diarrhea. Listeria contamination can occur in certain foods and can be particularly problematic with foods that are not cooked before consumption, including soft cheeses, deli meats, smoked fish, fruits, and salad greens. Several standard practices can be used to block the growth of bacteria in foods, including acidity, salt, and refrigeration.

Unfortunately, Listeria survives and grows relatively well under these conditions. As the industry reformulates their products to lower the sodium levels in processed foods, it is becoming more challenging to find options to keep foods free from Listeria and to store foods in a manner that prevents growth of Listeria when the bacterium is present in very low levels. An important area of research is to understand the mechanisms underlying Listeria's salt tolerance. Accordingly, we are attempting to identify genes that endow Listeria with salt tolerance. Using standard genetic manipulations, we identified a gene called lstC that, when eliminated, resulted in decreasing Listeria's growth in high salt environment. Further analysis revealed that the lstC gene product may be a protein involved in acetylation (a small chemical change) of other proteins. Acetylation of certain enzymes can alter cellular functions in a manner that alters microbial growth in different environments. A question remains as to what other enzyme activities, if any, are affected by the absence of lstC.

Using a sensitive molecular tool known as transcriptome analysis, we identified six genes whose expressions in Listeria were affected when lstC gene was eliminated. These six gene products are involved in different cellular functions like resistance to disinfectants, cell wall synthesis, and fatty acid synthesis. Although we do not know how these proteins affect Listeria salt tolerance, we speculate that changes in cell wall and/or fatty acids may play a key role. How does our study advance the goal of reducing Listeria growth in the presence of salt? The simplest approach entails identifying inhibitors of enzymes required for salt tolerance. For example, if one can identify specific inhibitors for LstC enzyme or enzymes affected by lstC gene mutation, we might be able to restrict Listeria growth in foods with lower levels of salt, aiding the goal of reducing Listeria growth in foods.

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Publication

A novel gene, lstC, of Listeria monocytogenes is implicated in high salt tolerance.
Burall LS, Simpson AC, Chou L, Laksanalamai P, Datta AR.
Food Microbiol. 2015 Jun