SIII-1-4

THE RPOS IS REQUIRED FOR A SUSTAINED ACID TOLERANCE RESPONSE IN SALMONELLA TYPHIMURIUM

Lee, In-Soo, Park, Yong-Keun[1], and John, W. Foster[2]
Dept. of Microbiology, Han Nam Univ., [1]Graduate School of Biotechnology, Korea Univ., and [2]Dept. of Microbiology and Immunology, School of Medicine, Univ. of S. Alabama

The acid tolerance response(ATR) of log-phase *Salmonella typhimurium* is induced by acid exposure below pH4.5 and will protect cells against more extreme acid. Two systems are evident: a transient induced system dependent on the iron regulator Fur that provides a moderate degree of acid tolerance and a more effective sustained ATR that requires the alternate sigma factor σ^s encode by *rpo*S. Differences between the acid responses of virulent *S. typhimurium* and the attenuated laboratory strain LT2 were attributed to disparate levels of RpoS caused by different translational starts. The sustained ATR includes seven newly identified acid shock proteins(ASPs) that are dependent upon σ^s for their synthesis. It is predicted that one or more of these ASPs is essential for the sustained system. The sustained ATR also provided cross-protection to a variety of other environmental stresses(heat, H_2O_2 and osmolarity); however, adaptation to the other stresses did not provide significant acid tolerance. Therefore, in addition to starvation, acid shock serves as an important signal for inducing general stress resistance. Consistent with this model, σ^s proved to be induced by acid shock. Our results also revealed a connection between the transient and sustained ATR systems. Mutations in the regulator *atb*R are known to cause the overproduction of ten proteins, of which one or more can suppress the acid tolerance defect of an *rpo*S mutant. One member of the AtbR regulon, designated atrB, was found to be co-regulated by σ^s and AtbR. Both regulators had a negative effect on *atr*B expression. The results suggest AtrB serves as a link between the sustained and transient ATR systems. When σ^s concentrations are low, a compensatory increase in AtrB is required to engage the transiently induced, RpoS-independent system of acid tolerance. Results also suggest different acid-senstive targets occur in log-phase versus stationary-phase cells.