DOWNLOAD PDF MICROBIAL STRESS ADAPTATION AND FOOD SAFETY

Chapter 1 : Microbial Stress Adaptation And Food Safety Download

The first book to address the subject, Microbial Stress Adaptation and Food Safety emphasizes the implications of stress adaptation and its consequences for food safety. It covers the basic science, kinetics, mechanisms, assessment, and control of stress adaptation and its impact on the safety of.

The stress sensor is not depicted, but this includes a lipid, protein, or nucleic acid component that senses the stress and ultimately causes a change in transcription or translation. Regulation of stress responses occurs at different levels depending on the stress and the bacterium. Control may occur at the transcriptional or translational levels or by adjusting the stability of the mRNA or protein Figure 1. Regulatory strategies vary considerably among bacteria and stresses. To add to the complexity, one stress response factor may be regulated at one or more levels. Transcriptional control of stress-induced genes and operons is a frequently encountered mechanism to control stress responses. One type of transcriptional control employs alternative sigma factors. The sigma subunit of RNA polymerase determines the specificity of promoter binding. Binding of an alternative sigma subunit to the RNA polymerase core enzyme changes its specificity, directing it to transcribe a different group of genes and operons. Several stress-related regulons coordinately regulated operons are positively controlled by the synthesis of an alternative sigma factor. Anti-sigma factors bind to a specific sigma factor forming a complex that prevents the sigma factor from binding to the RNA polymerase core enzyme Hughes and Mathee, A stress sensor may trigger release of the sigma factor from the anti-sigma factor complex, resulting in transcription of stressrelated genes. A sigma factor may be released from the anti-sigma factor by an antianti-sigma factor that binds to the anti-sigma factor. An anti-antisigma factor is present in a phosphorylated form in the absence of stress. Other transcriptional control mechanisms utilize repressor proteins that bind to the promoter region of a specific gene or operon, preventing transcription until conditions are appropriate, at which time the repressor protein is released from the DNA allowing transcription to proceed. The heat stress operons, dnaK and groE, are controlled in this manner in B. They are under the negative regulation by the HrcA repressor protein binding to the CIRCE controlling inverted repeat of chaperone expression operator Narberhaus, Synthesis of stress-related proteins can also be controlled at the translational level. Messenger RNA secondary structure near the ribosome binding site or translation start site can inhibit ribosome binding and translation of mRNA until stress conditions are experienced Takayama and Kjelleberg, Heat disrupts the hydrogen bonds holding the mRNA secondary structure together allowing the translation of the transcript under hot conditions Yura and Nakahigashi, Changes in mRNA and protein stability provide another method of controlling the activity of stress-related proteins. The half-life of some molecules can be increased or decreased in response to stress. Proteolytic degradation of stress-related proteins is also observed as a control mechanism. Activation of the general stress response usually results in reduced growth rate or entry into stationary phase Hengge-Aronis, Stress adaptive response in E. Expression of these genes is necessary for survival under stress conditions. Different stresses differentially affect these various levels of control. Heat causes damage to macromolecular cell components; thus the main function of heat-induced stress proteins is to repair or destroy these damaged components so they do not disrupt cellular metabolism. Many heat-induced stress proteins are protein chaperones that assist in folding and assembly of heat-damaged proteins e. In addition to these changes, some bacteria also alter their cell membrane in response to heat by increasing the ratio of trans to cis fatty acids in the membrane. This structural change is thought to decrease fluidity caused by increasing temperatures Cronan, After a temperature increase, the secondary structure is destabilized allowing translation to proceed. Gram-positive bacteria differ markedly in their regulation of heat shock response. This regulatory system is widespread and conserved within the bacterial kingdom and has been described in more than 40 different species Hecker et al. These genes are not only induced by heat, but also by other stresses, as discussed above Hecker and Volker Class III heatinduced genes are negatively controlled at the transcriptional level by a repressor protein, CtsR. CtsR binds to a specific sequence in the promoter region upstream of clp genes, clpP, clpE and clpC. These three genes are components of the Clp protease system which degrades damaged proteins Derre et al. It is not clear how CtsR

activity is changed after an increase in temperature. Other heat-induced genes, not controlled by the above mechanisms, are yet to be classified. Cold Physiological changes in response to cold include changes in the membrane fatty acid composition to promote optimum membrane fluidity Russell et al. Proteins synthesized in response to cold can be classified as Csps cold shock proteins or Caps cold-shock acclimation proteins. Csps are rapidly, but transiently overexpressed in response to cold. Caps are synthesized during continuous growth at cold temperatures; they are rapidly induced, but remain overexpressed several hours after the temperature downshift. A slow temperature downshift results in synthesis of some Csps and Caps Phadtare et al. Upon decrease in temperature, the phospholipid bilayer membranes of all cells decrease in fluidity. To maintain optimum fluidity, cells increase the unsaturation or decrease the chain length of the membrane fatty acids, resulting in increased fluidity at lower temperatures Russell et al. After cold shock in B. Cold shock also causes stabilization of the hydrogen bonds in nucleic acid secondary structures resulting in reduced efficiency of translation, transcription and DNA replication. These deleterious effects are overcome by induction of cold-shock proteins that serve as nucleic acid chaperones. CspA, the major cold-shock protein of E. CspA-like proteins contain two conserved RNA binding sequences. CspA is regulated at the transcriptional and translational levels and by increased mRNA stability at low temperatures Phadtare et al. The increase in cfa gene expression results in increased survival to the lethal challenge of pH 3 Chang and Cronan, The investigators suggest that the resulting changes may affect proton permeability through the membrane or the activity of a membrane-bound protein involved in acid stress. Limited information is available about the association of extracellular cell-tocell signaling and stress adaptation. Gram-positive bacteria, which regulate internal pH with an F0F1 ATPase, can increase synthesis or activity of the ATPase upon pH decrease, providing the cell with a higher capacity for proton efflux Foster, Low cytoplasmic pH can cause DNA damage. The importance of DNA repair in acid stressed cells is supported by data revealing that mutations in the ada gene, involved in DNA repair, cause acid sensitivity in Salmonella Foster, Amino acid catabolism can also help cells to fight a proton influx. Some Grampositive bacteria use the arginine deiminase system to alkalinize the cytoplasm Foster, Arginine is broken down into ornithine, carbon dioxide and ammonia. The resulting gamma amino butyric acid is exported via GadC. This system is induced by stationary phase or by acid in the exponential phase. A similar system involving arginine decarboxylase also protects E. Osmotic Stress Bacteria may encounter osmotic stresses in foods that are high in salt or sugar or in a dried state. Under such conditions, it is essential for the cell to maintain turgor pressure and hydration. The mechanisms described refer to bacteria that reside in environments with moderate or occasional hyperosmotic conditions. The best-characterized mechanism by which bacterial cells respond to hyperosmotic conditions involves intracellular accumulation of compatible solutes. This accumulation can be accomplished by synthesis or import from the environment. Compatible solutes are polar, highly soluble compounds that counteract osmotic pressure without affecting normal cellular functions, even at very high concentrations. Glycine betaine, proline, ectoine, carnitine, choline, and trehalose, among others, are common compatible solutes. Sensing of osmotic stresses is poorly understood Culham et al. Additional changes in cell metabolism in response to osmotic stress involve the cell membrane. An increase in the ratio of trans to cis unsaturated fatty acids is observed in cells exposed to high salt concentrations Cronan, Both regulons encode protein chaperones and proteases that assure proper assembly of proteins in the stressed cell Bianchi and Baneyx, Oxidative Stress In foods, bacteria may be exposed to increased levels of reactive oxygen species such as hydrogen peroxide, hydroxyl radicals and superoxide. Such oxidants cause damage to cellular proteins, lipids and nucleic acids. Many of the known proteins induced by oxidative stress have antioxidant roles. Others are involved in repair of oxidative damage, particularly damage to nucleic acids. OxyR senses oxidative damage via cysteine residues that are oxidized to form a disulphide bridge, altering the protein structure into the active form Mongkolsuk and Helmann, Under some stress conditions, microbial response is a protective effect, i. Higher levels of stress may injure the cells. Injured cells probably become energy-exhausted by multiple responses which decrease their capacity to react to additional insults. Additional stress usually kills injured cells see Figure 1. Food processors may learn about the consequences of mild treatments and the causes of resistance of pathogens to processes that are presumed lethal to these microorganisms. On the contrary, stresses that

sensitize pathogens to processing may have beneficial applications in food preservation. Using stress response to sense undesirable agents stressors in the food processing environment is another area of potential interest to food processors, but this has not been explored. To determine the conditions likely to lead to adaptive responses, researchers may vary stress level and apply stress at various physiological states of the targeted microorganism. Based on experience and a large amount of published literature, microbial adaptive response is most apparent at sublethal levels of stress and when the microorganism is in an active metabolic state, i. Many researchers, however, have demonstrated appreciable stationary-phase inducible adaptive responses e. Similarly, lethal doses of stress may trigger considerable adaptive responses in the fraction of the population that survives the treatment. After applying the stress under investigation, procedures to detect or quantify the response should be followed. Stress responses measured include changes in gene expression products RNA and proteins and stress tolerance see Figure 1. Although detection of stress adaptive response is generally laborious, distinction of injury is relatively simple. Details about adaptive responses are included in this contribution, but sensitization by stress will not be addressed. Included is a brief description of methods of applying theses stresses for inducing adaptive responses. Once the stress response is developed, cells should be handled in a way to preserve the response. Active metabolism and multiplication of stress-adapted cells deteriorate the adaptation and thus it becomes difficult to detect.

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The first book to address the subject, Microbial Stress Adaptation and Food Safety emphasizes the implications of stress adaptation and its consequences for food safety. It covers the basic science, kinetics, mechanisms, assessment, and control of stress adaptation and its impact on the safety of foods produced by minimal processing or non.

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Covers the basic science, kinetics, mechanisms, assessment, and control of stress adaptation and its impact on the safety of foods produced by minimal processing or non-thermal technologies.

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Adaptation is usually associated with the induction of a large number of genes, the synthesis of stress response proteins and the development of cross-resistance to a variety of stresses[29][

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While these arguments have some merits, we believe that the stress adaptation phenomenon has a profound effect on the safety of food: â€¢ Although stress adaptation is remarkable in actively metabolizing cultures, microorganisms at all phases of growth do adapt to stress.

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