Carbon dioxide and nisin act synergistically on Listeria monocytogenes

This paper examines the synergistic action of carbon dioxide and nisin on Listeria monocytogenes Scott A wild-type and nisin-resistant (Nis(r)) cells grown in broth at 4 degrees C. Carbon dioxide extended the lag phase and decreased the specific growth rate of both strains, but to a greater degree in the Nis(r) cells. Wild-type cells grown in 100% CO2 were two to five times longer than cells grown in air. Nisin (2.5 μg/ml) did not decrease the viability of Nis(r) cells but for wild-type cells caused an immediate 2-log reduction of viability when they were grown in air and a 4-log reduction when they were grown in 100% CO2. There was a quantifiable synergistic action between nisin and CO2 in the wild-type strain. The MIC of nisin for the wild-type strain grown in the presence of 2.5 μg of nisin per ml increased from 3.1 to 12.5 μg/ml over 35 days, but this increase was markedly delayed for cultures in CO2. This synergism between nisin and CO2 was examined mechanistically by following the leakage of carboxyfluorescein (CF) from listerial liposomes. Carbon dioxide enhanced nisin-induced CF leakage, indicating that the synergistic action of CO2 and nisin occurs at the cytoplasmic membrane. Liposomes made from cells grown in a CO2 atmosphere were even more sensitive to nisin action. Liposomes made from cells grown at 4 degrees C were dramatically more nisin sensitive than were liposomes derived from cells grown at 30 degrees C. Cells grown in the presence of 100% CO2 and those grown at 4 degrees C had a greater proportion of short-chain fatty acids. The synergistic action of nisin and CO2 is consistent with a model where membrane fluidity plays a role in the efficiency of nisin action.