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Acid Stress Response of *Streptococcus mutans* Isolated from Korean Children with Caries

Kyung-hee Kang[1] and Ingnyol Jin*

Department of Microbiology, Graduate School, Kyungpook National University, Daegu, South Korea
[1]Department of Dental Hygiene, Konyang University, Daejeon, South Korea

Much research has been devoted to studying streptococcal carbohydratemetabolism, particularly the biochemical and physiological adaptations that allow *S. mutans* to produce acids and to survive at low pH in the oral cavity. A number of recent reports have made use of the more holistic strategy enabled by proteome analysis to re-evaluate the mechanism of aciduricity in *S. mutans.* In this study, we are interested in comparing the protein profiles of acid-shocked cells with control cells of *S. mutans* and report proteins whose synthesis may be of importance in the acid tolerance response. *S. mutans* was identified at the species level using a 16S ribosomal DNA sequencing comparison method. The primer specificity was tested on eleven *S. mutans* strains isolated from Korean children with caries. The data showed that eleven strains are *S. mutans*. *S. mutans* K-11 exhibited the highest maximum culture OD compared with those of other groups. As a consequence, we examined the expression of protein under 20mM lactic acid stress using *S. mutans* K-11. The results of 2D gel electrophoresis by image analysis showed that twelve proteins are up-regulated. The identification of differentially expressed proteins associated with an acid-tolerant-growth phenotype provides new information on targets for mutagenic studies that will allow the future assessment of their physiological significance in the survival and proliferation of *S. mutans* in low pH environments.

Key words: Acid, Korean, Protein, *Streptococcus mutans,* Stress

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Salinomycin Induces Apoptosis in PC-3 Human Prostate Cancer Cells via G1 Cell Cycle Arrest and Ca^{2+}-Dependent Mitochondrial Pathway

Kwang youn Kim, Sun nyoung Yu, Dong min Kang,
Young rang Jin and Soon cheol Ahn

Department of Microbiology and Immunology, Pusan National University School of Medicine, Busan 602-739, Korea

Salinomycin is a polyether organic anion that is extensively used as microbial antibiotics. However, there is limited information on the effect of salinomycin on human prostate cancer cells. In the present studies, the effects of salinomycin on PC-3 human prostate cnacer cells were examined to better understand its effect on apoptosis and associated possible signal pathways in vitro. Apoptosis induction, cell cycle, reactive oxygen species (ROS), cytoplasmic Ca2+, mitochondrial membrane potential (MMP) were analyzed using the flow cytometric assay. Also, apoptosis-related proteins, such Bax, Bcl-2, caspase-3, PARP, and cell cycle-dependent proteins were determined by western blotting. As shown that, salinomycin promted the levels of Bax, Caspase-3 and reduced the level of Bcl-2, which were associated with the induction of apoptosis death of PC-3 cells. And salinomycin reduced the level of cell cycle dependent proteins. Salinomycin treated of cells results in mitochondrial depolarization, second mitochondrial activator of caspase release. Also, Ca2+ is involved in mitochondrial depolarization during salinomycin induced apoptosis. Our data suggest that Ca^{2+} modulates salinomycin-induced cell death via a Ca2+-dependent mitochondrial death pathway in PC-3 cells.

Key words: Salinomycin, Prostate cancer cell, Apoptosis, Ca^{2+} homeostasis