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Lantibiotics from *Streptococcus salivarius* as potential antimicrobials

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Lantibiotics are heat-stable, posttranslationally modified, ribosomally synthesized small peptides. Unlike other bacteriocins, this group contains some unusual residues such as (Lan) Lanthionin and (MeLan) β-methellanthionin. Prior to cleavage of the leader peptide posttranslational modification happens to the propeptide region of the precursor molecule. The modifications are the result of the dehydration of serine (Ser) and threonine (Thr) residues to 2,3-dehydoalanine (Dha) and (Z)-2,3-dehydrobutyrine (Dhb) while neighboring cysteines (Cys) link covalently to Dha and Dhb resulting in formation of (Lan) Lanthionin and (MeLan) B-methellanthionin bridges giving antibiotics their distinctive ring structure. Then the leader peptide with 23-59 amino acids will be proteolytically removed to give the lantibiotic its bioactive form. Lantibiotics can be produced by different genera of Gram-positive bacteria. *Streptococcus salivarius* is a member of lactic acid bacteria group or family within the normal flora of the human oral cavity and upper respiratory tract. It also inhabits the oral epithelial surfaces and is one of the first microorganisms to be established in the mouths of infants. It has been reported that some strains of S. salivarius can produce several bacteriocins most of which are lantibiotics. These lantibiotics produced by some of the S. salivarius strains keeps the oral cavity healthy and protected from many harmful microorganisms such as *Streptococcus pyogenes* and *S. sanguinis*. This protection comes by reduction in oral pathogen numbers. Two different lantibiotics namely salivaricin 9 and salivaricin G32 isolated from S. salivarius strains NU10 and YU10 respectively were characterized in this study. These lantibiotics showed antimicrobial activity against oral pathogens such as *Streptococcus pyogenes* that are a causal agent for sore throat and in developed stages can lead to rheumatic fever. The mechanism of antimicrobial action of salivaricin 9 was elucidated and it showed bactericidal activity towards test bacteria. The positively charged lantibiotic salivaricin 9 interacted with the negatively charged phospholipids of the cytoplasmic membranes of targeted bacterial cells which resulted in membrane depolarization and major morphological changes of the targeted cells. A new medium for enhanced lantibiotic production was developed and showed to increase the biomass accumulation of S. salivarius cells when grown aerobically. Subsequent recovery of the lantibiotics from the producer's cells was successful. This shows potentials for scale up for commercial production as antimicrobials for application in tooth paste or mouth wash. Attempts to study the regulation mechanism of lantibiotics production showed that salivaricin 9 lantibiotic is auto-regulated whereby small levels of the lantibiotic can enhance the production of the same molecule when it is introduced to the producer cells. On the other hand, salivaricin G32 showed no auto-regulation characteristic. However, the cell extracts of salivaricin G32 producer from strain YU10showed induction activity. Comparing the cell extracts of different lantibiotic-producing S. salivarius is a promising approach which may further contribute to lantibiotic production and regulation. Developing lantibiotics from lactic acid bacteria for clinical application may prove useful in view of increasing antibiotic resistance globally.

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