CELL WALL SYNTHESIS BY *STAPHYLOCOCCUS AUREUS* IN THE PRESENCE OF PROTEIN SYNTHESIS INHIBITORY AGENTS

III. BIOCHEMICAL STUDY

EIRYO KITANAKA, KAZUYORI OCHIAI, YASUHISA HAMASU, MASAFUMI NAKAO and SHOZO NAKAZAWA

Department of Microbiology, Kyoto College of Pharmacy, Kyoto, Japan

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Our previous reports[1,2] illustrated electron-microscopically that, following exposure to protein synthesis inhibitory antibiotics such as macrolides, lincomycins, and tetracyclines, *Staphylococcus aureus* thickened its cell wall and that such cell wall thickening phenomenon was not observed in clinically isolated resistant strains after similar exposure.

In our present study, elucidation of this cell wall thickening phenomenon has been attempted by allowing ^{14}C-L-lysine, one of the substances composing the bacterial cell wall, to be incorporated into the bacterial protein and cell wall. The PARK and HANCOCK method[3] was used in fractionating the bacterial protein and cell wall.

Figs. 1 and 2 represent the incorporation of the labeled L-lysine into the protein and cell wall fractions 15 minutes after the exposure of a sensitive *S. aureus* strain to clindamycin (CLDM), spiramycin (SPM), and chloramphenicol (CP), respectively. These figures show that the higher the concentration of each antibiotic, the less was the incorporation into the protein fraction, and the more the incorporation into the cell wall fraction. These results are similar to those of HANCOCK with chloramphenicol[4] and also to those of JOSTEN *et al.* with lincomycin.[5]

Figs. 3 and 4 show the incorporation of the labeled L-lysine into the protein and cell wall fractions 15 minutes after exposure of a clinically isolated macrolide-resistant *S. aureus* strain to erythromycin (EM), spiramycin (SPM), and oleandomycin (OL). Unlike with the sensitive strain, neither decreased incorporation into the protein fraction nor increased incorporation into the cell wall fraction was observed with this resistant strain. Similar findings were obtained also in another incorporation study in which ^{14}C-D-alanine was used.

The results obtained in the present study seem to be correlated with those obtained in our previous electron-microscopic study in that protein inhibition and cell wall thickening do not occur in resistant strains of *S. aureus* when they are treated with protein-inhibitory antibiotics.

Fig. 1. Incorporation of ^{14}C-L-lysine into protein fraction of *Staphylococcus aureus* FDA 209P.

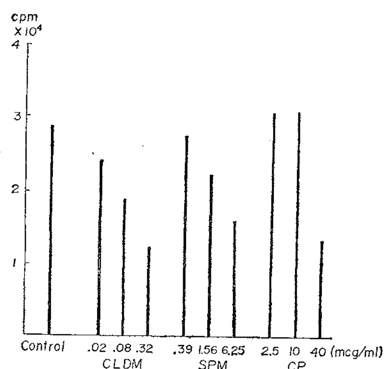


Fig. 2. Incorporation of ^{14}C-L-lysine into cell wall fraction of *Staph. aureus* FDA 209P.

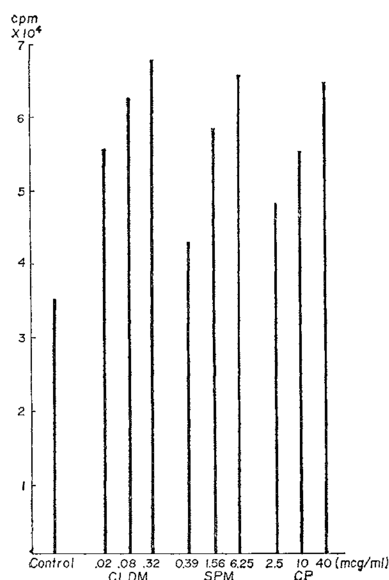


Fig. 3. Incorporation of ^{14}C-L-lysine into protein fraction of macrolide-resistant *Staph. aureus* strain.

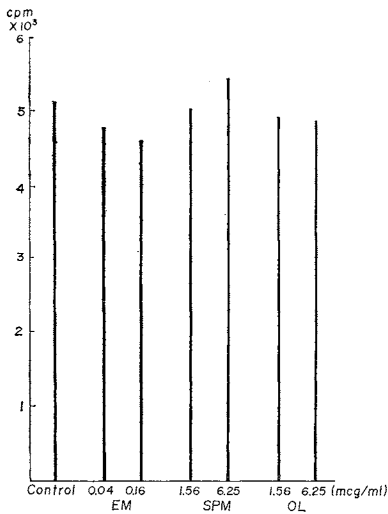
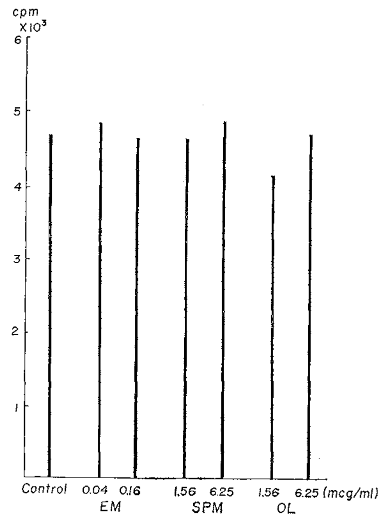


Fig. 4. Incorporation of ^{14}C-L-lysine into cell wall fraction of macrolide-resistant *Staph. aureus* strain.



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