

PABA	para-aminobenzoic acid
AcDHPs	3,5-diacetyl-1,4-dihydropyridines /G1-G11/
BzDHPs	3,5-dibenzoyl-1,4-dihydropyridines /GB1-GB15/
CAM	camphor-degradation plasmid
COM	catechol-O-methyl-transferase
CFU	colony formit unit
CNS	coagulase negative Staphylococcus
CP	clomipramine
2,4-D	dichlorophenoxyacetic acid
EMB	eosine methylene blue
ERY	erythromycin
F-plasmid	fertility plasmid
FIC	fractional inhibitory concentration
GFP	green fluorescence protein
GISA	glycopeptide intermediate <i>Staphylococcus aureus</i>
Hfr	high frequency protein
IM	inner membrane
LB-broth	Luria -Bertani broth
MB	methylene blue
MDR	muldrug resistance
MFP	major facilitator protein
MFS	major facilitator superfamily
MIC	minimum inhibitory concentration
MLS	macrolides lincosamid streptogramin
MRSA	methicillin-resistant <i>Staphylococcus aureus</i>
MSSA	methicillin-sensitive <i>Staphylococcus aureus</i>
MTT	3-(4,5-dimethylthiazol)-2,5-diphenyltetrazoliumbromide
MTY	minimal tryptone yeast extract
NAH	naphthoic acid hydroxydase
OD	optical density
OM	outer membrane
Oxa	oxacillin

NP	nifedipine
PABA	para-aminobenzoic acid
PBP	penicillin binding protein
PMF	proton motive force
PZ	promethazine
R-plasmid	resistance plasmid
RND	resistance nodulation division
RTF	resistance transfer factor
SMR	small multidrug resistance
TC	tetracycline
TNase	termonuclease
Tn	transpozon
TPP	tetraphenylphosphoricum
VP	verapamil

L. INTRODUCTION

Drug-resistant infectious agents are an increasingly important public health concern. Antimicrobial resistance is becoming a factor in virtually all hospital-acquired or nosocomial infections. Resistance to antimicrobial agents among bacteria and fungi is a persistent problem that complicates the management of critically ill patients (2).

Staphylococcus aureus and enterococci are most commonly isolated bacteria that cause hospital-acquired infections. Among those giving rise to therapeutic problems are methicillin-resistant staphylococci and vancomycin-resistant enterococci (3).

When penicillin was introduced in 1944 over 94% of *S. aureus* isolates were susceptible, by 1950 half were resistant. By 1960, many hospitals had outbreaks of virulent multidrug-resistant *S. aureus*. These were overcome with penicillinase-stable penicillins, but victory was brief; strains of methicillin-resistant *S. aureus* (MRSA) were recorded in the year of the drug's launch. MRSA owe their behaviour to an additional, penicillin-resistant peptidoglycan transpeptidase, PBP-2', encoded by *mecA* gene. Their spread is clonal, with transfer of *mecA* being extremely rare. MRSA accumulated and then declined in the 1960s and 1970s, but became re-established in the early 1980s.

Coagulase-negative staphylococci (CNS) are less pathogenic than *S. aureus* but are important in line-associated bacteraemias and prosthetic device infections. They are even more often resistant than *S. aureus*, notably to teicoplanin. Few anti-staphylococcal agents were launched from 1970 to 1995, but the situation is now improving (4).