Skin and mucosal infections, especially purulent, can often cause diagnostic and treatment problems. Therefore, we conduct a microbiological study of isolated bacterial strains from ambulatory patients with various cutaneous-mucosal infections. In isolated strains we determined the sensitivity to antibiotics and we established the phenotypes of resistance in which they fall. We took into account 98 strains isolated by a private laboratory in Timisoara between January 2016 and December 2017. After bacterial identification, sensitivity testing to antibiotics was realized using Kirby-Bauer disc diffusion according to the CLSI standard. Antibiogram was performed for all strains except for Streptococcus pyogenes. By interpreting the antibiograms, the phenotypes of resistance were determined. Most infections were caused by Staphylococcus aureus. In order of frequency following strains were isolated: Staphylococcus aureus (75.52%), Streptococcus pyogenes (11.22%), Pseudomonas aeruginosa (7.14%), E. coli (4.08%) and Enterobacter spp. (2.04%). S. aureus strains were resistant to penicillin in 94.60% of cases. We observed a higher sensitivity to oxacillin (87.84%), tobramycin (86.49%), gentamicin (87.84%), ciprofloxacin (83.78%) and clindamycin (89.20%). Most strains of S. aureus were of phenotype Peni-R Meti-S (82.44%). 12.16% were of phenotype Peni-R Meti-R (MRSA) and only 5.40% were sensitive to betalactams (Peni-S Meti-S). Gram-negative bacilli strains (Pseudomonas aeruginosa, E. coli, Enterobacter spp.) were less resistant, all being wild strains. Determining antibiotic resistance phenotypes is necessary in order to be able to make the right decision when choosing anti-infectious treatment, but also to prevent the selection of multi-resistant bacterial strains. The presence of MRSA at a rate of 12.16% is an alarm signal because the MRSA strains are multi-resistant to antibiotics with cross-resistance to the betalactams. Resistance usually extends also to other classes of antibiotics. For a correct diagnosis and treatment, the results of the bacteriological testing need to be corroborated with the clinical signs.

Keywords: skin- and mucosal infections, resistance phenotypes, antibiotics, chemotherapeutical agents, MRSA
isolates in amounts of $10^{3-4}$ UFC/g of tissue have clinical significance if the histopathological examination confirms the presence of inflammatory infiltrates, vasculitis and thrombosis lesions;
-β-haemolytic streptococci have clinical significance regardless of the amount of their isolation [2, 4, 5, 22, 23].

After bacterial identification, sensitivity testing to antibiotics was realized using Kirby-Bauer disc diffusion according to the CLSI standard [14, 23, 24]. Antiobigram was performed for all strains except for Streptococcus pyogenes - to which no penicillin-resistant strains have been reported [22-25].

By interpreting the antibiograms, the phenotypes of resistance were determined.

Results and discussions

Most infection, as expected, were caused by Staphylococcus aureus. Of the 98 strains, the following germs were isolated in order of frequency: Staphylococcus aureus (75.52%), Streptococcus pyogenes (11.22%), Pseudomonas aeruginosa (7.14%), E. coli (4.08%) and Enterobacter spp. (2.04%) (fig. 1).

In hospitalized antibiotic-treated patients, antibiotic sensitive Staphylococcus aureus strains can be replaced with resistant or multi-resistant germs [7, 26, 27].

Antibiotic sensitivity was tested in all isolates except S. pyogenes [2, 4, 5, 22, 23, 26]. The strains of S. aureus had the following characteristics:
- 94.60% were resistant to penicillin, most of the strains being penicillinase producing;
- intermediate sensitivity we determined for: kanamycin (60.81%), erythromycin (70.27%) and trimethoprim-sulfamethoxazole (71.62%);
- they were susceptible to: oxacillin (87.84%), tobramycin (86.49%), gentamicin (87.84%), ciprofloxacin (83.78%) and clindamycin (89.20%).

Interpretative analysis of antibiograms allowed stratification of S. aureus strains in resistance phenotypes.

By determining the antibiotic resistance phenotypes, information on the mechanism of antibiotic resistance can be obtained and the substances with which the test bacteria is resistant can be deduced - based on these mechanisms [26, 28-30].

Three phenotypes of beta-lactam resistance are found in S. aureus: Peni-S Meti-S, Peni-R Meti-S, Peni-R Meti-R (MRSA) [26, 28, 29]. Most of the isolated strains fell into the Peni-R Meti-S phenotype (61 strains - 61.44%). 9 strains (12.16%) fell into the Peni-R Meti-R phenotype (MRSA) and only 4 strains (5.40%) were sensitive to betalactamins (Peni-S Meti-S) (fig. 2).

The presence of MRSA at a rate of 12.16% is an alarm signal because the MRSA strains are multi-resistant to antibiotics with cross-resistance to the betalactams. Resistance usually extends also to other classes of antibiotics [27, 28].

The phenotype K (kanamycin resistance) reported in 25.68% of the strains, as well as the KT phenotype (kanamycin resistance and tobramycin), found in 1.35% of the strains, are predictive of resistance to amikacin; netilmicin remaining active [28].

![Fig. 1. Bacterial strains isolated from cutaneous-mucosal infections](image1)

![Fig. 2. Phenotypes of betalactamine resistance of S. aureus strains](image2)

### Table 1

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Abbreviations: P penicillin, OX oxacillin, K kanamycin, TM tobramycin, GM gentamicin, E erythromycin, CLI clindamycin, CIP ciprofloxacin, SXT trimethoprim-sulfamethoxazole.
Resistance to kanamycin, tobramycin and gentamycin (KTG phenotype) present in 12.16% of the strains is predictive of resistance to amikacin and netilmicin [28]. For the determination of resistance to MLS (macrolides, lincosamide, streptogramins), the erythromycin association with clindamine was tested by disc diffusion. Phenotypic expression of resistance may be inducible or constitutive: MLSB inducible phenotype, MLSB constitutive phenotype [24-27].

Of 22 (29.73%) resistant strains, 21 (28.38%) presented the MLSB phenotype: 13 (17.57%) - MLSB inducible phenotype and 8 (10.81%) - MLSB constitutive phenotype. An antibiotic efflux resistance was observed in a (1.35%) strain of S. aureus (phenotype M) (fig. 4).

For the detection of resistance to fluoroquinolones, a single quinolone was used - ciprofloxacin, staphylococci having cross-resistance to all fluoroquinolones [28]. Most of the strains were sensitive - 83.78%, a small part gained resistance - 16.22% (fig. 5).

At trimetroprim-sulfamethoxazole 71.62% of the strains were susceptible, the remaining 28.38% being resistant (fig. 6).

Taking into account gram-negative bacterial strains isolated from cutaneous-mucosal infections (Pseudomonas aeruginosa, E. coli, Enterobacter spp.), they were less resistant, all of them being wild strains with natural sensitivity to antibiotics preserved.

Conclusions
Establishing antibiotic resistance phenotypes is necessary in order to be able to make the right decision in choosing anti-infectious treatment, but also to prevent the selection of multi-resistant bacterial strains.
Isolation in outpatient patients of 12.16% of MRSA strains is an alarm signal because they are multi-resistant to antibiotics with cross-resistance to the beta-lactamase complex, and the resistance usually extends to other classes of antibiotics.
Taking into account the isolation of multi-resistant strains, it is necessary to target antimicrobial agents based on antibiograms and to implement an epidemiological surveillance system of the resistance phenomenon.

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References
24. *** CLSI -Performance Standards for Antimicrobial Disk Susceptibility Tests; Approved Standard M02, M100-Clinical and Laboratory Standards Institute, USA 2015, 2016.

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