Publications Template

#	Research Title	Field	Abstract	Year of Publication Publishing	Publishing Link "URL"
1	**Community-acquired methicillin-resistant *Staphylococcus aureus* from skin and soft tissue infections (in a sample of Egyptian population): analysis of *mec* gene and staphylococcal cassette chromosome**	**Microbiology**	Background *Staphylococcus aureus* has been recognized as an important pathogen associated with inpatients and community infections. Community-acquired methicillin-resistant *S. aureus* (CA-MRSA) infections commonly present as skin and soft-tissue infections (SSTIs). Treatment often includes incision and drainage with or without adjunctive antibiotics. Objectives This study aimed to identify CA-MRSA infections both phenotypically and genotypically, to determine their spectrum of antibiotic resistance, and to establish the best scheme for molecular distinction between hospital-acquired MRSA (HA-MRSA) and CA-MRSA by staphylococcal cassette chromosome *mec (SCCmec*) typing and detection of Panton Valentine leukocidin (PVL). Materials 50 swabs, from skin and soft tissue of infected lesions of outpatients attending the dermatology department of the Medical School, Alexandria University, were collected. Additionally, a nasal swab was taken from every participant. Methods Collection of swabs from the infected skin and soft tissues,	**2012**	https://www.sciencedirect.com/science/article/pii/S1413867012001158

			followed by laboratory testing to phenotypically and genotypically identify MRSA. Also, nasal swabs were taken from every patient to identify MRSA colonization. Results *Staphylococcus aureus* strains were identified in 38 (76%) of the 50 clinical isolates. 18 (47.37%) out of the 38 *S. aureus* strains were resistant to oxacillinand cefoxitin discs, were penicillin binding protein 2a (PBP2a) producers, and were initially diagnosed as MRSA. All of the 18 strains were definitively diagnosed as MRSA by *mecA* gene detection using real time PCR, while only six (33.33%) strains were PVL positive. Using the sets of primers of Zhang et al.: nine (50%) out of the 18 CA-MRSA strains were *SCCmec* type V, and one (5.56%) was *SCCmec* type IVc. Then, using the set of primers by Oliveira et al., two (25%) out of the eight untypable MRSA strains were found to be *SCCmec*type IV, and six (75%) remained untypable. Conclusions CA-MRSA must be considered when treating skin and soft tissue infections, especially in developing countries. Empirical use of agents active against CA-MRSA is warranted for patients presenting with serious SSTIs.		
2	**Emergence of *bla*VEB and *bla*GES among VIM-producing *Pseudomonas aeruginosa* clinical isolates**	**Microbiology**	Thirty-three *Pseudomonas aeruginosa* isolates, resistant to one or more β-lactams, were included in this study. Identification of tested strains was confirmed using MALDI-TOF/MS. Phenotypic and genotypic β-lactamase patterns were investigated. Most of the isolates were resistant to carbapenems (32 out of 33) and to the extended-spectrum cephalosporins	**2019**	https://akademiai.com/doi/abs/10.1556/030.65.2018.044

	in Alexandria, Egypt		(ESC) (30 out of 33). Phenotypically, the production of extended-spectrum beta-lactamase (ESBL), metallo-β-lactamases (MBL), and carbapenemases was detected in 10, 23, and 9 isolates, respectively. However, AmpChyperproduction was not phenotypically detected among all isolates. Genotypically, ESBL and MBL encoding genes were detected in 23 and 27 isolates, respectively. Altogether 27 strains were detected as *bla*$_{VIM}$ positive and 16 strains carried *bla*$_{OXA-10}$ gene. To the best of our knowledge, this is the first report of *P. aeruginosa* clinical isolates harboring *bla*$_{VEB}$ together with *bla*$_{GES}$ in Egypt, where 5 of our 30 ESC-resistant isolates showed this genotype. Our results confirmed that resistance of *P. aeruginosa*isolates to β-lactam antibiotics is mediated via multiple β-lactamases belonging to different molecular classes. To the best of our knowledge, this is the first report of *bla*$_{VEB}$ among *P. aeruginosa* clinical isolates from Egypt. Ten isolates harbored *bla*$_{VEB}$ and five of them co-harbored *bla*$_{VEB}$ together with *bla*$_{GES}$, *bla*$_{VIM}$, and *bla*$_{OXA-10.}$		
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