Prevalence of some virulence factors in *Staphylococcus aureus* isolated from health care personnel: haemolysis and enterotoxigenic genes profile

A. Castro, N. Silva, J. Silva, P. Teixeira*

CBQF - Centro de Biotecnologia e Química Fina – Laboratório Associado, Escola Superior de Biotecnologia,
Universidade Católica Portuguesa/Porto, Rua Dr. António Bernardino Almeida, 4200-072 Porto, Portugal

*pcteixeira@porto.ucp.pt

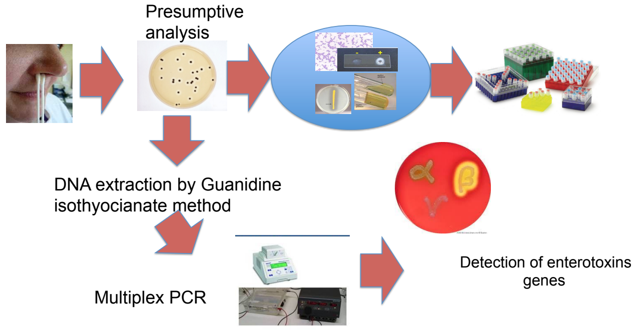
Objective

The purpose of this study was to evaluate virulence, haemolysins and enterotoxins genes, of *Staphylococcus aureus* isolated from nares of health care professionals of a central hospital in Porto and to compare prevalence of these virulence factors among Methicillin-Resistant *Staphylococcus aureus* (MRSA) and Methicillin-Sensitive *Staphylococcus aureus* (MSSA).

Introduction

Staphylococcus aureus is a common cause of infection and is one of the leading causes of nosocomial infections [1]. *Staphylococcus aureus* has developed ability to cause human disease either by multiplication both locally and systemically or by exerting their pathogenic effects producing exotoxins or enzymes that act at distant sites [2]. Various virulence factors have been described in the literature facilitating host invasion and growth of this bacteria [2]. *Staphylococcus aureus* haemolysins have several biological activities: α-haemolysin causes lyse of erythrocytes and damage platelets having a powerful action on vascular smooth muscle; β-haemolysin degrades sphingomyelin and is toxic for many cells, including human blood red cells; γ-and δ-haemolysins also cause lysis of a variety of cell types. *Staphylococcus aureus* enterotoxins are at least nine (A-E and G-J) and function as superantigens yielding host T cells stimulation, are heat stable and resistant to the action of gut enzymes [2]. This study contributes for better knowledge if MRSA are in general more virulent than MSSA.

Methods



Results and discussion

Seventy isolates were tested for production of α- and β-hemolysin on Columbia agar supplemented with 5% blood and for the presence of enterotoxigenic genes (*sea* to *sej*) by multiplex PCR.

MSSA

Isolate	OXA	Haemolysis	Staphylococcal enterotoxins genes
10N	S	β	-
11N	S	α	*secbov, seg, tsst*
12N	S	β	*seg, sei*
26N	S	α	*sea, seg*
33N	S	α	*seg, sei*
34N	S	α	*sea, seg, sei, tsst*
37N	S	β	*secbov, seg, sei, tsst*
38N	S	α	*seh, seg, tsst*
45N	S	α	-
52N	S	α	*seh, seg, sei, tsst*
57N	S	α	*sea, seg, sei*
58N	S	β	*secbov*
60N	S	α	*seg, tsst*
63N	S	α	*seh, sei*
67N	S	α	*sea, seg*
71N	S	γ	*seg, sei*
75N	S	γ	*sea, seg*
76N	S	γ	*seg, sei*
81N	S	α	*seh, seg, sei, tsst*
85N	S	β	-
91N	S	α	*seg, sei, tsst*
101N	S	α	*seb*
107N	S	α	*sed, seg, sei, sej*
108N	S	β	*secbov, seg, sei*
114N	S	α	*seg, tsst*
116N	S	γ	-
122N	S	β	*sed, seg, sei, sej*
131N	S	α	-
133N	S	α	-
141N	S	α	-
144N	S	α	*secbov, seg, sei*
148N	S	α	*sea*

MRSA

Isolate	OXA	Haemolysis	Staphylococcal enterotoxins genes
18N	R	α	*secbov, seg, sei*
22N	R	β	*secbov, seg, sei*
29N	R	β	*secbov*
30N	R	β	*secbov, seg, sei*
42N	R	β	*seh, seg, sei*
47N	R	β	-
49N	R	β	*secbov, seg, sei, tsst*
51N	R	β	*secbov, seg, sei*
53N	R	α	*secbov, seg, sei*
54N	R	α	*secbov, seg, sei*
59N	R	β	*secbov, seg, sei*
60N	R	α	*seg, tsst*
61N	R	β	*secbov, seg*
69N	R	β	*secbov, seg, sei*
72N	R	β	*secbov, seg, sei*
73N	R	β	*secbov, seg, sei*
80N	R	β	*secbov, seg, sei*
82N	R	β	*secbov, seg, sei*
83N	R	α	*seg, sei*
94N	R	β	*secbov, seg, sei*
103N	R	β	*sea, seg, tsst*
124N	R	β	*secbov, seg, sei*
126N	R	β	*seg*
130N	R	α	*seh, seg*
134N	R	β	*secbov, seg, sei*
137N	R	β	*seb, seg, sei*
140N	R	β	*secbov, seg, sei*
146N	R	α	-
152N	R	β	*seh, seg*
4NII	R	β	*secbov*
6NII	R	β	*secbov, seg, sei*
9NII	R	β	*secbov, seg, sei*
10NII	R	β	*secbov, seg, sei*
12NII	R	β	*secbov, seg, sei*
14NII	R	β	-
17NII	R	β	*secbov, seg, sei*

Conclusions

The majority of MRSA (76%) presented β- hemolysis while the majority of MSSA (66%) presented α- hemolysis. The majority of MRSA (70%) presented an enterotoxin gene profile (*secbov, seg, sei*) while MSSA enterotoxin gene profiles were uniformly distributed

References

[1].R. J. Gorwitz, D. Kruszon-Moran, S.K. McAllister, G. McQuillan, L. K. McDougal, G. E. Fosheim, B. J. Jensen, G. Killgore, F. C. Tenover and M. Kuehnert ,The Journal of Infectious Diseases 2008, 197: 1226-34. [2]. G.F. Brooks, J. S. Butel, L.N. Ornston, Medical Microbiology, 1995, Jawetz, Melnick & Adelberg´s.

Governo da República Portuguesa

Acknowledge

This work was supported by National Funds from FCT – Fundação para a Ciência e a Tecnologia through project PEst-OE/EQB/LA0016/2011. FCT for PhD grant BD/39315/2007 to Ana Isabel Castro.