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Molecular epidemiology, antigenic profile and macrolide resistance of Bordetella pertussis clinical isolates circulating in Barcelona from 2007 to 2014

Alba Mir-Cros[1], Maite Martin[1], María Gema Codina[1], Tomás Pumarola Sole[2], Juan-Jose González-López*[1]

[1]*Hospital Universitari Vall D'hebron, Microbiology, Barcelona, Spain*

[2]*Hospital Universitari Vall D'hebron, Microbioloogy, Microbiology, Barcelona, Spain*

Background: Whooping cough is nowadays a reemerging disease despite extensive vaccination campaigns and high immunization rates. One of the causes postulated that may be involved in this phenomenon is the adaptation of *Bordetella pertussis* to the immunity induced by the acellular pertussis vaccines (ACV). The ACV contains the following proteins variants: pertussis toxin (PtxA2/Ptx4), pertactin (Prn1/Prn7) and type 3 fimbriae (Fim3-1). Recently, resistant isolates of *B. pertussis* to erythromycin, the first choice for treatment of whooping cough, have been reported. The objectives of this study are to determine the molecular epidemiology, the ACV antigens variants and the presence of the genetic macrolide-resistance marker of *B. pertussis* isolates from the Barcelona metropolitan area.

Material/methods: 109 non-duplicate *B. pertussis* clinical isolates, collected between 2007 and 2014 at Hospital Vall d'Hebron (Barcelona, Spain), were studied. Genetic relatedness was determined by pulsed-field gel electrophoresis (PFGE). Clonally related PFGE profiles were grouped into different clades using a similarity criterion of 82%. The ACV antigens variants were studied by PCR and sequencing. Finally, the A2047G mutation in the 23S rRNA conferring macrolide resistance was assessed by allele-specific PCR.

Results: The 109 isolates were distributed in 15 different pulsotypes. Among them, the most prevalent were pulsotype A, C and E (27.52%, 20.18% and 17.43%, respectively). The pulsotype similarity analysis showed that the 90.83% of the isolates were grouped into two clades: clades I (47.7%), in which the 57.7% of the isolates belonged to pulsotype A, and clade II (43.1%), in which the 49.0% and the 40.43% of the isolates belonged to pulsotype C and E, respectively. Clade I isolates were the most prevalent from 2007 to 2010. However, clade II isolates were the most prevalent from 2011 to 2014. The 100% of the isolates encoded the *ptxA1* allele, the 97.2% the *prn2*, the 54.1% the *fim3-2,* and the 45.9% the *fim3-1*. Type 3 fimbriae alleles were distributed differently among clades; *fim3-1* was found mainly in clade II (89.4%) and *fim3-2* in clade I (100%). Finally, all isolates were negative for the A2047G mutation in 23S rRNA.

Conclusions: In our area, two *B. pertussis* populations coexist from 2007 to 2014. The first population was more prevalent until 2010 when progressively was replaced by the second one. All the alleles

found in both populations differ from those included in the ACV except for the type 3-2 fimbriae, which has been gradually displaced by the type 3-1 since 2011. Overall, the results obtained in this study suggest that *B. pertussis* producing whooping cough in Barcelona, may have adapted to the immunity induced by the ACV. Finally, any studied isolates possessed the genetic marker associated to *B. pertussis* macrolide resistance.