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Influenza and respiratory viruses

Full genome sequence analysis of influenza H1pdm09 and H3N2 viruses related to severe respiratory illness at a tertiary hospital from 2012 to 2015 in Catalonia, Spain

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Background: In addition to host factors, viral genetic markers might be related to severe influenza respiratory infection. The aim of this study was to describe the genetic features of influenza A viruses detected in non-hospitalised and in severe hospitalised patients who were attended at a tertiary hospital from 2012 to 2015.

Material/methods: From week 40/2012 to week 20/2015, respiratory tract specimens were tested for influenza laboratory-confirmation. Full influenza genome amplification was performed using the universal primer set and the PCR protocol by Zhou et al (Zhou et al. 2012). Sequencing libraries were constructed by nebulisation (shot-gun) and subsequent ligation of adapter with multiplex identificators. Roche 454 sequencing was carried out according to manufacturer's recommendations using Roche 454 GS Junior platform. Bioinformatic analysis was performed to characterise the sequences and the quasispecies composition. The sequences corresponding to the recent vaccine strains A/California/04/2009 and A/Victoria/361/2011 were used as reference for H1pdm09 or H3 viruses, respectively.

Results: Complete genome of 40 laboratory-confirmed samples from ICU-admitted patients and of 45 laboratory-confirmed samples from non-hospitalised cases were sequenced. Many mutations in influenza H1pdm09 and H3 samples showing a frequency over 90% among viral variant population, which might be associated to virulence or reduced antiviral susceptibility, were found in both groups of patients. Among H1pdm09 viruses, mutations P83S and T197A in HA; V241I and N248D in NA; V100I in NP; N321K and A343T in PA; and, V344M and I354L in PB2 were found. Regarding H3 viruses, Y155F was found in NA and I588T in PB2. Other mutations in a viral frequency below 50% also related to pathogenicity were found, such as mutations T139I in MP, F103L in NS1 and S344L in NP for H1pmd09 viruses, as well as, mutations F450Gin NP, S245N in NA, and V255I in PB2.

Conclusions: Mutations related to higher virulence or reduced susceptibility to antiviral drugs were found, in several proportions in the mixed viral population of respiratory specimens. However, these

were in respiratory samples from both non-hospitalised and ICU-admitted patients, suggesting that they did not contribute to severity of disease. Therefore, the possibility that other host factors might be related to worse or fatal outcome cannot be excluded.