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A glycosylation near antigenic site A in H7 hemagglutinin determines H7 influenza virus sensitivity to neutralizing antibodies

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Human infections with H7 subtype of influenza virus have been reported, including an A(H7N7) outbreak in Netherlands in 2003 and A(H7N9) infection in China in 2013. We previously isolated three murine monoclonal antibodies (mAbs), 5A6, 4A2 and 2C4 that target the antigenic site A of H7 hemagglutinin (HA). To better understand the efficacy of H7 vaccines and vaccine candidate selection, we used our panel of mAbs to assess antigenic relatedness among H7 hemagglutinins and residues of HA that affect the susceptibility to neutralization. We found that these mAbs neutralized wild type A/Shanghai/02/2013 HA-pseudovirus, but not A/Netherlands/219/2003. Furthermore, the glycosylation of residue aspartic acid at 141 (N141) in A/Netherlands/219/2003 HA is responsible for this resistance. The glycosylation of N141 is due to A143T substitution in A/Netherlands/219/2003 HA. This mutation of residue 143 also plays an important role in infectivity of the HA-pseudoviruses. The A143 decreases the HA-pseudovirus infectivity. These results demonstrate that residue 143 not only affects H7 sensitivity to neutralizing antibodies, but also viral infectivity.

Biography

Esmeralda Alvarado-Facundo completed her PhD in 2013 from Instituto Politecnico Nacional (Mexico) in collaboration with CBER, US FDA. She is doing post-doctoral studies at CBER-FDA studying influenza virus hemagglutinin and its influence on virus infection, immunogenicity, and susceptibility to neutralizing antibodies.

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