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Inactivation of influenza virus haemagglutinin by chlorine dioxide: oxidation of the conserved tryptophan 153 residue in the receptor-binding site

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Abstract

Airborne influenza virus infection of mice can be prevented by gaseous chlorine dioxide (ClO(2)). This study demonstrated that ClO(2) abolished the function of the haemagglutinin (HA) of influenza A virus (H1N1) in a concentration-, time- and temperature-dependent manner. The IC(50) during a 2 min reaction with ClO(2) at 25 °C was 13.7 μM, and the half-life time of HA with 100 μM ClO(2) at 25 °C was 19.5 s. Peptides generated from a tryptic digest of ClO(2)-treated virus were analysed by mass spectrometry. An HA fragment, (150)NLLWLTGK(157) was identified in which the tryptophan residue (W153) was 32 mass units greater than expected. The W153 residue of this peptide, which is derived from the central region of the receptor-binding site of HA, is highly conserved. It was shown that W153 was oxidized to N-formylkynurenine in ClO(2)-treated virus. It was concluded that the inactivation of influenza virus by ClO(2) is caused by oxidation of W153 in HA, thereby abolishing its receptor-binding ability.

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