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The NS1 protein of influenza A virus strain SC35 confers virulence in adult chickens

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We analyzed the contribution of the NS1 gene to virulence of laboratory influenza A virus strain SC35 (H7N7) by infecting 6-week-old white leghorn chickens intratracheally with 106 plaque-forming units of either wild-type SC35 or mutant SC35-delNS1. All animals infected with wild-type SC35 developed severe disease within 5-6 days. They did not transmit the virus to cage mates, although it was excreted as evidenced from RT-PCR-positive tracheal and cloacal swabs on days 2 to 6 post infection. All chickens infected with SC35-delNS1 remained healthy, did not excrete virus at any time post infection, and seroconverted within 8 days of infection. Lung pathology at day 3 post infection was far more prominent in chickens infected with wild-type SC35 than SC35-delNS1. At day 3 post infection, lungs of chickens infected with wild-type SC35 contained higher levels of type I interferon (IFN) and other cytokines than lungs of birds infected with SC35-delNS1. At 16 hours post infection, SC35-delNS1 was also a surprisingly poor inducer of cytokines in chicken lungs. The latter result was unexpected, given the fact that the NS1-deficient mutant virus is a potent IFN inducer in cell culture and lungs of mice. We presently evaluate the possibility that the NS1 protein of avian influenza viruses is primarily not acting as IFN antagonist in chicken lungs but rather contributes to virulence by another as yet undefined mechanism.

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