Adamantine resistance increasing in the US

There has been an increase in the frequency of amantadine and rimantadine resistance among circulating influenza A viruses in the US "over the past few years", representing a need for rapid surveillance for antiviral resistance, according to researchers from the US.¹

Early during the US 2005–2006 influenza season, they used a pyrosequencing method to detect mutations in the M2 gene of isolates collected from patients in 26 states.

Of 209 influenza A H3N2 virus isolates analysed, 193 (92.3%) contained a serine to asparagine point mutation at amino acid 31 (S31N), a change that conferred resistance to amantadine and rimantadine; six of these isolates also contained a valine to isoleucine change at amino acid 27. Adamantine resistance was widespread across the US. Additional isolates from Mexico and Canada showed 100% resistance to amantadine and rimantadine.

In an editorial accompanying the study, Drs David M Weinstock and Gianna Zuccotti from Memorial Sloan-Kettering Cancer Center, New York, US, state that the response to this increase in adamantine resistance needs to be "global and immediate".² They also suggest that, "if antiviral use is curtailed, susceptible strains could emerge and adamantanes could regain their utility against both epidemic and pandemic influenza".

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Bright RA, et al. Adamantane resistance among influenza A viruses isolated early during the 2005-2006 influenza season in the United States. JAMA: the Journal of the American Medical Association 295: 891-894, No. 8, 22 Feb 2006

^{2.} Weinstock DM, et al. Adamantane resistance in influenza A. JAMA: the Journal of the American Medical Association 295: 934-936, No. 8, 22 Feb 2006.