

## The 1918 Spanish Influenza Pandemic

Lai KY\*, George WYN and Fanny FC

Department of Intensive Care, Queen Elizabeth Hospital, Hong Kong

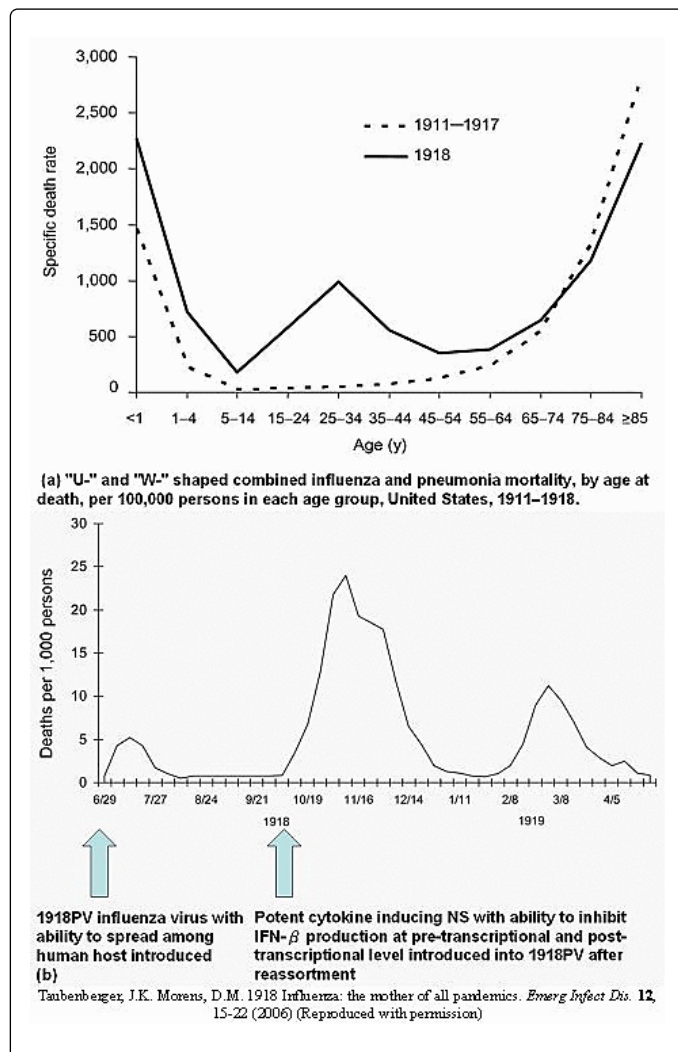
\*Corresponding author: Lai KY, Department of Intensive Care, Queen Elizabeth Hospital, Hong Kong, Tel: 852-2958-8888; E-mail: laiky@ha.org.hk

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### Short Report

1918 H1N1 Spanish influenza pandemic virus (1918PV) killed 4 million people. The first wave of the 1918 H1N1 Spanish influenza pandemic had a mortality comparable to the usual seasonal influenza with a U-shaped mortality-age distribution that involved the very young and elderly. However, the second wave of the 1918 H1N1 Spanish influenza pandemic had a dramatic surge in mortality and a W-shaped mortality-age distribution that involved young adults with a distinct peak of death in individuals between 20 and 40 years of age (Figure a and b) [1].



Analysis of individual gene segment of the 1918PV isolated from archived samples of the 1918 H1N1 Spanish influenza pandemic's second wave showed that the non-structural gene segment (NS) contributed to the virulence of 1918PV due to its ability to induce cytokine dysregulation and inhibit human inducible pre-transcriptional interferon-beta (IFN- $\beta$ ) production [2] and post-transcriptional maturation and nuclear export of host interferon-related mRNAs [3]. The NS of the 1918PV entered the swine population in 1918 and re-emerged in the 2009 novel H1N1 pandemic virus (2009PV). The 2009PV is able to induce cytokine dysregulation and produced an enhanced mortality with a W-shaped mortality-age distribution that involved the young population. Adaptation in pigs has abolished the ability of the NS of 2009PV to block interferon production at the post-transcriptional level and may account for reduced mortality of 2009PV infection compared with the original 1918PV [3].

With new information from the 2009PV, the surge in mortality and the W-shaped mortality in the second wave of the 1918 Spanish influenza pandemic may be due to the reassortment of a potent cytokine inducing NS with the ability to suppress human interferon production at both the pre-transcriptional and post-transcriptional level into the 1918PV after the first wave (Figure b).

### References

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