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***Host-parasite coevolution***
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**Influenza A virus avian-derived PB1 gene has evolved to match its codon usage to interferon-altered human tRNA pools**
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**Abstract:** Influenza A viruses cause an annual highly contagious respiratory disease in humans, and are responsible for periodic human pandemics that have high mortality rates. Pandemic influenza A viruses can result from reassortment of one or more gene segments between a human and an avian virus. These avian virus gene segments need to adapt to humans post introduction. The role of synonymous mutations in this adaptation is not known. Here we focus on the human adaptation of the synonymous codons of the avian virus PB1 gene of the 1968 H3N2 pandemic virus. We generate recombinant H3N2 viruses differing only in codon usage of PB1 mRNA, and demonstrate that the codon usage of recent virus isolates enhances replication in interferon-treated human cells rather than in untreated cells, thereby partially alleviating the interferon-induced antiviral state. High-throughput sequencing of tRNA pools explains this virus phenotype: the levels of some tRNAs differ between interferon-treated and untreated human cells; and the codon usage of H3N2 PB1 mRNA has been evolving over time to match the tRNA pools in interferon-treated human cells. Consequently, our results identify a previously unknown mechanism by which influenza A virus counteracts the host interferon-induced antiviral response and highlight the important role of tRNA pools in the regulation of gene expression.

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