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## Transmission of influenza A in human beings

We read with interest the Review by Gabrielle Brankston and colleagues<sup>1</sup> on the transmission of influenza A, but were disappointed to find it very biased against any evidence presented in favour of the airborne transmission of influenza.

One surprising example of this was the authors' discussion of the classic study by Moser and co-workers,<sup>2</sup> which they dismiss in a single sentence: "because of the free movement of passengers throughout the aircraft, close range transmission of influenza through droplet or direct contact could not be ruled out".<sup>1</sup> Although we do not dispute the relevance and presence of this phrase in the original study, many other papers (including the reply by Tellier<sup>3</sup> to recent criticism of his earlier Review<sup>4</sup>) have cited this particular study as more supportive than not, of the airborne transmission of influenza.<sup>5</sup> What makes the interpretation of this study by these other authors any less accurate than that of Brankston and colleagues?<sup>2</sup>

Another example of bias against evidence of airborne transmission in Brankston and co-workers' Review is their discussion about whether the ferret is a good model for human influenza. In fact, the ferret is now one of the preferred small animal models for studying human influenza in terms of pathogenesis and transmission.<sup>6</sup> Are the authors now saying that these other researchers are using an inappropriate model for studying human influenza?

Regarding droplet dynamics, it is likely that the use of respiratory-assist devices, such as high-flow (up to 10–15 L/min) oxygen masks and mechanical ventilation, is likely to increase the potential risk of naturally (as opposed to artificially) produced aerosols containing influenza, as was suggested during the severe acute respiratory syndrome (SARS) outbreaks of 2003.<sup>7,8</sup> Also, at one point, Brankston and colleagues seem to underinterpret one of their own references,<sup>9</sup> in which particles of 6–10 µm diameter are listed as being able to remain suspended for "several hours" while falling a height of 3 m during which "deposition in nasal passages" is possible. Even accepting their statement that coughing mostly produces particles greater than 8 µm, this does not preclude coughed particles of sizes 8–10 µm being able to remain suspended and transmit infection over long distances. They themselves admit that "there is no exact particle size cut-off at which pathogen

transmission changes from exclusively droplet to airborne, or vice versa". The exact proportion of different sized droplets produced in a cough will differ between individuals in different situations. After the droplets have left the mouth, their size will also be affected by the ambient temperature and relative humidity. For these reasons at least, Brankston and colleagues' generalisation that coughed particles are too large to sustain airborne influenza transmission is unacceptable.

We realise that the practical and economical consequences of accepting influenza as an airborne infection are significant. However, this should not make us deliberately downplay or underinterpret any data that are supportive of this route of transmission. Hence, we would offer the counterpoint that this issue is not closed and echo Oshitani<sup>10</sup> who stated that "the proportion of influenza infections that can be acquired by the airborne transmission is largely unknown". The potential role for airborne influenza transmission, therefore, still remains an important issue in pandemic influenza preparedness.

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