Supplemental Material: Other Viral Diseases

*Manuscript Title: Skin as a Potential Source of Infectious Foot and Mouth Disease Aerosols*

The biological plausibility of FMDV transmission via infectious skin cell is enhanced if a skin cell source of disease transmission has been established (or is likely) for other viruses. FMD is not the only viral pathogen for which (a) there is known skin trophism (e.g. rash or lesions), (b) the respiratory tract is known to be a significant (or dominant) infectious pathway, and (c) viral transmission is present co-incident with the skin trophism (often peaking with the skin trophism onset).¹ For example, airborne transmission of Marek’s disease (a herpesviridae affecting poultry) is known to be associated with desquamated epidermal cells shed from feather follicles [S1]. In addition, a number of human viruses,² from several virus families, are well known to share these traits, including herpesviridae, e.g. Varicella-Zoster (chickenpox) [S2]; poxviridae, e.g. Variola Major and Minor (smallpox) [S3]; togaviridae, e.g. Rubivirus (rubella), and paramyxoviridae, e.g. Measles [S4].

Published data on Varicella-Zoster Virus (VZV) is particularly relevant to the current discussion. VZV is the cause of chickenpox and reactivation of dormant viral infection later in life causes localized cutaneous herpes zoster. VZV is believed to be transmitted by direct contact via fomites contaminated by the infected serous exudate from ruptured skin vesicles, but an important secondary route of transmission is hypothesized to be the airborne route via infected skin scales [S2].³ VZV is detected in air samples taken from patient rooms and nearby locations. This is true both in room air samples for patients with widespread rashes (primary varicella) as well as in room air samples for cases presenting solely as a localized skin rash (reactivated local cutaneous herpes zoster) [S8,S9,S10,S11]. VZV DNA is also detected in environmental dust samples obtained up to 1.5 months after the clinical development of a rash [S11,S12]. Suzuki et al. [S10] demonstrated that when localized VZV rashes were covered with an impenetrable (hydrocolloid) dressing, viral samples from the patient’s throat, the ambient room air, and outer surface of the dressing were nearly universally negative for VZV. In contrast, the corresponding samples from patients using standard gauze dressings (which are not expected to retard skin aerosol emissions) were nearly universally positive. Earlier work [S9] indicated that the sequence of positive virus detections progressed first from the patient’s skin, then to ambient air samples and then to patient throat samples – suggesting that airborne VZV skin aerosols may be a source of disease transmission.

The data for variola major and minor (smallpox) is more circumstantial. The respiratory system is well-known to be the typical site of initial infection, but the aerosol generation pathway is not well understood [S3]. High viral levels are found in respiratory secretions during periods of high infectivity, suggesting a respiratory emission pathway. However, this period is also co-incident with the onset of the rash. Published studies [S13,S14] suggest that infectious aerosol emissions

¹ This screening criteria does not attempt to distinguish between infected material residing within or outside the skin cell aerosols (the latter would be expected from surface contamination via ruptured lesion).
² For human diseases, inhalation of virally infected skin cells may be a particularly efficient mechanism of disease transmission due to 1) the high (1 to 10%) fraction of indoor dust that is comprised of human skin fragments, 2) the large amount of time people spend indoors [S5], and 3) the known ability for large (>10 μm) aerosols to be inhaled by humans [S6].
³ The degree to which respiratory emissions contribute to the overall disease transmission in primary VZV infection is still a point of debate [S7].
are primarily associated with relatively large aerosols (skin cell size) and the disturbance of bedsheets (which would harbor skin cells). Air samples taken near patients’ mouths yielded relatively little virus. The composition of the carrier aerosol(s) has not been elucidated. We are unaware of a study that examined the concentration, lifetime, or infectivity of the variola virus in intact stratum corneum. However, it is well known that the variola virus can remain infectious for over 10 years in scab material, although scab-bound virus infectivity is low [S3].

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References:


