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1 **Combating SARS-CoV-2: Leveraging microbicidal experiences with other emerging/re-**
2 **emerging viruses**

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15
16 Keywords: 2019-nCoV, Coronavirus, Ebola virus, Enterovirus D68, Hantaan virus; Lassa virus,
17 microbicides, MERS-CoV, Nipah virus, SARS-CoV, SARS-CoV-2, SFTSV, targeted hygiene
18

19 **Abstract**

20 The emergence of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) in Wuhan
21 City, China, late in December 2019 is another example of an emerging zoonotic virus that
22 threatens public health and international travel and commerce. When such a virus emerges, there
23 is often insufficient specific information available on mechanisms of virus dissemination from
24 animal to human or from person to person, on the level or route of infection transmissibility or of
25 viral release in body secretions/excretions, and on the survival of virus in aerosols or on surfaces.
26 The effectiveness of available virucidal agents and hygiene practices as interventions for
27 disrupting the spread of infection and the associated diseases may not be clear for the emergent
28 virus. In the present review, we recommend approaches for infection prevention and control for
29 SARS-CoV-2 which can be invoked based on pre-existing data on microbicidal and hygiene
30 effectiveness for related and unrelated enveloped viruses.

31
32 Late in December 2019, cases of pneumonia began appearing in Wuhan City, Hubei Province,
33 China. By early January 2020, these cases were attributed to a novel coronavirus that was
34 temporarily referred to as 2019 Novel Coronavirus (2019-nCoV)¹. This member of the
35 *Coronaviridae* family has now officially been named SARS-CoV-2². As of April 17, 2020³,
36 there have been over 2,074,529 confirmed cases globally, with 139,378 deaths (mortality rate of
37 ~6.7%). This emerging virus, and the associated disease (COVID-19), are not only impacting
38 public health, but also international commerce and travel. As with the Middle East Respiratory
39 Syndrome coronavirus (MERS-CoV) that emerged in Saudi Arabia in 2012 and the Severe Acute
40 Respiratory Syndrome coronavirus (SARS-CoV) that emerged in China in early 2003, SARS-
41 CoV-2 is considered a zoonosis, with bats suspected as the primary host species (Table 1)⁴.

42 The *Coronaviridae* family is just one of several families of enveloped viruses that have
 43 emerged/re-emerged in recent years⁵⁻⁹ (Table 1). While the list of viruses in Table 1 is not
 44 intended to be comprehensive, it contains most of the virus families attributed to the World
 45 Health Organization’s current list of disease priorities needing urgent R&D attention¹⁰ (i.e.,
 46 MERS and SARS [*Coronaviridae*], Crimean Congo hemorrhagic fever (*Nairoviridae*), Rift
 47 Valley fever [*Phenuiviridae*], Ebola virus disease and Marburg virus disease [*Filoviridae*], Nipah
 48 and Hendra virus disease [*Paramyxoviridae*], and Lassa fever [*Arenaviridae*]).

49

Table 1. Characteristics of selected emerging/re-emerging viruses

Virus	Family	Particle size	Lipid Envelope	Genome* (segments)	Reservoir species	References
Lassa virus	<i>Arenaviridae</i>	110-130 nm	yes	±ssRNA(2)	rodent	6,61
SFTSV†	<i>Phenuiviridae</i>	80-100 nm	yes	-ssRNA(3)	tick	9
Hantaan virus	<i>Hantaviridae</i>	80-120 nm	yes	-ssRNA(3)	rodent	7,8,62
MERS-CoV	<i>Coronaviridae</i>	118-136 nm	yes	+ssRNA(1)	bat	2,18,63
SARS-CoV	<i>Coronaviridae</i>	80-90 nm	yes	+ssRNA(1)	bat	2,18,63
SARS-CoV-2	<i>Coronaviridae</i>	60-140 nm	yes	+ssRNA(1)	bat‡	4,15,64
Ebola virus	<i>Filoviridae</i>	80 × 14000 nm	yes	-ssRNA(1)	bat	61
Influenza H5N1	<i>Orthomyxoviridae</i>	80-120 nm	yes	-ssRNA(8)	avian	65
Nipah virus	<i>Paramyxoviridae</i>	40-1900 nm	yes	-ssRNA(1)	bat	5
EV-D68	<i>Picornaviridae</i>	~30 nm	no	+ssRNA(4)	unknown	16,66

50 *Abbreviations used: ±, ambisense; -, negative sense; +, positive sense; ss, single-stranded.
 51 Segments (1) equates to a non-segmented genome. †Now referred to as Huaiyangshan
 52 banyangvirus.‡Suspected primary host based on >90% sequence homology to bat coronaviruses
 53 ⁶³.

54

55 The emerging/re-emerging viruses shown in Table 1, with the exception of enterovirus D68,
 56 each are relatively large, enveloped, zoonotic viruses with single-stranded RNA genomes.
 57 Enterovirus D68 (EV-D68), a small non-enveloped virus of the *Picornaviridae* family is an
 58 example of a re-emerging virus from that family. While EV-D68, may also be zoonotic^{11,12}, a
 59 reservoir species has yet to be identified.

60 Aside from the characteristics described in Table 1, what other commonalities exist for these
 61 emerging/re-emerging zoonotic viruses? Can we use these commonalities as the basis for
 62 proposing approaches for infection prevention and control (IPAC)? In the remainder of this
 63 review, we examine various aspects of the emerging/re-emerging viruses that are important in
 64 formulating approaches for IPAC, namely transmissibility, infectivity, viral shedding,
 65 environmental survival, and expectations regarding microbicidal efficacy for targeted hygiene
 66 practices. This information may then be leveraged to effectively mitigate the health risks
 67 associated with SARS-CoV-2 and its associated disease (COVID-19), as well as with future
 68 emerging/re-emerging enveloped viruses.

69 **Transmissibility of Emerging/Re-emerging Viruses**

70 According to several authors^{13,15}, sustained person-to-person transmission of viruses is favored
 71 by certain viral characteristics, including lack of a lipid envelope, small particle size, limited

72 genomic segmentation, and low mortality of the associated disease¹³⁻¹⁵. Tropism of the virus for
 73 the liver, central nervous system (CNS), or the respiratory tract, and lack of vector-borne
 74 transmission also appear to favor sustained person-to-person transmission^{13,14}. On the other hand,
 75 possession of an RNA vs. a DNA genome was not found to contribute to the likelihood of such
 76 sustained transmission^{13,14}.

77 It is of interest that many of the viral characteristics mentioned above that are considered
 78 predictive of sustained person-to-person transmissibility are not shared by the viruses associated
 79 with the World Health Organization (WHO) diseases of concern¹⁰. Namely, all of the
 80 emerging/re-emerging diseases mentioned in the WHO list¹⁰ involve relatively large enveloped
 81 viruses with ssRNA genomes, many of which are segmented. Of the emerging/re-emerging
 82 viruses listed in Table 1, only EV-D68 is a small, non-enveloped virus. In addition, many of the
 83 listed viruses exhibit high human mortality (Tables 1 and 2).

84

Table 2. Transmission and mortality of emerging/re-emerging viruses

Virus	Tropism for organs	Mode of transmission*	Mortality	Reference
Lassa virus	Vascular system	Contact, aerosols	15-20%	25,61
SFTSV	Vascular system	Vector (tick)	12-30%	9,67
Hantaan virus	Lower respiratory, renal	Contact, Aerosols	1-15%	61,62,68,69
MERS-CoV	Lower respiratory, GI	Contact, aerosols	34-36%	25,42,70,71
SARS-CoV	Lower respiratory	Contact, aerosols	15 ± 11%	25,72
SARS-CoV-2	Lower respiratory, GI	Contact, aerosols	7%	3,19
Ebola virus	Vascular system	Contact, aerosols	41%	25,42,73
Influenza H5N1	Upper respiratory	Contact, aerosols	>60%	25,74
Nipah virus	CNS, respiratory	Contact, ingestion	65 ± 28%	5,27
EV-D68	Respiratory, CNS	Aerosols, contact	Up to 10%	66,75

85 *”Contact” refers to contact with bodily fluids or with fomites; “aerosols” equates to respiratory
 86 aerosols/droplets. CNS, central nervous system, GI, gastrointestinal

87

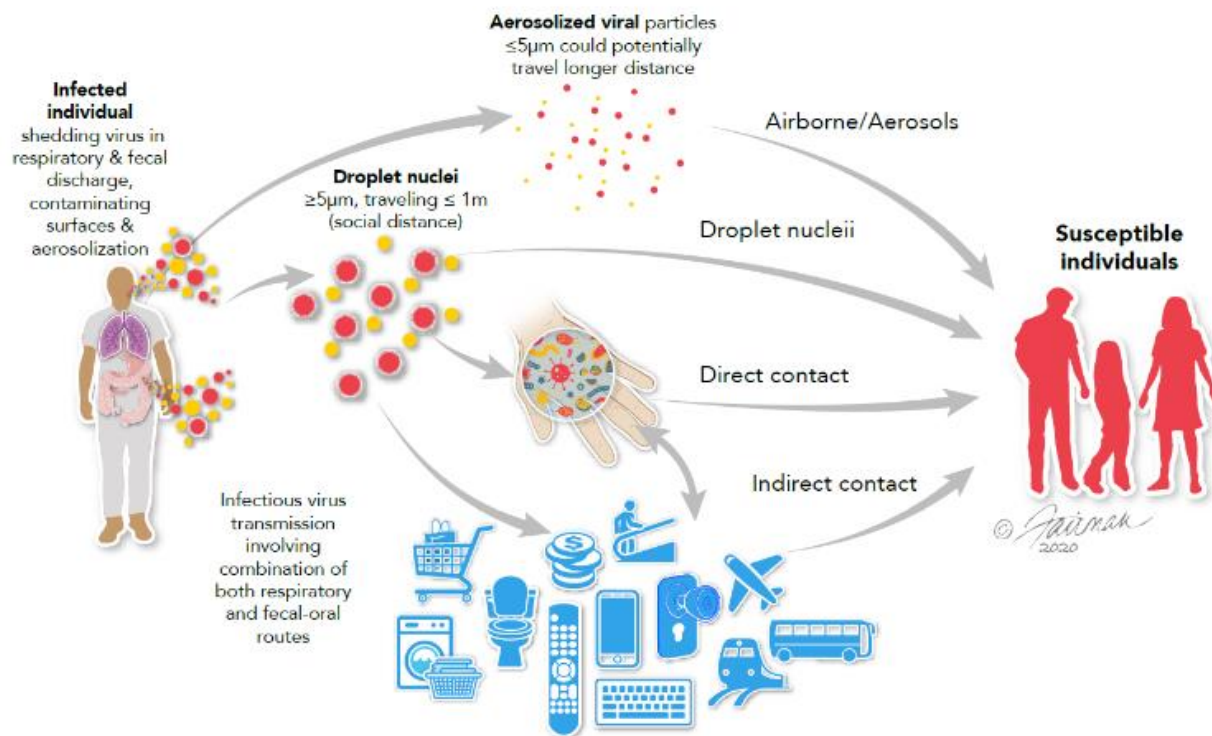
88 Certain predictive factors¹³⁻¹⁵ that do seem to be shared by the emerging/re-emerging viruses in
 89 the list in Table 1 include tropism for the respiratory tract or the CNS, and lack of vector-borne
 90 transmission. While most enteroviruses are less susceptible to acid and are disseminated by the
 91 fecal-oral route, EV-D68 is acid-labile and has a lower temperature optimum, reflecting its
 92 tropism for the upper respiratory tract rather than the gastrointestinal tract (i.e., EV-D68 acts
 93 more like a rhinovirus than an enterovirus)¹⁶.

94 It is unknown if sustained person-to-person transmissibility necessarily equates to a high level
 95 of concern for an emergent zoonotic virus. For instance, there appears to be no evidence that
 96 Hendra virus (another zoonotic enveloped virus) has shown person-to-person transmission¹⁷, yet
 97 this virus is similar to Nipah virus in many respects and is of concern, due its high mortality rate
 98 in humans.

99 As mentioned in Table 2, the most common modes of transmission for the emerging/re-
 100 emerging viruses discussed in this review are contact with infected bodily secretions/excretions
 101 and contaminated fomites, especially high-touch surfaces (HITES), and inhalation of respiratory

102 droplets/aerosols containing infectious virus (Fig 1). The intermediacy of hands in transmission
103 through contact is emphasized in Fig. 1.

104



105

106 **Figure 1.** Modes of transmission of viruses, emphasizing respiratory infections such as SARS-
107 CoV, MERS-CoV, and SARS-CoV-2 (modified from Otter et al.¹⁸).

108

109 The animal-to-human and person-to-person transmission of SARS-CoV-2 and associated
110 COVID-19 disease appears to occur in a manner similar to that described for MERS-CoV and
111 SARS-CoV.

112 **Infectivity and Virus Shedding of Emerging/Re-emerging Viruses**

113 The infectivity of a virus refers to its ability to initiate infection of host cell with production of
114 viral progeny. The infectious dose₅₀ (ID₅₀) is the smallest number of infectious virus particles
115 that will lead to infection of 50% of an exposed population²⁰, and is dependent a number of
116 factors, such as the species, age, or race of the host, the receptor, immune, and nutritional status
117 of the host or host tissues, and the portal of entry of the virus. In the case of most viruses, only a
118 percentage of those infected actually develop clinical illness²¹. Those who remain asymptomatic
119 represent subclinical cases of the infection in whom the virus may still replicate and be released
120 into the environment. IPAC may be difficult in the face of such silent disseminators (virus
121 carriers/shedders). Exposure to as low as one infectious viral particle has a probability of causing
122 an infection leading to disease, although that probability varies from virus to virus²². Typically,
123 infectious doses are empirically derived and reported in units of 50% infective dose (ID₅₀) values

124 that reflect the doses capable of infecting half of the subjects exposed. As prospective studies in
125 humans of highly pathogenic viral diseases with potentially fatal outcomes cannot ethically be
126 performed, very limited data exist on the infectivity of the emerging/re-emerging viruses in
127 Table 1. Where studies have been performed using animals, extrapolations of such data to
128 humans must be made with caution.

129 The estimates that have been reported for viruses listed in Tables 1 and 2 are discussed below,
130 acknowledging the unavoidable variability in literature with regard to such assessments of
131 infectivity. It has been stated that 1-10 infectious aerosolized Ebola virus particles can cause an
132 infection in humans^{23,24}. A similar range has been reported for the Lassa virus²⁵. Influenza virus
133 infectivity values specific to the H5N1 and H7N9 strains are not known²² but estimates of 100 to
134 1000 infectious viral particles have been reported^{22,25}. The human infective dose for SARS-CoV
135 has been estimated at 16 to 160 plaque-forming units²⁶. Data on the human infectious doses for
136 MERS-CoV, SFTSV, Nipah virus, and EV-D68 have not been reported. Until such data become
137 available, it should be assumed that these emerging/re-emerging viruses, including SARS-CoV-
138 2, have relatively low ID₅₀ values.

139 Once infected with one of these emerging/re-emerging viruses, during the prodromal period
140 before actual appearance of symptoms, as well as once symptoms appear, the infected individual
141 may become a shedder of infectious particles. The extent to which virus shedding might lead to
142 dissemination of the associated disease depends upon a number of factors, including the amount
143 of virus released (shed), the infectivity of the virus within the released matrix (droplets/aerosols,
144 fecal/diarrheal discharge, and other excretions including respiratory secretions), and the survival
145 of the released viruses within such matrices once dried on HITES. Extent of virus shedding,
146 unfortunately, is commonly measured through detection of genomic material^{e.g., 18,22,27,28}, rather
147 than through use of infectivity assays, so there are only limited data available on infectious viral
148 shedding.

149 As displayed in Fig. 1, transmission of respiratory infections commonly involves the
150 intermediacy of the hand. The same can be said about gastrointestinal infections (i.e., through the
151 fecal-oral route). The SARS-CoV, MERS-CoV, and SARS-CoV-2 have been reported^{18,19} to be
152 shed from patients both within respiratory and gastrointestinal secretions/excretions, therefore it
153 is likely that contaminated HITES and respiratory droplets/aerosols also play an important role in
154 dissemination of the SARS-CoV-2 through the intermediacy of hands.

155 **Viral Survival on Environmental Surfaces and in Air**

156 Knowledge of the transmissibility and infectivity of emerging/re-emerging viruses enables one to
157 assess the risk of spread of a viral disease in the case that infectious virus is shed from an
158 infected individual and is deposited on environmental surfaces/fomites or in droplets/aerosols.
159 Another important factor to consider when assessing risk is the survival (i.e., the continued
160 infectivity) of these viruses on the environmental surfaces/fomites or in air in the form of
161 droplets/aerosols.

162 There is much more information addressing survival of infectious viruses on environmental
163 surfaces than in aerosols. The data that are available address a number of environmental factors
164 of relevance¹⁸, including the types and porosities of the surfaces, the matrices in which the
165 viruses have been suspended prior to being deposited onto the surfaces the temperature and
166 relative humidity (RH), and methods used for measuring survival (e.g., log₁₀ reduction in

167 infectivity per unit time, infectivity half-life, etc., infectious titer after a measured duration, etc.).
 168 For Table 3, the results that have been displayed focus on room temperature (ambient) conditions
 169 at relatively low and medium RH. Table 3 should not, therefore, be considered to represent a
 170 comprehensive review of literature for survival of these viruses. For a more systematic review of
 171 coronaviruses survival on environmental surfaces under various conditions, see the reviews by
 172 Otter et al.¹⁸ and Kampf et al.²⁹. For certain viruses (e.g., SFTSV), survival data are not yet
 173 available, so data for surrogate viruses from the same or similar families are shown. Persistence
 174 of SARS-CoV-2 on surfaces and in air has recently been reported by van Doremalen et al.³⁰
 175 SARS-CoV-2 was found to remain viable in aerosols for at least 3 hours, and for up to 24 hours
 176 on cardboard and 2 to 3 days on plastic and stainless steel surfaces³⁰.

177

Table 3. Environmental survival of emerging/re-emerging viruses at room temperature

Virus	Survival on surfaces	Survival in aerosols	Reference
Lassa virus	0.41 log ₁₀ /d (glass)	t _{1/2} = 0.62 h	76,77
SFTSV	t _{1/2} = 0.75 h (aluminum)†	No data	78
Hantaan virus	t _{1/2} = 1.0 h (aluminum)	No data	78
MERS-CoV	t _{1/2} = 0.94 h (steel)	t _{1/2} = 27 h*	31,71
SARS-CoV	t _{1/2} = 10 h (steel), 18 h (plastic)	At least 3 h¶	30,72
SARS-CoV-2	t _{1/2} = 13 h (steel), 16 h (plastic)	At least 3 h¶	30
Ebola virus	0.68 log ₁₀ /d (glass) 0.88 log ₁₀ /d (steel)	t _{1/2} = 0.25 h	39,73,76,79,80
Influenza H5N1	<1 d (glass, metal)	No data	65
Nipah virus	1 h (plastic)	No data	79
EV-D68	t _{1/2} = 0.17 to 0.25 h (steel)‡	No data	81

178 †No data for SFTSV are available, the result displayed is for Crimean-Congo virus.

179 *Aerosol data for coronavirus 229E³¹. Survival half-life depended on humidity. The values
 180 ranged from 3.3 h (~80% RH), 67 h (50% RH), to 27 h (30% RH).

181 ‡No data for EV-D68 are available; the result displayed is for human rhinovirus type 14 at 15-
 182 55% RH⁶⁵.

183 ¶The authors only evaluated times up to 3 h.

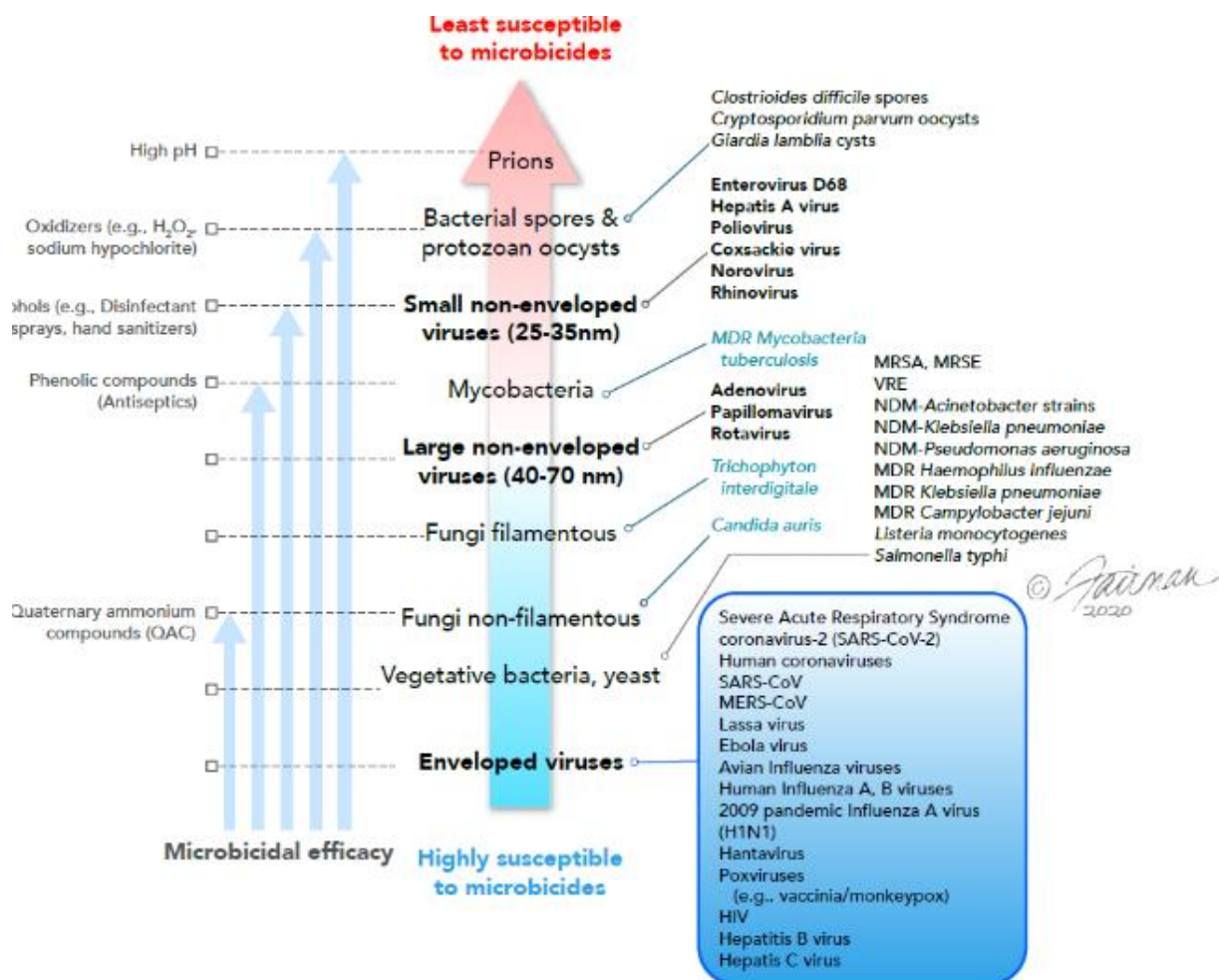
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185 Hierarchy of Microbicidal Efficacy for Inactivating Pathogens

186 Infectious virus survival in aerosols or on HITES represents a source for dissemination of
 187 emerging /re-emerging viruses, including SARS-CoV-2. The enveloped viruses listed in Tables 1
 188 and 2 should be relatively susceptible to the inactivating activity of a variety of microbicides, as
 189 discussed below. Sattar³² previously has advanced the concept of utilizing the known knowledge
 190 of the susceptibility human viral pathogens to chemical disinfecting agents (microbicides)^{33,34} to
 191 predict the efficacy of such agents for inactivating emerging /re-emerging viral pathogens. This
 192 concept, referred to as a hierarchy of susceptibility to microbicides^{32,33}, is portrayed in Fig. 2. As
 193 shown, pathogenic agents can be viewed as displaying a continuum of susceptibilities to
 194 microbicides, with enveloped viruses at the bottom of this hierarchy, highlighting their relatively
 195 high susceptibilities to formulated microbicides^{32,33}.

196 Among pathogens, prions are considered to be the least sensitive to microbicides, requiring
 197 highly caustic solutions for inactivation. Bacterial spores and protozoan cysts/oocysts are next on
 198 the microbicidal susceptibility spectrum. Small, non-enveloped viruses are considered to be less
 199 susceptible to microbicides, although have increased susceptibility to high pH, oxidizers such as
 200 sodium hypochlorite, formulated hydrogen peroxide, alcohols, and a variety of microbicidal
 201 actives, relative to spores and protozoan cysts/oocysts. Mycobacteria, fungi, vegetative bacteria
 202 and enveloped viruses appear to be less resistant to certain formulated microbicides, such as
 203 alcohols, oxidizers, quaternary ammonium compounds (QAC), and phenolics (e.g., p-chloro-m-
 204 xylenol)^{29,32-45}.

205



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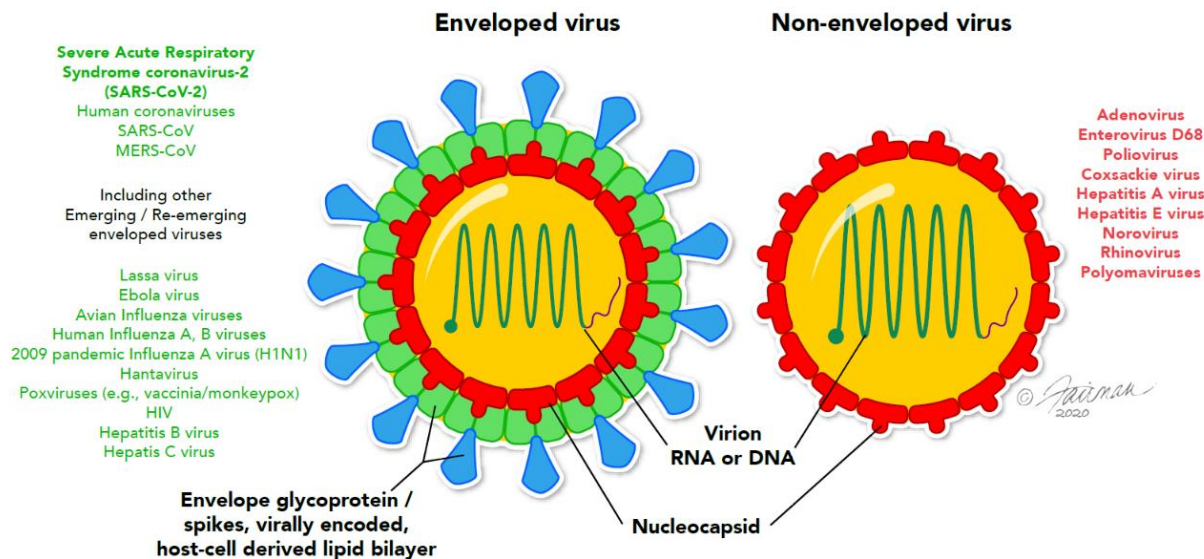
207 **Figure 2.** Hierarchy of sensitivity of pathogens to formulated chemical microbicides (adapted
 208 from Sattar³²). Data are from a variety of sources^{29,32-45}.

209

210 It is of interest that the enveloped viruses are considered to be the most susceptible to a
 211 variety of formulated microbicidal actives, even more so than fungi and vegetative bacteria,
 212 yeast, and mycobacteria (Fig. 2). Viral envelopes are typically derived from portions of the host

213 cell membrane and therefore comprise host cell phospholipids and proteins (Fig. 3), as well as
 214 some virally inserted glycoproteins. Since the envelopes contain lipid material, they are readily
 215 destroyed by phenolics such as p-chloro-m-xlenol (PCMX), oxidizing agents such as sodium
 216 hypochlorite and activated hydrogen peroxide, quaternary ammonium compounds, alcohols, and
 217 detergents. Even mild detergents such as soap may inactivate enveloped viruses by denaturing
 218 the lipoproteins in the envelope. This makes them more susceptible to most of the formulated
 219 virucidal microbicides commonly used for IPAC.

220



221

222

223 **Figure 3.** Ultrastructural differences between enveloped and non-enveloped viruses.
 224 Genotypically, these viral genomes may be single-or double-stranded, and segmented or non-
 225 segmented (examples are not shown in the figure).

226

227 It can be assumed as a starting point, therefore, that the enveloped emerging/re-emerging
 228 viruses listed in Table 1 should be readily inactivated by a variety of formulated microbicidal
 229 actives. This assumption has, in fact, been verified by extensive empirical data^{29,32-40,42-44}, and
 230 has been embraced by the U.S. Environmental Protection Agency⁴⁶. The data for various
 231 members of the *Coronaviridae* family have recently been reviewed by Kampf et al.²⁹, and these
 232 data support the expectation that SARS-CoV-2 and other coronaviruses of concern (e.g., MERS-
 233 CoV, SARS-CoV, mouse hepatitis virus, porcine epidemic diarrhea virus, etc.) should be readily
 234 inactivated by commonly employed and commercially available microbicides, including QAC.
 235 In addition, a European guidance document⁴⁷ recently has been issued that lists a variety of
 236 microbicidal agents that have demonstrated efficacy against a variety of human and animal
 237 coronaviruses, and that, therefore, could be applied for decontamination of surfaces in non-
 238 healthcare facilities.

239 Aqueous solutions of the phenolic, PCMX, at concentrations of 0.12 -0.48% by weight were
240 shown to inactivate $>4 \log_{10}$ of infectious Ebola virus - Makona variant (EBOV/Mak) suspended
241 in an organic load and evaluated in liquid inactivation studies^{43,48} or dried on a steel surface in a
242 carrier inactivation study⁴⁸. In each case, complete inactivation of $\geq 6.8 \log_{10}$ of EBOV/Mak was
243 observed after contact times ≥ 5 min. In addition, EBOV/Mak dried on prototypic steel carriers
244 was completely inactivated ($\geq 6.5 \log_{10}$) by aqueous solutions of 70% ethanol or 0.5% or 1%
245 NaOCl ($\geq 0.5\%$) after contact times ≥ 2.5 min³⁹.

246 Microbicidal formulations based on oxidizing agents, QAC, alcohols, phenolics, and
247 aldehydes displaying virucidal efficacy for enveloped viruses and relatively less susceptible non-
248 enveloped viruses (such as human norovirus surrogates) have been recommended for
249 decontaminating environmental surfaces or materials used for food preparation^{49,50}. The efficacy
250 of ethanol and QAC actives for inactivating the norovirus surrogate feline calicivirus depends on
251 how the microbicides are formulated. Factors such as the addition of an alkaline agent were
252 found to increase their efficacy⁵¹. Microbicides satisfying these requirements can be regarded as
253 effective against emerging/re-emerging viruses such as SARS-CoV-2. Following this logic, the
254 U.S. EPA has invoked an Emerging Viral Pathogen Policy in the past for the 2009 pandemic
255 influenza, for the Ebola virus, and most recently for SARS-CoV-2⁵².

256 In the case of highly pathogenic emerging/re-emerging viruses such as SARS-CoV-2,
257 effective and frequent targeted hygiene using appropriate microbicides is essential for prevention
258 of dissemination. Practicing hygiene inappropriately and only once daily may not be sufficient.
259 For instance, infectious coronavirus 229E was detected on HITES (e.g., door knobs) in a
260 university classroom in which samples were collected daily over a one-week period⁵³. Vigilant
261 decontamination of HITES becomes of paramount importance when dealing with highly
262 pathogenic viruses with relatively low human infectious doses, as is the case with many of the
263 emerging/re-emerging viruses, including SARS-CoV-2, being discussed in this review.

264 The enveloped emerging/re-emerging viruses listed in Table 1 display high susceptibility to
265 inactivation by ultraviolet light at 254 nm, an inactivation approach amendable to inactivation of
266 aerosolized viruses³¹. For instance, empirical data⁵⁴ for Lassa virus, Hantavirus, and Ebola virus,
267 and for the virus families (*Coronaviridae*, *Orthomyxoviridae*, *Paramyxoviridae*, *Phenuiviridae*)
268 indicate that UV fluencies of 3 to 14 mJ/cm² should inactivate 4 \log_{10} of the enveloped viruses in
269 Table 1. These fluency values are relatively low, compared to those needed to inactivate 4 \log_{10}
270 of the least susceptible viruses, such as those of the *Adenoviridae* (98-222 mJ/cm²) and
271 *Polyomaviridae* (235-364 mJ/cm²) families of non-enveloped viruses⁵⁵.

272 **Personal Hygiene Practices for Preventing Infectious Virus Acquisition**

273 A 2005 study on SARS-CoV indicated the presence of genomic material for that virus in air and
274 on HITES within a SARS patient's room⁵⁶, indicating the likelihood of airborne droplet
275 transmission for this coronavirus. This suggests that appropriate respiratory protection, as well as
276 targeted HITES decontamination and hand hygiene, represent important interventional practices
277 for limiting virus dissemination during outbreaks such as that occurring now with SARS-CoV-2.

278 The WHO has posted on their website a webpage entitled Coronavirus Disease (COVID-19)
279 advice for the public⁵⁷. Basic protective measures against SARS-CoV-2 recommended by the
280 WHO⁵⁷ include: frequent hand washing with soap and water or an alcohol-based rub, and
281 maintenance of social distancing (at least 1m, see Fig 1) especially in the presence of people who

282 are coughing, sneezing, or have a fever. The latter recommendation is applicable to any of the
283 viruses listed in Table 2 for which transmission by respiratory aerosols is expected. Avoidance of
284 touching eyes, nose, mouth, or other mucus membranes with hands post-contact with HITES is
285 also recommended⁵⁷. As displayed in Fig 1, the hands play an important role in transfer of
286 infectious virus from contaminated HITES to susceptible host's mucus membranes, enabling
287 virus-host interactions initiating infection. Following the appropriate hygiene practices described
288 above can potentially help in prevention and control of emerging and re-emerging viruses,
289 including the currently circulating SARS-CoV-2.

290 Discussion

291 As Dr. Anthony Fauci eloquently stated in 2005⁵⁸ “Public health officials once suggested that it
292 might someday be possible to ‘close the book’ on the study and treatment of infectious diseases.
293 However, it is now clear that endemic diseases as well as newly emerging ones (e.g., severe
294 acute respiratory syndrome [SARS]), reemerging ones (e.g., West Nile virus), and even
295 deliberately disseminated infectious diseases (e.g., anthrax from bioterrorism) continue to pose a
296 substantial threat throughout the world.” Recent experience certainly verifies these predictions.
297 Weber et al.⁴¹ have correctly emphasized that “Preventing disease acquisition via person-to-
298 person transmission or contact with the contaminated environment depends on rapid and
299 appropriate institution of isolation precautions, appropriate hand hygiene, and appropriate
300 disinfection of medical equipment, devices, and the environmental surfaces. Importantly, once
301 the nature of the emerging infectious agent is known (i.e., enveloped virus, bacteria, fungi,
302 nonenveloped virus, mycobacteria, or non-enveloped virus), it is possible to determine the
303 appropriate hygienic interventions. For example, an enveloped virus (e.g., Ebola, MERS-CoV)
304 or vegetative bacterium (e.g., CRE, MRSA) would be inactivated by any formulated
305 microbicial active(s) known to be effective against vegetative bacteria, filamentous fungi,
306 mycobacteria, or non-enveloped viruses.”⁴¹.

307 It is fortunate, though perhaps a little perplexing, that so many of the emerging/re-emerging
308 viruses (examples listed in Table 1 and below) are enveloped viruses. It is not clear why there are
309 not more small, non-enveloped viruses mentioned in the WHO list of viral diseases of concern¹⁰.
310 The small non-enveloped viruses are much less susceptible to commonly employed cleaning
311 agents (antiseptics, detergents, non-formulated microbicial actives) and, in general, display
312 relatively longer survival on environmental surfaces. According to theoretical modeling of
313 sustained person-to-person transmissibility¹²⁻¹⁴, small non-enveloped viruses are predicted to be
314 more likely to lead to sustained infections within the community. The reality is that the
315 emerging/re-emerging viruses of concern, both in humans and in economically important
316 animals, have more typically included enveloped viruses. Recent examples include porcine
317 epidemic diarrhea virus, MERS-CoV, SARS-CoV and SARS-CoV-2 (*Coronaviridae*), African
318 swine fever virus (*Asfarviridae*), Schmallenberg virus (*Peribunyaviridae*), Crimean-Congo
319 hemorrhagic fever virus (*Nairoviridae*), Rift Valley fever virus (*Phenuiviridae*), and West Nile
320 virus and Zika virus (*Flaviviridae*), Hantaviruses (*Hantaviridae*), and Lassa viruses
321 (*Bunyaviridae*).

322 The fact that the emerging/re-emerging viruses are predominantly RNA viruses might be
323 explained in part by the notion⁵⁹ that RNA viruses can more readily adapt to the rapidly changing
324 global and local environment due to the high error rate of the polymerases that replicate their

325 genomes. The RNA viruses are thought therefore to display higher evolution rates through
326 mutation, gnomonic reassortment, or recombination.

327 The likelihood of experiencing future emergent zoonotic viruses is high⁶⁰, and therefore
328 defining in advance appropriate approaches for limiting spread of such viruses through IPAC is
329 essential. We now have the sequencing tools necessary for rapidly identifying a novel virus such
330 as SARS-CoV-2, the genetic sequence of which was determined within just over one week⁴.
331 Provided that a novel emerging virus is found to be a member of a viral family for which we
332 have sufficient microbicidal data available, including IPAC expertise, it should be possible to
333 make accurate predictions as to viral transmission, survival on surfaces, and microbicidal
334 efficacy. SARS-CoV-2 is no exception in this regard.

335

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556 **Declarations**

557

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