



HAL
open science

Modelling the inactivation of viruses from the Coronaviridae family in response to temperature and relative humidity in suspensions or surfaces.

Laurent Guillier, Sandra Martin-Latil, Estelle Chaix, Anne Thébault, Nicole Pavio, Sophie Le Poder, Christophe Batéjat, Fabrice Biot, Lionel Koch, Don Schaffner, et al.

► To cite this version:

Laurent Guillier, Sandra Martin-Latil, Estelle Chaix, Anne Thébault, Nicole Pavio, et al.. Modelling the inactivation of viruses from the Coronaviridae family in response to temperature and relative humidity in suspensions or surfaces.. Applied and Environmental Microbiology, 2020, 10.1128/AEM.01244-20 . pasteur-02908234

HAL Id: pasteur-02908234

<https://pasteur.hal.science/pasteur-02908234>

Submitted on 13 Aug 2020

HAL is a multi-disciplinary open access archive for the deposit and dissemination of scientific research documents, whether they are published or not. The documents may come from teaching and research institutions in France or abroad, or from public or private research centers.

L'archive ouverte pluridisciplinaire **HAL**, est destinée au dépôt et à la diffusion de documents scientifiques de niveau recherche, publiés ou non, émanant des établissements d'enseignement et de recherche français ou étrangers, des laboratoires publics ou privés.



Distributed under a Creative Commons Attribution - NonCommercial - NoDerivatives | 4.0 International License

1 **Modelling the inactivation of viruses from the *Coronaviridae* family in response to**
2 **temperature and relative humidity in suspensions or surfaces.**

3

4 **Authors:**

5 Laurent Guillier,^a Sandra Martin-Latil,^b Estelle Chaix,^a Anne Thébault,^a Nicole Pavio,^c
6 Sophie Le Poder,^c on behalf of Covid-19 Emergency Collective Expert Appraisal Group,^d
7 Christophe Batéjat,^e Fabrice Biot,^f Lionel Koch,^f Don Schaffner,^g Moez Sanaa,^a

8

9 **Affiliations:**

10 ^aRisk Assessment Department, French Agency for Food, Environmental and
11 Occupational Health & Safety, 14, rue Pierre et Marie Curie, Maisons-Alfort, France

12 ^bLaboratory for food safety French Agency for Food, Environmental and Occupational
13 Health & Safety, University of Paris-EST, Maisons-Alfort, France

14 ^cUMR Virologie 1161, ENVA, INRAE, Anses, Maisons-Alfort, France

15 ^dMembership of the Covid-19 Emergency Collective Expert Appraisal Group is provided
16 in the Acknowledgments

17 ^eEnvironment and Infectious Risks Unit, Laboratory for Urgent Response to Biological
18 Threats (CIBU), Institut Pasteur, Paris, France

19 ^fBacteriology Unit, French Armed Forces Biomedical Research Institute (IRBA),
20 Brétigny-sur-Orge, France

21 ^gDepartment of Food Science, Rutgers University, New Brunswick, NJ, USA

22

23 **Affiliations:**

24 Running Head: Modelling the inactivation of coronaviruses on fomites

25 #Address correspondence to laurent.guillier@anses.fr

26 **Abstract:**

27 Temperature and relative humidity are major factors determining virus inactivation in the
28 environment. This article reviews inactivation data of coronaviruses on surfaces and in
29 liquids from published studies and develops secondary models to predict coronaviruses
30 inactivation as a function of temperature and relative humidity. A total of 102 D-values
31 (time to obtain a \log_{10} reduction of virus infectivity), including values for SARS-CoV-2,
32 were collected from 26 published studies. The values obtained from the different
33 coronaviruses and studies were found to be generally consistent. Five different models
34 were fitted to the global dataset of D-values. The most appropriate model considered
35 temperature and relative humidity. A spreadsheet predicting the inactivation of
36 coronaviruses and the associated uncertainty is presented and can be used to predict
37 virus inactivation for untested temperatures, time points or any coronavirus strains
38 belonging to *Alphacoronavirus* and *Betacoronavirus* genera.

39 **Importance:** The prediction of the persistence of SARS-CoV-2 on fomites is essential to
40 investigate the importance of contact transmission. This study collects available
41 information on inactivation kinetics of coronaviruses in both solid and liquid fomites and
42 creates a mathematical model for the impact of temperature and relative humidity on
43 virus persistence. The predictions of the model can support more robust decision-
44 making and could be useful in various public health contexts. Having a calculator for the
45 natural clearance of SARS-CoV-2 depending on temperature and relative humidity could
46 be a valuable operational tool for public authorities.

47 **Keywords:** Persistence, coronavirus, modelling, fomites, SARS-CoV-2

48

49 **1. Introduction**

50 The pandemic of coronavirus respiratory infectious disease (COVID-19) initiated in
51 Wuhan, China in December 2019 was caused by an emergent virus named Severe
52 Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2). SARS-CoV-2 belongs to the
53 order *Nidovirales*, family *Coronaviridae*. These enveloped viruses have a positive,
54 single-stranded RNA genome (directly translated) surrounded by a nucleocapsid protein.
55 Coronaviruses are classified into four genera: alpha (α CoV), beta (β CoV), gamma
56 (γ CoV), and delta (δ CoV). SARS-CoV-2 belongs to the *Betacoronavirus* genus and the
57 *Sarbecovirus* sub-genus.

58 The route of transmission of respiratory viruses is airborne via inhalation of droplets and
59 aerosols or through contact with contaminated intermediate objects (fomites), e.g. by
60 self-inoculation of mucous membranes (mouth, eyes) by contaminated hands (1). The
61 transmission route for SARS-CoV-2, SARS-CoV and Middle East respiratory syndrome
62 (MERS-CoV) is primarily airborne (2-5) while environmental contamination through
63 surfaces is uncertain (6-8). No study has currently quantified the importance of surface
64 contact transmission in the spread of coronavirus diseases (9). Viral genomes have
65 been detected in the stools of COVID-19 patients and sewage (10), but the role of liquid
66 fomites has not yet been addressed.

67 Working with highly virulent coronavirus requires biosafety level 3 laboratory
68 containment conditions and since SARS-CoV2 emerged very recently, few data on its
69 survival related to environmental conditions are available (11, 12). The use of surrogate
70 coronaviruses has been suggested to overcome these challenges and expand the
71 available data on coronavirus survival likelihood (13). Surrogates can be used under the

72 assumption that they have similar physicochemical properties that mimic the viruses
73 they represent (14, 15).
74 Temperature and relative humidity have been shown to impact the kinetics of
75 inactivation of coronaviruses. Increased temperatures have been shown to increase the
76 rate of the inactivation (11, 16), and decreased relative humidity have been associated
77 with a reduction of coronaviruses inactivation rate on surfaces (13, 17-19). Inactivation
78 rates were lower in suspensions compared to surfaces in studies that tested both
79 suspensions and surfaces at similar temperatures (11, 20).
80 Hence, the prediction of the persistence of SARS-CoV-2 on fomites is essential to
81 investigate the importance of contact transmission. This study collects available
82 information on inactivation kinetics of coronaviruses in both solid and liquid fomites and
83 models the impact of temperature and relative humidity on virus persistence.

84

85 **2. Materials & methods**

86 **2.1. Selection of the studies**

87 Four inclusion criteria were used to identify studies that characterized inactivation of
88 coronaviruses according to temperature and relative humidity. Selected studies had to
89 focus on one virus from the *Coronaviridae* family. Inactivation must have been carried
90 out in suspensions or on inert non-porous surfaces. Only surfaces without antimicrobial
91 properties were considered. The quantification of infectious viruses had to be assessed
92 by cell culture, since RT-qPCR can underestimate actual virus infectivity (21, 22).
93 Finally, the available kinetics data points should be sufficient to allow precise statistical
94 estimation of the rate of viral inactivation without bias. In this context, kinetic data with

95 no significant inactivation observed during the experiment or with values below the
96 quantification limit in the first time interval were not included.

97 **2.2. Data collection**

98 The kinetics were gathered from either the figures or the tables of the selected studies.
99 The digitize R package (23) was used to retrieve data from scatter plots in figures. This
100 package loads a graphical file of a scatterplot (in jpeg format) in the graphical window of
101 R and calibrates and extracts the data. Data were manually reported in R vector for data
102 provided in tables. A key was attributed to kinetics collected in each study (Table 1). The
103 specific list of tables and figures used for each kinetics is given in appendix 1.

104 **2.3. Modelling of inactivation**

105 A simple primary model was used for describing each inactivation kinetics. The D-values
106 (or decimal reduction times) were determined from the kinetics of the \log_{10} number of
107 infectious viruses (N) over time at each experimental temperature. D is the inverse of
108 the slope of the inactivation kinetics.

$$109 \log_{10}(N) = \log_{10}(N_0) - t/D \quad \text{eq. (1)}$$

110 Several secondary models describing the impact of temperature (T) and relative
111 humidity (RH) on D values were tested. The gamma concept of inactivation was used
112 (24, 25). In this approach, the inactivation of a microbial population could be estimated
113 by:

$$114 \log_{10}(D) = \log_{10}(D_{ref}) - \sum \log_{10}(\lambda_{xi}(x_i)) \quad \text{eq. (2)}$$

115 Where λ_{x_i} quantifies the influence of each environmental factors (x_i corresponds to
116 temperature and relative humidity in this study) on the microbial resistance (D_{ref})
117 observed in reference conditions.

118 Based on eq. (2), five different secondary models were established. Models #1, #2 and
119 #3 do not consider the nature of the fomite.

120 Model #1 is the classical Bigelow model (26). It models only the effect of temperature.

121 The z_T , the increase of temperature which leads to a tenfold reduction of D , value was
122 determined as the negative inverse slope of the plot of $\log_{10}(D)$ versus temperature. z_T is
123 the increase of temperature which leads to a ten-fold reduction of the decimal reduction
124 time. T_{ref} is the reference temperature (set to 4°C in our study) and $\log_{10}(D_{ref})$ is the
125 $\log_{10}(D)$ at T_{ref} .

126 Model #1

$$127 \log_{10}(\lambda_T(T)) = \frac{T-T_{ref}}{z} \text{ and } \log_{10}(\lambda_{RH}(RH)) = 0$$

128 Model #2 considers the effect of temperature, however D values were fitted according to
129 temperature using a semi-log approach, derived from Mafart (25).

$$130 \log_{10}(\lambda_T(T)) = \left(\frac{T-T_{ref}}{z_T}\right)^2 \text{ and } \log_{10}(\lambda_{RH}(RH)) = 0$$

131 Model #3 is similar to model #2 but the shape parameter n was estimated instead of
132 being set to 2.

$$133 \log_{10}(\lambda_T(T)) = \left(\frac{T-T_{ref}}{z_T}\right)^n \text{ and } \log_{10}(\lambda_{RH}(RH)) = 0$$

134

135 The last two models (#4 and #5) consider the effect of temperature and the nature of the
 136 fomites. The type of fomite was taken into account through the use of relative humidity.
 137 Suspensions correspond to more than 99% RH conditions while surfaces are associated
 138 with RH conditions below to this threshold. The models consider that surfaces at higher
 139 relative humidity allow for more rapid inactivation and that inactivation in suspensions is
 140 equivalent to inactivation on surfaces exposed to low RH. In model #4, the shape
 141 parameter for temperature was set to 2 as in model #2.

$$142 \quad \log_{10}(\lambda_T(T)) = \left(\frac{T-T_{ref}}{z_T}\right)^2 \quad \text{and} \quad \log_{10}(\lambda_{RH}(RH)) = \begin{cases} \frac{RH}{z_{RH}} & RH < 99\% \\ 0 & RH \geq 99\% \end{cases}$$

143 In model #5, n is a model parameter to be estimated.

$$144 \quad \log_{10}(\lambda_T(T)) = \left(\frac{T-T_{ref}}{z_T}\right)^n \quad \text{and} \quad \log_{10}(\lambda_{RH}(RH)) = \begin{cases} \frac{RH}{z_{RH}} & RH < 99\% \\ 0 & RH \geq 99\% \end{cases}$$

145 In models #4 and #5, z_{RH} is the increase of relative humidity, which leads to a ten-fold
 146 reduction of the decimal reduction time.

147 **2.4. Model's parameters estimation**

148 The model's parameters were fitted with nls() R function. Confidence intervals of fitted
 149 parameters were assessed by bootstrap using nlsBoot() function from nlsMicrobio R
 150 package (27). The five models were compared according to penalized-likelihood criteria,
 151 the Aikaike information criterion (AIC) (28) and Bayesian information criterion (BIC) (29).

$$AIC = p \cdot \ln\left(\frac{RSS}{p}\right) + 2k$$

$$BIC = p \cdot \ln\left(\frac{RSS}{p}\right) + k \cdot \ln(p)$$

152 Where RSS is the residual sum of squares, p is the number of experimental points and k
153 the number of parameters in the model. The lower the AIC and BIC, the better the model
154 fits the dataset.

155 **2.5. Data availability**

156 The detailed information on the tables and figures where the data were collected are
157 given in appendix 1. All the scripts and data used to prepare figures and tables of this
158 manuscript are available in a Github repository (30).

159

160 **3. Results**

161 **3.1. Literature review results**

162 Table 1 shows the detailed characteristics of the twenty-six studies that characterized
163 inactivation of a virus from the *Coronaviridae* family according to temperature and or
164 relative humidity. Some kinetics were not appropriate for characterizing inactivation rate
165 either because the duration of the experiments was too short to observe any significant
166 decrease of virus infectivity, or because the quantification limit was reached before the
167 first time point (Table 1). A total of 102 estimates of D-value were collected from 25 of
168 the 26 studies (Appendix 1). These kinetic values represent 605 individual data points.
169 For each curve, a D-value (i.e. decimal reduction time) was estimated. The 102 D-values
170 are given in Appendix 1. Among the 102 kinetic values, 44 are from members of the
171 *Alphacoronavirus* genus including one from Canine coronavirus (CCV), two for the feline
172 infectious peritonitis virus (FIPV), five for the porcine epidemic diarrhea virus (PEDV), 14
173 for the Human coronavirus 229E (HCoV-229E) and 22 from the porcine transmissible
174 gastroenteritis coronavirus (TGEV). The remaining 58 kinetics are related to the

175 *Betacoronavirus* genus, including two Human coronavirus - OC43 (HCoV-OC43), two for
176 the bovine coronavirus, 13 for the murine hepatitis virus (MHV), eight for the MERS-
177 CoV, 22 for the SARS-CoV and 11 for the SARS-CoV-2. Figure 1 shows the 102
178 estimates of D-values, including 40 values on inert surfaces and 62 values in
179 suspension from temperatures ranging from 4°C to 68°C. Different suspensions were
180 noted, but most were laboratory media (Table 1).

181 **3.2. Modelling the inactivation**

182 The 102 D-values were fitted with five different models. Table 2 shows the performance
183 of these models to describe D-values according to temperature and relative humidity.

184 For the tested range of temperatures (between 4 and 68°C), model #1 (the classical
185 Bigelow model) based on a log-linear relation between D-values and temperature does
186 not perform as well as model #2 that considers a linear second-degree equation. Model
187 #3 offers a further refinement over model #2 by also fitting the degree of the equation (n
188 parameter). The fitted value of n was equal to 1.9 with a confidence interval that
189 includes 2 (*i.e.* model #2). Accordingly, the values taken by the parsimony criteria for
190 model selection AIC and BIC for model #2 and #3, indicate that n can be set to 2.

191 Figure 2 illustrates the performance of models #1 (Fig. 2A), #2 (Fig. 2B) and #3 (Fig. 3C)
192 for which only temperature effect is considered for predicting D-values.

193 Table 2 demonstrates that the inclusion of relative humidity should be considered.

194 Models #4 and #5 that describe the D-values according to temperature and relative
195 humidity were more appropriate models than models #1, #2 and #3 with a decrease of
196 AIC of more than 2 points in comparison with other models (31). The estimated value for
197 the shape parameter in model #5 is not different from the value two. According to BIC

198 criterion, model #4 and model #2 were the most appropriate and undistinguishable.
199 Based on these comparisons, model #4 was retained. Figure 3A shows the prediction of
200 inactivation rate according to T and RH for this model. The high z_{RH} value (Table 2)
201 indicates that the impact of RH is far less important than temperature. For example,
202 increasing the relative humidity by 80%, e.g. from 10% to 90%, only reduces the D
203 values by a factor of 1.7. The same reduction factor of D-values can be obtained by a
204 small change of temperature, (e.g. changing from 10 to 15°C or from 60 to 61°C). Model
205 #2 was retained as well as it provides very similar performance. Figures 3B shows the
206 residuals for model #4. Comparative analysis of residuals of models #2 and #4 are
207 provided in Appendix 2 (Figure A2-1).

208

209 **3.3. Potential use of the model**

210 An Excel spreadsheet implementing model #4 has been prepared and is available in
211 Appendix 3. The spreadsheet can be used to estimate the number of decimal reductions
212 of infectivity of coronaviruses according to user defined time, temperature and relative
213 humidity. For example, the predicted inactivation at a temperature of 70°C for 1 minute
214 in liquid is $-11.8 \log_{10}$ with a 95% CI [-6.4; -22.1] for model #4 and $-11.1 \log_{10}$ with a
215 95% CI [-5.7; -21.4] for model #2. The spreadsheet also allows an estimate of the time
216 necessary to reach a target number of decimal reductions of infectivity with a certain
217 confidence level for both model #4 and model #2. For example, the time to reach a 5
218 \log_{10} inactivation at 20°C and 75% relative humidity is 304 h with a 95% CI of [215; 426].
219 It will be much longer at 20% relative humidity as the time to reach a 5 \log_{10} inactivation
220 is predicted to be 438 h with a 95% CI of [339; 569]. Model #2 (that does not take into

221 account relative humidity), provides an estimate of the time to reach a 5 log₁₀
222 inactivation at 20°C of 412 h with a 95% CI of [322; 539].

223

224 **4. Discussion**

225 Our study identified 102 kinetic values for inactivation of coronaviruses on surfaces and
226 in suspensions. The included studies cover those identified in three recently published
227 articles that conducted a systematic review on coronaviruses inactivation (32-34). These
228 data were used to suggest a novel inactivation model specific to the *Coronaviridae*
229 family. The modelling approach identified temperature and relative humidity as major
230 factors needed to predict infectious coronavirus persistence on fomites.

231 The log₁₀ of D values was not linearly related to temperature in the range of
232 temperatures studied (4 – 68°C). Bertrand et al. (15) made a similar observation in a
233 meta-analysis for virus and phage inactivation in foods and water and proposed two
234 different models on either side of the threshold temperature of 50°C. Laude (16)
235 suggested a similar approach for TGEV with a threshold temperature at 45°C (16). The
236 modelling approach we used in our study allows fitting the inactivation values with a
237 single relation. In other meta-analysis on inactivation of viruses, Boehm et al. (22) and
238 Heßling et al. (35) did not observe such different trends but also studied smaller
239 temperature ranges. In the highest range of temperature (above 60°C), coronaviruses
240 are found to be far less heat resistant than non-enveloped viruses (36).

241 The present modelling approach considers the non-monotonous impact of relative
242 humidity on inactivation. Coronaviruses persisted better at low RHs and at 100% RH,
243 than for intermediate RHs. Another study has confirmed that low RH makes viruses

244 more resistant to thermal inactivation (37). Lin and Marr (38) recently observed the
245 same relation for two bacteriophages, where the observed RH where survival was worst
246 is close to 80% while in the present study, the less favorable condition for coronaviruses
247 was set to 99%. The data collected in the present study do not cover a uniform
248 distribution of temperatures and RH values. Further data corresponding to inactivation of
249 coronaviruses on surfaces at low humidities for temperature between 40 and 60°C
250 would help to refine assessment of impact of RH. Using a worst-case RH set to 99%
251 may be appropriate to estimate reductions in those situations until the model can be
252 refined.

253 As noted in the methods, all the kinetic values analyzed were established based on the
254 quantification of coronavirus infectivity with cell cultures. The model prediction did not
255 include other inactivation results from methods combining dyes with RT-qPCR. This
256 method (although more appropriate than classical RT-qPCR) can underestimate virus
257 infectivity (21, 22).

258 The data collected from the literature does not permit models specific to species at this
259 time. Our findings suggest that persistence potential of different coronaviruses is similar.
260 It confirms previous finding that advocates for the use of surrogates' coronavirus such
261 as TGEV (39). This could considerably simplify the acquisition of relevant data for
262 persistence potential for other environmental factors. The data analyzed here only
263 include *Alpha-* and *Betacoronavirus*, as no data for the two other major genera, *Delta-*
264 and *Gammacoronavirus*, were identified. Inclusion of such data would help to challenge
265 the present model robustness.

266 The models developed in our study are specific to viruses from the *Coronaviridae* family.
267 Several studies on the inactivation of other viruses have suggested that the impact of

268 temperature can be modelled, as a whole, with a unique parameter (15, 22, 40).
269 Variability of behavior by virus type has been observed and model parameters to
270 account these differences have been proposed (22, 40), e.g. non-enveloped viruses are
271 known to show greater persistence in the environment (40). Like a recently proposed
272 model for SARS-CoV-2 (41), our model takes into consideration of relative humidity in
273 the prediction of inactivation. This integration is of high interest in the perspective of
274 assessment of seasonality on virus persistence (42).
275 It's also worth noting our model is specific to fomites. Survival kinetics in fecal materials
276 were identified (43) but not considered for inclusion. The level of matrix contamination
277 with fecal materials has been shown to significantly increase the inactivation rate of
278 viruses (40), so by excluding these data, model predictions are biased to be fail-safe.
279 Inactivation data on porous surfaces were also not considered since it may be difficult to
280 determine if any measured inactivation is associated with real loss of infectivity or
281 difficulty in recovering viruses absorbed inside the porous material. That said, there is no
282 reason to consider that model predictions for coronaviruses are not pertinent to survival
283 on porous material (e.g. face masks).
284 Inactivation on anti-microbial surfaces, such as copper and silver, was also not
285 considered. For the same reason, model predictions are fail safe as surfaces including
286 copper or other antimicrobial compounds increase the inactivation rate of coronaviruses
287 (12, 44).
288 The predictions of the present model could support more robust decision-making and
289 could be useful in various contexts such as blood safety assessment (45) or validation of
290 thermal inactivating treatments for room air, surfaces or suspensions. Indeed, an
291 important issue is the possibility of reusing privates or public offices, rooms of hotels, or

292 vehicles that are difficult to decontaminate. Moreover, many devices like electronics or
293 more sensitive materials, are not suitable for chemical decontamination processes which
294 could make them inoperative. Another aspect of decontamination is the economical
295 challenge as large scale decontamination of buildings can cost billions of dollars (46).
296 Furthermore, the use of detergents and/or disinfectants may have environmental
297 consequences. Thus the large scale decontamination of surfaces for SARS-CoV-2 that
298 are not necessarily in contact with people may not be required. For these reasons the
299 waiting time needed before handling suspected contaminated materials in absence of
300 decontamination is more than ever an important question. Having a calculator for the
301 natural clearance of SARS-CoV-2 depending on temperature could be a valuable
302 operational tool for public authorities (41).

303 The present model also opens the way for risk assessment for SARS-CoV-2
304 transmission through contact (47). Further model developments including data on matrix
305 pH, salinity and exposure to visible and UV light would also be important to consider (40,
306 48).

307 **5. Acknowledgements**

308 The Covid-19 Emergency Collective Expert Appraisal Group members included the co-
309 authors LG, S.M-L, E.C, N.P, S.L.P, M.S. and (in alphabetical order): Paul Brown,
310 Charlotte Dunoyer, Florence Etoire, Elissa Khamisse, Meriadeg Legouil, François
311 Meurens, Gilles Meyer, Elodie Monchatre-Leroy, Gaëlle Simon and Astrid Vabret.

312

313

314

315 **6. References**

- 316 1. Kutter JS, Spronken MI, Fraaij PL, Fouchier RA, Herfst S. 2018. Transmission
317 routes of respiratory viruses among humans. *Current Opinion in Virology* 28:142-
318 151.
- 319 2. Lu J, Gu J, Li K, Xu C, Su W, Lai Z, Zhou D, Yu C, Xu B, Yang Z. 2020. COVID-
320 19 Outbreak associated with air conditioning in restaurant, Guangzhou, China,
321 2020. *Emerging Infectious Diseases* 26.
- 322 3. Lee N, Hui D, Wu A, Chan P, Cameron P, Joynt GM, Ahuja A, Yung MY, Leung
323 C, To K. 2003. A major outbreak of severe acute respiratory syndrome in Hong
324 Kong. *New England Journal of Medicine* 348:1986-1994.
- 325 4. Kim S-H, Chang SY, Sung M, Park JH, Bin Kim H, Lee H, Choi J-P, Choi WS,
326 Min J-Y. 2016. Extensive viable Middle East respiratory syndrome (MERS)
327 coronavirus contamination in air and surrounding environment in MERS isolation
328 wards. *Reviews of Infectious Diseases* 63:363-369.
- 329 5. Liu J, Liao X, Qian S, Yuan J, Wang F, Liu Y, Wang Z, Wang F, Liu L, Zhang Z.
330 2020. Community transmission of Severe Acute Respiratory Syndrome
331 Coronavirus 2, Shenzhen, China, 2020. *Emerging Infectious Diseases* 26.
- 332 6. Chen Y-C, Huang L-M, Chan C-C, Su C-P, Chang S-C, Chang Y-Y, Chen M-L,
333 Hung C-C, Chen W-J, Lin F-Y. 2004. SARS in hospital emergency room.
334 *Emerging Infectious Diseases* 10:782.
- 335 7. Danis K, Epaulard O, Bénét T, Gaymard A, Campoy S, Bothelo-Nevers E,
336 Bouscambert-Duchamp M, Spacciferri G, Ader F, Mailles A, Boudalaa Z, Tolsma
337 V, Berra J, Vaux S, Forestier E, Landelle C, Fougere E, Thabuis A, Berthelot P,
338 Veil R, Levy-Bruhl D, Chidiac C, Lina B, Coignard B, Saura C, Team I. 2020.
339 Cluster of coronavirus disease 2019 (Covid-19) in the French Alps, 2020. *Clinical*
340 *Infectious Diseases* doi:10.1093/cid/ciaa424.
- 341 8. Otter JA, Donskey C, Yezli S, Douthwaite S, Goldenberg SD, Weber DJ. 2016.
342 Transmission of SARS and MERS coronaviruses and influenza virus in
343 healthcare settings: the possible role of dry surface contamination. *Journal of*
344 *Hospital Infection* 92:235-250.
- 345 9. Wolff MH, Sattar SA, Adegbunrin O, Tetro J. 2005. Environmental survival and
346 microbicide inactivation of coronaviruses, p 201-212, *Coronaviruses with special*
347 *emphasis on first insights concerning SARS*. Springer.
- 348 10. Medema G, Heijnen L, Elsinga G, Italiaander R, Brouwer A. 2020. Presence of
349 SARS-Coronavirus-2 in sewage. medRxiv
350 doi:<https://doi.org/10.1101/2020.03.29.20045880>.
- 351 11. Chin A, Chu J, Perera M, Hui K, Yen H-L, Chan M, Peiris M, Poon L. 2020.
352 Stability of SARS-CoV-2 in different environmental conditions. . *The Lancet*
353 *Microbe* [https://doi.org/10.1016/S2666-5247\(20\)30003-3](https://doi.org/10.1016/S2666-5247(20)30003-3).
- 354 12. van Doremalen N, Bushmaker T, Morris DH, Holbrook MG, Gamble A, Williamson
355 BN, Tamin A, Harcourt JL, Thornburg NJ, Gerber SI. 2020. Aerosol and surface
356 stability of SARS-CoV-2 as compared with SARS-CoV-1. *New England Journal of*
357 *Medicine*.
- 358 13. Casanova LM, Jeon S, Rutala WA, Weber DJ, Sobsey MD. 2010. Effects of air
359 temperature and relative humidity on coronavirus survival on surfaces. *Appl*
360 *Environ Microbiol* 76:2712-2717.

- 361 14. Bozkurt H, D'Souza DH, Davidson PM. 2015. Thermal inactivation kinetics of
362 human norovirus surrogates and hepatitis A virus in turkey deli meat. *Appl*
363 *Environ Microbiol* 81:4850-4859.
- 364 15. Bertrand I, Schijven J, Sánchez G, Wyn-Jones P, Ottoson J, Morin T, Muscillo M,
365 Verani M, Nasser A, de Roda Husman A. 2012. The impact of temperature on the
366 inactivation of enteric viruses in food and water: a review. *Journal of Applied*
367 *Microbiology* 112:1059-1074.
- 368 16. Laude H. 1981. Thermal inactivation studies of a coronavirus, transmissible
369 gastroenteritis virus. *Journal of General Virology* 56:235-240.
- 370 17. Van Doremalen N, Bushmaker T, Munster V. 2013. Stability of Middle East
371 respiratory syndrome coronavirus (MERS-CoV) under different environmental
372 conditions. *Eurosurveillance* 18:20590.
- 373 18. Ijaz M, Brunner A, Sattar S, Nair RC, Johnson-Lussenburg C. 1985. Survival
374 characteristics of airborne human coronavirus 229E. *Journal of General Virology*
375 66:2743-2748.
- 376 19. Ijaz M, Karim Y, Sattar S, Johnson-Lussenburg C. 1987. Development of
377 methods to study the survival of airborne viruses. *Journal of Virological Methods*
378 18:87-106.
- 379 20. Rabenau H, Cinatl J, Morgenstern B, Bauer G, Preiser W, Doerr H. 2005. Stability
380 and inactivation of SARS coronavirus. *Medical Microbiology and Immunology*
381 194:1-6.
- 382 21. Coudray-Meunier C, Fraisse A, Martin-Latil S, Guillier L, Perelle S. 2013.
383 Discrimination of infectious hepatitis A virus and rotavirus by combining dyes and
384 surfactants with RT-qPCR. *BMC microbiology* 13:216.
- 385 22. Boehm AB, Silverman AI, Schriewer A, Goodwin K. 2019. Systematic review and
386 meta-analysis of decay rates of waterborne mammalian viruses and coliphages in
387 surface waters. *Water research* 164:114898.
- 388 23. Poisot T. 2011. The digitize package: extracting numerical data from scatterplots.
389 *The R Journal* 3:25-26.
- 390 24. Coroller L, Kan-King-Yu D, Leguerinel I, Mafart P, Membré J-M. 2012. Modelling
391 of growth, growth/no-growth interface and nonthermal inactivation areas of
392 *Listeria* in foods. *International Journal of Food Microbiology* 152:139-152.
- 393 25. Mafart P. 2000. Taking injuries of surviving bacteria into account for optimising
394 heat treatments. *International Journal of Food Microbiology* 55:175-179.
- 395 26. Bigelow W. 1921. The logarithmic nature of thermal death time curves. *The*
396 *Journal of Infectious Diseases*:528-536.
- 397 27. Baty F, Delignette-Muller M. 2017. nlsMicrobio: data sets and nonlinear
398 regression models dedicated to predictive microbiology. R package version 0.0-1.
- 399 28. Akaike H. 1974. A new look at the statistical model identification. *IEEE*
400 *Transactions on Automatic Control* 19:716-723.
- 401 29. Schwarz G. 1978. Estimating the dimension of a model. *The Annals of Statistics*
402 6:461-464.
- 403 30. Guillier L. 2020. Data and models related to coronaviruses inactivation.
404 <https://github.com/lguillier/Persistence-Coronavirus>.
- 405 31. Burnham KP, Anderson DR. 1998. Practical use of the information-theoretic
406 approach, p 75-117, *Model selection and inference*. Springer.

- 407 32. Kampf G, Voss A, Scheithauer S. 2020. Inactivation of coronaviruses by heat.
408 Journal of Hospital Infection.
- 409 33. La Rosa G, Bonadonna L, Lucentini L, Kenmoe S, Suffredini E. 2020.
410 Coronavirus in water environments: Occurrence, persistence and concentration
411 methods-A scoping review. Water Research:115899.
- 412 34. Kampf G, Todt D, Pfaender S, Steinmann E. 2020. Persistence of coronaviruses
413 on inanimate surfaces and its inactivation with biocidal agents. Journal of Hospital
414 Infection.
- 415 35. Heßling M, Hoenes K, Lingenfelder C. 2020. Selection of parameters for thermal
416 Coronavirus inactivation—A data-based recommendation. GMS Hygiene and
417 Infection Control in press.
- 418 36. Firquet S, Beaujard S, Lobert P-E, Sané F, Caloone D, Izard D, Hober D. 2014.
419 Viruses contained in droplets applied on warmed surface are rapidly inactivated.
420 Microbes and Environments 29:408-412.
- 421 37. Sauerbrei A, Wutzler P. 2009. Testing thermal resistance of viruses. Archives of
422 virology 154:115-119.
- 423 38. Lin K, Marr LC. 2019. Humidity-Dependent Decay of Viruses, but Not Bacteria, in
424 Aerosols and Droplets Follows Disinfection Kinetics. Environmental Science &
425 Technology 54:1024-1032.
- 426 39. Casanova L, Rutala WA, Weber DJ, Sobsey MD. 2009. Survival of surrogate
427 coronaviruses in water. Water research 43:1893-1898.
- 428 40. Brainard J, Pond K, Hunter PR. 2017. Censored regression modeling to predict
429 virus inactivation in wastewaters. Environmental science & technology 51:1795-
430 1801.
- 431 41. United States Department of Homeland Security. 2020. SARS-CoV-2 Indoor
432 Environmental Stability Predictive Model. Available at:
433 [https://www.dhs.gov/sites/default/files/publications/sars-cov-](https://www.dhs.gov/sites/default/files/publications/sars-cov-2_environment_predictive_model_factsheet_2.pdf)
434 [2_environment_predictive_model_factsheet_2.pdf](https://www.dhs.gov/sites/default/files/publications/sars-cov-2_environment_predictive_model_factsheet_2.pdf)
- 435 42. Prussin AJ, Schwake DO, Lin K, Gallagher DL, Buttlng L, Marr LC. 2018.
436 Survival of the enveloped virus Phi6 in droplets as a function of relative humidity,
437 absolute humidity, and temperature. Appl Environ Microbiol 84:e00551-18.
- 438 43. Lai MY, Cheng PK, Lim WW. 2005. Survival of severe acute respiratory
439 syndrome coronavirus. Clinical Infectious Diseases 41:e67-e71.
- 440 44. Warnes SL, Little ZR, Keevil CW. 2015. Human coronavirus 229E remains
441 infectious on common touch surface materials. MBio 6:e01697-15.
- 442 45. Chang L, Yan Y, Wang L. 2020. Coronavirus Disease 2019: Coronaviruses and
443 Blood Safety. Transfusion Medicine Reviews
444 doi:<https://doi.org/10.1016/j.tmr.2020.02.003>.
- 445 46. Schmitt K, Zacchia NA. 2012. Total decontamination cost of the anthrax letter
446 attacks. Biosecurity and bioterrorism: biodefense strategy, practice, and science
447 10:98-107.
- 448 47. Haas C. 2020. Coronavirus and Risk Analysis. Risk Analysis 40:660-661.
- 449 48. Heßling M, Hönes K, Vatter P, Lingenfelder C. 2020. Ultraviolet irradiation doses
450 for coronavirus inactivation—review and analysis of coronavirus photoinactivation
451 studies. GMS Hygiene and Infection Control 15.

- 452 49. Mullis L, Saif LJ, Zhang Y, Zhang X, Azevedo MS. 2012. Stability of bovine
453 coronavirus on lettuce surfaces under household refrigeration conditions. *Food*
454 *Microbiology* 30:180-186.
- 455 50. Saknimit M, Inatsuki I, Sugiyama Y, Yagami K. 1988. Virucidal efficacy of
456 physico-chemical treatments against coronaviruses and parvoviruses of
457 laboratory animals. *Jikken dobutsu Experimental animals* 37:341.
- 458 51. Christianson K, Ingersoll J, Landon R, Pfeiffer N, Gerber J. 1989.
459 Characterization of a temperature sensitive feline infectious peritonitis
460 coronavirus. *Archives of Virology* 109:185-196.
- 461 52. Gundy PM, Gerba CP, Pepper IL. 2009. Survival of coronaviruses in water and
462 wastewater. *Food and Environmental Virology* 1:10.
- 463 53. Bucknall RA, King LM, Kapikian AZ, Chanock RM. 1972. Studies with human
464 coronaviruses II. Some properties of strains 229E and OC43. *Proceedings of the*
465 *Society for Experimental Biology and Medicine* 139:722-727.
- 466 54. Sizun J, Yu M, Talbot P. 2000. Survival of human coronaviruses 229E and OC43
467 in suspension and after drying on surfaces: a possible source of hospital-acquired
468 infections. *Journal of Hospital Infection* 46:55-60.
- 469 55. Lamarre A, Talbot PJ. 1989. Effect of pH and temperature on the infectivity of
470 human coronavirus 229E. *Canadian Journal of Microbiology* 35:972-974.
- 471 56. Leclercq I, Batejat C, Burguière AM, Manuguerra JC. 2014. Heat inactivation of
472 the Middle East Respiratory Syndrome coronavirus. *Influenza and Other*
473 *Respiratory Viruses* 8:585-586.
- 474 57. Ye Y, Ellenberg RM, Graham KE, Wigginton KR. 2016. Survivability, partitioning,
475 and recovery of enveloped viruses in untreated municipal wastewater.
476 *Environmental Science & Technology* 50:5077-5085.
- 477 58. Hofmann M, Wyler R. 1989. Quantitation, biological and physicochemical
478 properties of cell culture-adapted porcine epidemic diarrhea coronavirus (PEDV).
479 *Veterinary Microbiology* 20:131-142.
- 480 59. Quist-Rybachuk G, Nauwynck H, Kalmar I. 2015. Sensitivity of porcine epidemic
481 diarrhea virus (PEDV) to pH and heat treatment in the presence or absence of
482 porcine plasma. *Veterinary microbiology* 181:283-288.
- 483 60. Hulst MM, Heres L, Hakze-van der Honing R, Pelsler M, Fox M, van der Poel WH.
484 2019. Study on inactivation of porcine epidemic diarrhoea virus, porcine
485 sapelovirus 1 and adenovirus in the production and storage of laboratory spray-
486 dried porcine plasma. *Journal of Applied Microbiology* 126:1931-1943.
- 487 61. Darnell ME, Subbarao K, Feinstone SM, Taylor DR. 2004. Inactivation of the
488 coronavirus that induces severe acute respiratory syndrome, SARS-CoV. *Journal*
489 *of virological methods* 121:85-91.
- 490 62. Chan K, Peiris J, Lam S, Poon L, Yuen K, Seto W. 2011. The effects of
491 temperature and relative humidity on the viability of the SARS coronavirus.
492 *Advances in Virology* 2011.
- 493 63. Pagat A-M, Seux-Goepfert R, Lutsch C, Lecouturier V, Saluzzo J-F, Kusters IC.
494 2007. Evaluation of SARS-Coronavirus decontamination procedures. *Applied*
495 *Biosafety* 12:100-108.
- 496 64. Kariwa H, Fujii N, Takashima I. 2006. Inactivation of SARS coronavirus by means
497 of povidone-iodine, physical conditions and chemical reagents. *Dermatology*
498 212:119-123.

499 65. Batejat C, Grassin Q, Manuguerra J-C, Leclercq I. 2020. Heat inactivation of the
500 Severe Acute Respiratory Syndrome Coronavirus 2.
501 bioRxiv:<https://doi.org/10.1101/2020.05.01.067769>.
502

503 **Table 1. Characteristics of the studies that explored inactivation of infectivity of coronavirus.**

Virus	Genus	Sub-genus	Strain	Measurement	Temperatures (°C)	Conditions associated treatment with	Study reference
BCoV	<i>Betacoronavirus</i>	<i>Embecovirus</i>	Strain 88	PFU in Human rectal tumor (HRT)-18 cells	4	Salad, Minimal Essential Media (MEM) containing 2% Fetal Bovine Serum (FBS)	(49)
CCV	<i>Alphacoronavirus</i>	<i>Tegacovirus</i>	I-71	CRFK cells (PFU)	60, 80 ^b	MEM containing 2% Fetal Calf Serum (FCS)	(50)
FIPV	<i>Alphacoronavirus</i>	<i>Tegacovirus</i>	DF2-WT	Feline kidney (NLFK) cells	54	Basal Medium Eagle	(51)
FIPV	<i>Alphacoronavirus</i>	<i>Tegacovirus</i>	ATCC-990	Crandell Reese feline kidney cell line	4 ^b , 23	Dechlorinated, filtered tap water	(52)
HCoV	<i>Alphacoronavirus</i>	<i>Duvinacovirus</i>	229E	Cellular infectivity in cell strain (HDGS) WI38	33, 37	Maintenance medium 2% FCS	(53)
HCoV	<i>Alphacoronavirus</i>	<i>Duvinacovirus</i>	229E	Cellular infectivity in lung cell line L132	21 ^b	PBS, Earle's MEM, Earle's MEM to with added suspended cells	(54)
HCoV	<i>Alphacoronavirus</i>	<i>Duvinacovirus</i>	229E	Cellular infectivity in lung cell line L132	21 ^b	Aluminum, sponge, latex ^a at 65% RH	(54)
HCoV	<i>Alphacoronavirus</i>	<i>Duvinacovirus</i>	229E	CPE on MRC-5 cells	21	Teflon, PVC, Rubber, Steel, Plastic	(44)
HCoV	<i>Alphacoronavirus</i>	<i>Duvinacovirus</i>	229E	-	23	Cell culture supernatant with or without FBS	(20)
HCoV	<i>Alphacoronavirus</i>	<i>Duvinacovirus</i>	229E	MRC-5 cells (TCID-50)	4 ^b , 23	Dechlorinated, filtered tap water	(52)
HCoV	<i>Alphacoronavirus</i>	<i>Duvinacovirus</i>	229E	Cellular infectivity in lung cell line L132	4 ^b , 22, 33, 37	Earle's MEM	(55)
HCoV	<i>Betacoronavirus</i>	<i>Embecovirus</i>	OC43	Cellular infectivity in cell strain (HDGS) WI38	33, 37	Maintenance medium 2% FCS	(53)
HCoV	<i>Betacoronavirus</i>	<i>Embecovirus</i>	OC43	Cellular infectivity in human rectal tumor cell line HRT-18	21 ^b	PBS, Earle's MEM, Earle's MEM to with added suspended cells	(54)
HCoV	<i>Betacoronavirus</i>	<i>Embecovirus</i>	OC43	Cellular infectivity in human rectal tumor cell line HRT-18	21 ^b	Aluminum, sponge ^a , latex ^a at 65% RH	(54)
MERS-	<i>Betacoronavirus</i>	<i>Sarbecovirus</i>	FRA2	Cellular infectivity in	25 ^b , 56, 65	Cell culture supernatant	(56)

CoV				Vero cells (TCID-50)			
MERS-CoV	<i>Betacoronavirus</i>	<i>Sarbecovirus</i>	HCoV-EMC/2012	Cellular infectivity Vero cells (TCID-50)	in	20, 30	Plastic (30%, 40% or 80% RH) (17)
MERS-CoV	<i>Betacoronavirus</i>	<i>Sarbecovirus</i>	HCoV-EMC/2012	Cellular infectivity Vero cells (TCID-50)	in	20, 30	Plastic (30%, 40% or 80% RH) (17)
MHV	<i>Betacoronavirus</i>	<i>Embecovirus</i>	-	Cellular infectivity DBT cells	in	4 ^b , 25	Reagent-grade water (39)
MHV	<i>Betacoronavirus</i>	<i>Embecovirus</i>	-	Cellular infectivity DBT cells	in	4 ^b , 25	Lake water (39)
MHV	<i>Betacoronavirus</i>	<i>Embecovirus</i>	-	Cellular infectivity DBT cells	in	4 ^b , 20, 40	Stainless steel surface with 20% humidity (13)
MHV	<i>Betacoronavirus</i>	<i>Embecovirus</i>	-	Cellular infectivity DBT cells	in	4, 20, 40	Stainless steel surface with 50% humidity (13)
MHV	<i>Betacoronavirus</i>	<i>Embecovirus</i>	-	Cellular infectivity DBT cells	in	4, 20, 40	Stainless steel surface with 80% humidity (13)
MHV	<i>Betacoronavirus</i>	<i>Embecovirus</i>	MHV-2	Cellular infectivity DBT cells (PFU)	in	40 ^b , 60, 80 ^a	MEM containing 2% FCS (50)
MHV	<i>Betacoronavirus</i>	<i>Embecovirus</i>	MHV-N	Cellular infectivity DBT cells (PFU)	in	40 ^b , 60, 80 ^a	MEM containing 2% FCS (50)
MHV	<i>Betacoronavirus</i>	<i>Embecovirus</i>	A59	Plaque assay on cells	L2	10 ^b , 25	Pasteurized wastewater (57)
PEDV	<i>Alphacoronavirus</i>	<i>Pedacovirus</i>	V215/78	PFU on Vero cells		50	Diluted medium for virus replication (58)
PEDV	<i>Alphacoronavirus</i>	<i>Pedacovirus</i>	CV777	Vero cells (TCID-50)		40, 44, 48	MEM at pH 7.2 (59)
PEDV	<i>Alphacoronavirus</i>	<i>Pedacovirus</i>	CV777	Vero cells (TCID-50)		4 ^b , 44 ^b , 48	Medium at pH 7.5 (60)
SARS-CoV	<i>Betacoronavirus</i>	<i>Sarbecovirus</i>	FFM-1	Cellular infectivity Vero cells	in	56	Cell culture supernatant with or without FBS (20)
SARS-CoV	<i>Betacoronavirus</i>	<i>Sarbecovirus</i>	Urbani	Cellular infectivity Vero cells	in	56, 65, and 75 ^a	Dulbecco's MEM (61)
SARS-CoV	<i>Betacoronavirus</i>	<i>Sarbecovirus</i>	HKU39849	Cellular infectivity FRH-K4 (TCID-50)	in	28, 33, 38	Plastic stored at 95% RH (62)
SARS-CoV	<i>Betacoronavirus</i>	<i>Sarbecovirus</i>	HKU39849	Cellular infectivity FRH-K4 (TCID-50)	in	28 ^b , 33, 38	Plastic stored at 80-89% RH (62)
SARS-CoV	<i>Betacoronavirus</i>	<i>Sarbecovirus</i>	GVU6109	Cellular infectivity Vero cells (TCID-50)	in	20	Viral Transport Medium(VTM) (43)
SARS-CoV	<i>Betacoronavirus</i>	<i>Sarbecovirus</i>	GVU6109	Cellular infectivity Vero cells (TCID-50)	in	4, 20	Nasopharyngeal aspirate (NPA), throat and nasal swab (TNS) (43)

									or VTM	
SARS-CoV	Betacoronavirus	Sarbecovirus	Tor2 AY274119.3	Cellular infectivity Vero cells (TCID-50)	in	22		Plastic and stainless steel stored at 40°C	(12)	
SARS-CoV	Betacoronavirus	Sarbecovirus	Utah	Cellular infectivity Vero cells (TCID-50)	in	58, 68		Iscove's 4% FCS medium	(63)	
SARS-CoV	Betacoronavirus	Sarbecovirus	Utah	Cellular infectivity Vero cells (TCID-50)	in	22		Glass surface store at 10-25% RH	(63)	
SARS-CoV	Betacoronavirus	Sarbecovirus	Hanoi	Cellular infectivity Vero cells (TCID-50)	in	56		MEM	(64)	
SARS-CoV2	Betacoronavirus	Sarbecovirus	-	Cellular infectivity Vero cells (TCID-50)	in	4, 22, 37, 56, 70 ^a		VTM	(11)	
SARS-CoV2	Betacoronavirus	Sarbecovirus	-	Cellular infectivity Vero cells (TCID-50)	in	22		Plastic and stainless steel at 65% RH	(11)	
SARS-CoV2	Betacoronavirus	Sarbecovirus	WA1-2020 (MN985325.1)	Cellular infectivity Vero cells (TCID-50)	in	22		Plastic and stainless steel stored at 40°C	(12)	
SARS-CoV2	Betacoronavirus	Sarbecovirus	-	Cellular infectivity Vero cells (TCID-50)	in	56, 65 ^a		Cell culture supernatants	(65)	
SARS-CoV2	Betacoronavirus	Sarbecovirus	-	Cellular infectivity Vero cells (TCID-50)	in	65, 95 ^a		Nasopharyngeal samples	(65)	
SARS-CoV2	Betacoronavirus	Sarbecovirus	-	Cellular infectivity Vero cells (TCID-50)	in	56		Sera	(65)	
TGEV	Alphacoronavirus	Tegacovirus.	D52	Cellular infectivity RPTg cells	in	31, 35, 39, 43, 47, 51 and 55		In HEPES solution at pH 7	(16)	
TGEV	Alphacoronavirus	Tegacovirus.	D52	Cellular infectivity RPTg cells	in	35, 39, 43, 47, and 51		In HEPES solution at pH 8	(16)	
TGEV	Alphacoronavirus	Tegacovirus.	-	Cellular infectivity in ST cells		4 ^b , 20, 40		Stainless steel surface with 20% RH	(13)	
TGEV	Alphacoronavirus	Tegacovirus.	-	Cellular infectivity in ST cells		4, 20, 40		Stainless steel surface with 50% RH	(13)	
TGEV	Alphacoronavirus	Tegacovirus.	-	Cellular infectivity in ST cells		4, 20, 40		Stainless steel surface with 80% RH	(13)	
TGEV	Alphacoronavirus	Tegacovirus.	-	Cellular infectivity in ST cells		4 ^b , 25		Reagent-grade water	(39)	
TGEV	Alphacoronavirus	Tegacovirus.	-	Cellular infectivity in ST cells		4 ^b , 25		Lake water	(39)	

504 ^a not included: limit of quantification reached for the first sample time
505 ^b not included: not enough decrease observed during experimentation
506 - data not specified

507 **Table 2. Characteristics of the different models fitted to the 102 decimal reduction time**
 508 **data of coronaviruses according to temperature (T_{ref} set at 4°C) and relative humidity**

Model	Fitted parameters	Best fit values [95%CI bootstrap intervals]	Bayesian information criterion	Aikaike information criterion
Model #1	log10Dref z_T	3.1 [2.8 - 3.3] 13.8 [12.7 - 15.1]	-124.7	-130.0
Model #2	log10Dref z_T	2.2 [2.1 - 2.3] 29.4 [28.4 - 30.5]	-160.6	-165.9
Model #3	log10Dref z_T n	2.3 [2.1 - 2.6] 27.7 [23.2 - 31.6] 1.9 [1.5 - 2.2]	-156.7	-164.6
Model #4	log10Dref z_T z_{RH}	2.3 [2.2 - 2.5] 29.1 [28.1 - 30.1] 341.4.7 [190.1 - 5631.4]	-160.2	-168.0
Model #5	log10Dref z_T z_{RH} n	2.4 [2.2 - 2.6] 27.5 [23.6 - 31.2] 330.7 [182.8 - 7020,1] 1.9 [1.6 - 2.2]	-156.2	-166.6

509

510

511

512 **Figure 1. Decimal reduction times of ten coronaviruses according to temperature in**
513 **suspension or on inert surfaces.**

514

515 **Figure 2. Observed (points) and fitted (grey lines) log decimal reduction time values**
516 **according to temperature for model #1 (A), model #2 (B) and #3 (C). One thousand (1000)**
517 **bootstrap values of uncertainty characterization are shown. Estimates of model**
518 **parameters are given in Table 2.**

519

520 **Figure 3. (A) Observed inactivation rate values (grey points) according to temperature**
521 **(°C) and relative humidity (%) and Model #4 surface predictions. Scatter points of**
522 **observed versus predicted D-values (D in hours) for model #4 (B). The dashed line**
523 **represents a perfect match between observations and predictions.**









