¹ Comment for Session 3 of RSS Meeting on R₀

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I congratulate: Pellis and colleagues, and Dunbar and Held on their excellent 7 papers describing a variety of mechanistic models of SARS-Cov-2 transmission, 8 and more generally on their work to support policy formulation during the 9 COVID-19 pandemic. Both papers address the difficulties of predicting and then 10 evaluating the impact if non-pharmaceutical interventions (NPIs) against the 11 transmission of severe respiratory pathogens. These are likely to remain key 12 ongoing challenges for the analytical science of pandemic preparedness, with high 13 demand from policy makers for accurate estimates of the epidemiological benefits 14 of NPIs. Here, I would like to make one related methodological point. 15

There may be benefits in making the null hypotheses in mechanistic modelling 16 studies of NPIs more explicit and more general. For example, models usually 17 contain an underlying basic rate of transmissibility per unit time per infected 18 individual, often denoted β . The parameter β is used to calculate the risk of 19 infection per susceptible and is modified by other parameters to reflect differences 20 in infectiousness, susceptibility and mixing [Keeling and Rohani, 2011]. For 21 example, when schools are closed, it may be assumed that mixing patterns for 22 children change on that day and that the efficacy of school closures can be 23 estimated by fitting a version of the model to incidence data which includes a free 24 parameter describing the strength of change in mixing. However, this type of 25 calculation is implicitly making the strong assumption that a step change on the 26 day of the intervention is a good explanation for the overall pattern of changing 27

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transmissibility at that time, which may not be the case. It may be useful to
explicitly represented β as a smooth function of time in an alternative model, as is
common practise for similar parameters in other analytical frameworks
[Wood, 2017], so that typical measures of parsimony can be used to assess the
information contained in specific model fits when strong assumptions are made
about the timing of interventions.

34 **References**

- ³⁵ [Keeling and Rohani, 2011] Keeling, M. J. and Rohani, P. (2011). *Modeling*
- 36 Infectious Diseases in Humans and Animals. Princeton University Press.
- ³⁷ [Wood, 2017] Wood, S. N. (2017). *Generalized Additive Models: An Introduction* ³⁸ with R, Second Edition. CRC Press.