less defensible 5 years after the fact. Important questions such as those raised by Wood et al. warrant answers that draw on a broader range of data sources, including those that are more directly informative about transmission dynamics. For example, if the hypothesis is that infections peaked prior to the lockdown announcement due to voluntary or anticipatory behavioural changes, then it should be evaluated using additional evidence—such as mobility data, seroprevalence studies, healthcare service utilization data, or other innovative data sources. Relying solely on daily deaths within a single, unstratified analysis stretches this data stream beyond the limits of what it can reliably inform.

More broadly, while the current method identifies a single surprising and interesting result—an earlier-than-expected peak in infections—which emerges in part from the modelling assumptions themselves, what is missing is a positive, mechanistic explanation for the underlying dynamics. In the absence of such an explanation, it is difficult to assess the plausibility or generalizability of the conclusions. Strong claims require not only identifying anomalies but also articulating coherent alternative hypotheses that are consistent with a broader body of evidence. Without this, the argument risks becoming a post hoc rationalization of an artefact of model structure, rather than a robust inference about real-world epidemic processes.

We thank Wood et al. for their contribution and believe that their important questions will ultimately strengthen infectious disease modelling methods.

Conflicts of interest: None declared.

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Michael Whitehouse, Lorenzo Rimella, and Nick Whiteley's contribution to the Discussion of 'Some statistical aspects of the Covid-19 response' by Wood et al.

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We commend the authors of Wood et al. (2025) for their thorough contribution to the retrospective analysis of the statistical response to the Covid-19 crisis. We focus on the issues raised in Section 4 around the use of models with increasing extents of heterogeneity. The authors correctly recognize that omitting such considerations can lead to incorrect inference, e.g. poor estimation of final size. It is important, however, to acknowledge the challenges associated with learning model parameters from available data.

As opposed to aggregated incidence/prevalence counts in classical epidemic models and metapopulation models (Danon et al., 2021; Kermack & McKendrick, 1927), individual-based models (IBMs), such as the influential CovidSim model (Ferguson et al., 2020), explicitly consider individuals' disease states. This provides a refined representation of the population and in principle allows practitioners to work with covariates defined at an individual level (e.g. health records, geographical locations, contact networks). A simple example is a regression modelling of the rates at which the kth individual infects the nth: $\log \beta_{nk} = \log \beta + \mathbf{c}_n^\top \mathbf{b}_S + \mathbf{c}_k^\top \mathbf{b}_I$, where $\mathbf{c}_n$, $\mathbf{c}_k$ are observed covariates associated with the n, kth individuals and β, $\mathbf{b}_S$, $\mathbf{b}_I$ are model parameters respectively controlling background infection and how covariates contribute to attracting (susceptibility) and transmitting (infectivity) infection. This sliding scale of heterogeneity can be linked to the population distribution of the individual specific susceptibility parameter α of the model defined in subsection 4.1 of Wood et al. (2025).

Since transmission processes are typically only partially observed, exact statistical inference, e.g. aimed at learning regression parameters β, $\mathbf{b}_S$, $\mathbf{b}_I$ or informing properties of α, requires marginalization over the latent disease state of the entire population. This incurs a prohibitively expensive cost which is exacerbated by increasing individual heterogeneity, motivating the development of approximate inference methods (Bu et al., 2024; Ionides et al., 2023; Rimella, Jewell, et al., 2025; Rimella, Whiteley, et al., 2025; Whitehouse, 2025; Whitehouse et al., 2023). Furthermore, the identifiability of models incorporating individual heterogeneity, in general, depends on the observation model, for instance whether individual test results or aggregated incidence/prevalence counts are reported. In particular, Whitehouse et al. (2023) and Rimella, Whiteley, et al. (2025) show that, in the context of both metapopulation models and IBMs, parameters defining processes with coinciding large population behaviour cannot be distinguished.

We are curious about the balance between realism and feasibility and how the research community will address this in the future: given the prohibitive cost of fitting increasingly heterogeneous models, how far should modellers go? In the presence of identifiability issues, how should modellers acknowledge and account for these limitations? Will further development of efficient inference methods, identifiability conditions, and optimal observation sampling, e.g. in a Bayesian experimental design framework, benefit future outbreak response efforts?

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Jason Wyse, Dhorasso Temfack, Eishita Yadav and James Sweeney's contribution to the Discussion of 'Some statistical aspects of the Covid-19 response' by Wood et al.

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We congratulate the authors on a thought-provoking and timely paper evaluating the effectiveness and impact of decisions made during the COVID-19 pandemic on society at large. The authors produce compelling arguments as to why some of the approaches taken to modelling, model checking, and prediction may not have been optimal. They suggest that narratives around disease risks and the benefits of disease mitigation strategies, communicated to the public, did not meet statistical best practice. We fully agree with their sentiments on the inappropriateness of data being made available only to siloed groups as opposed to the research community at large.

However, while robust, we do wonder if the authors' criticisms of the models that were used fully appreciate the context in which they were built and deployed. We have applied the Section 5 code to Irish death case data where it performs well on a long time series of 1.5 years, matching the timeframe of the analysis in the paper. However, at pandemic outset where data is sparse and the pathogen poorly understood, how well would the approach work from an advisory perspective? The first lockdown was announced in the UK in March 2020; however, we note that the paper is reliant on a metanalysis (McAloon et al., 2020) that was published in July 2020. How would the authors' approaches have performed using contemporaneous estimates of infection to death duration and data available at time of first lockdown in March 2020? We also wonder how the authors' framework would perform for diseases characterized by abrupt changes in the disease dynamics, especially to reconstruct the change in R_t (Section 5 of the paper). Can the smoothness assumption of the model lead to underestimating the sharpness of real changes, causing the