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Capturing superspreading patterns of SARS-CoV-2 with phylodynamics

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Phylodynamic approach affords an in-depth view of emergence and spread of infectious diseases by allowing characterization of epidemiological dynamics based on sequentially sampled viral sequences. Most often the aim is to infer an estimate of reproductive number that describes the average number of secondary infections per infectious individual. As well as for many other viral agents for SARS-CoV-2 superspreading has been recognized as an important driver of disease transmission. This implies that a small number of individuals are responsible for a disproportionately large number of secondary infections. Superspreaders and superspreading events are traditionally identified by contact tracing, albeit such a practice is not always feasible. Nevertheless, broader understanding of transmission inequality is crucial as it might provide enhanced tools to prevent and manage viral outbreaks.

Despite phylodynamics being routinely used to make epidemiological inferences, little is known how accurately the approach can infer the signals of superspreading. We address this question with a simulation study within a Bayesian statistical framework. By applying a structured birth-death model on simulated data we evaluate whether transmission heterogeneity can be captured purely based on viral sequences. Additionally, by examining two sets of SARS-CoV-2 sequences from Germany – one for which the superspreading event has been validated by contact tracing and another for which the same information is lacking – we further quantify the performance of the model. Our preliminary results suggest that the model is capable of reconstructing the underlying transmission dynamics indicating that phylodynamics might provide an alternative way to identify the patterns of superspreading.