4.93 vs. 4.73). There was no difference in presence of transmitted drug resistance. This study confirms the current epidemiological knowledge of local spread of HIV-1 in Belgium and provides a solid base for more in-depth characterization of transmission and for future real-time follow-up of cluster dynamics.

A6 Using phylodynamic modelling to estimate the population attributable fraction of HIV spread due to key populations in Dakar, Senegal

E. Geidelberg and E. Volz

Imperial College London, London, UK

Although it has long been believed that certain key populations contribute disproportionately to HIV infection, the proportion of transmission events attributable to them is poorly understood. Most existing methods for estimating the population attributable fraction (PAF) are derived from the proportion of prevalent infections found in each group, or from static modes of transmission studies. Although these methods are useful in obtaining a cross-sectional estimate of the fraction of incident infections acquired in each group, they do not take into account the chain of transmission, and thus may underestimate the contribution of key populations. Using a transmission dynamics model, we aim to estimate the PAF of female sex workers, clients, and men who have sex with men to the HIV epidemic of Dakar, Senegal. On top of behavioural and epidemiological data, we will have access to genetic data from these key populations from an ongoing study, as well as historical samples from the Los Alamos database. As genetic diversity is shaped by epidemiological history, population genetic modelling of our sequence data can be informative about epidemic size and the migration of lineages through space and between risk groups. Our model will be first parameterised and fitted to behavioural and epidemiological data. We will then perform a phylogenetic analysis on our sequence data, using known dates of sampling and a molecular clock model of sequence evolution. Using structured coalescent models, we can look at the balance of phylogenies and infer patterns of transmission (although we will not have a large enough sample to determine clusters). We can then refit the transmission model to the sequence data as well, and provide new estimates of the PAFs. The comparison of PAFs estimated with or without using sequence data will provide an insight into the added value of phylodynamic modelling, and may help reassess the role of key populations in this setting.

A7 The effect of the mechanism and amount of missingness on phylodynamic inference of heterosexual HIV transmission networks

David Niyukuri,[1] and Wim Delva[1,2,3,4]

[1]South Africa Centre of Epidemiological Modelling and Analysis, Stellenbosch University, South Africa, [2]Department of Microbiology and Immunology, Rega Institute for Medical Research, Clinical and Epidemiological Virology, KU Leuven, Belgium, [3]Center for Statistics, I-BioStat, Hasselt University, Belgium and [4]International Centre for Reproductive Health, Ghent University, Belgium

Successful HIV prevention requires a better understanding of the structure and the dynamics of the sexual network. The latter evolves over time and the transmission network emerges from that dynamic sexual network. At the same time, the genetic material of the virus evolves across the transmission network. Due to the fact that population-level HIV transmission dynamics and HIV evolutionary dynamics are on the same time scale, the molecular evolution of the virus becomes a footprint of the transmission network. Therefore, the analysis of HIV phylogenetic trees holds the promise to provide more objective methods to estimate transmission network characteristics. We aimed to investigate the effect of sub-optimal sequence coverage on the characteristics of the transmission network (e.g., degree distribution and link density) inferred from the phylogenetic tree. We simulated a small epidemic (seventy-two transmission events) using agent-based models. Across the transmission chain, the molecular evolution of the virus was simulated using appropriate substitution models and evolutionary rates for HIV-1. We considered one consensus sequence per individual, and we simulated five levels of sequence coverage. In addition, we simulated two sampling strategies: a cross-sectional sampling design at the end of the simulation time window, and longitudinal sampling design. For each level, we constructed a phylogenetic tree and the subsequent transmission network, which were compared to the true transmission network. We found that a reconstructed transmission network from a phylogenetic tree has characteristics close to those of the true transmission network when sequence coverage was at least 60 per cent of the infected individuals (forty-five sequences). The increase of taxa (sequence coverage) improves the inferred transmission network characteristics. In addition, transmission networks reconstructed with the cross-sectional sampling design had less overestimation of links (which are seen as potential transmission events).

A8 Improving the accuracy and precision of estimated temporal trends in HIV incidence among MSM populations by calibrating agent-based simulation models to phylogenetic tree data

W. Delva,[1,2,3,4] N. Hens,[3,5,6] and A.-M. Vandamme[2,7]

[1]South Africa Centre of Epidemiological Modelling and Analysis, Stellenbosch University, South Africa, [2]Department of Microbiology and Immunology, Rega Institute for Medical Research, Clinical and Epidemiological Virology, KU Leuven—University of Leuven, Belgium, [3]Center for Statistics, I-BioStat, Hasselt University, Belgium, [4]International Centre for Reproductive Health, Ghent University, Belgium, [5]Centre for Health Economic Research and Modelling Infectious Diseases, Vaxinfectio, University of Antwerp, Belgium, [6]Epidemiology and Social Medicine, University of Antwerp, Belgium and [7]Center for Global Health and Tropical Medicine, Unidade de Microbiologia, Instituto de Higiene e Medicina Tropical, Universidade Nova de Lisboa, Portugal

Effective prevention of sexually transmitted HIV infections requires knowledge of the sexual networks across which these infections are transmitted. Sexual behaviour surveys are routinely being conducted to ask people about their recent sexual partners, leading to emergence of star-shaped egocentric network data. At the same time, phylogenetic tree analysis has long been conducted to analyse HIV transmission clusters. In an ongoing project, we aim to show that (1) agent-based simulation models, embedded in a Bayesian framework, can provide a platform for combining these complementary data sources, and that (2) such an integrated approach can lead to more accurate and robust inferences in HIV epidemiology. Within this project, a first case study aims to provide evidence that phylogenetic tree data can increase the validity of agent-based model projections. Specifically, in a small-scale proof-of-concept study, we use synthetic data from a 'master model' to demonstrate that the accuracy and precision of estimated temporal trends in HIV incidence improves when agent-based simulation models of HIV transmission in MSM populations in Western Europe are not only calibrated to reported behavioural and epidemiological data, but also to phylogenetic tree data. Our model calibration