Annual meeting of the Coordinated Action « Modelling infectious diseases »

13-15 November 2024

Wednesday 13 NOV. "Young Researcher Day"
Thursday 14 NOV.

9:30-9:40 Opening talk

Session 1 (Chairs: Pierre-Yves Boëlle and Juliette Paireau)
9:40-10:15 **Florence Débarre** (iESS Paris)- What modelling can bring to the search for the origin of the COVID-19 pandemic
10:15-10:40 **Charlotte Perlant** (MMMI)- Investigating spatial patterns of the 1892 Cholera epidemic in France.
10:40-11:05 **Pascal Crépey** (EHESP) - Epidemiological modeling to assess early response strategies to cholera epidemics: a case study for humanitarian actors in Cameroon

11:05-11:35 *Coffee break*

Session 2 (Chairs: Mircea Sofonea and Camille Pelat)
11:35-12:00 **Vincent Garot** (LCQB/CIRB)- Transformers for EpiDemiological DYnamics: from genomic data to epidemiological parameters
12:00-12:05 **Suprabhath Kalahasti** (IPLESP) - Transfer learning to inform the spread of other respiratory viruses : Application to Influenza using COVID19
12:05-12:10 **Georges Shirreff** (EMAE/MESuRS)- Epidemiological interactions between Influenza and Respiratory Syncytial Viruses
12:10-12:45 Moderator: **Amandine Véber** (MAP5)- Presentation of the AC activities

12:45-14:15 *Lunch break*

Session 3 (Chairs: Raphaëlle Metras and Olivier Supplisson)
14:15-14:40 **Laura Temime** (MESuRS)- Teleworking and health in an epidemic context: contrasting the infectious and non-communicable diseases perspectives
14:40-15:05 **Sophie Chervet** (EMAE) - Impact of age and intra-household non-pharmaceutical interventions on the SARS-CoV-2 transmission in French households - comparison of alpha and omicron variants
15:05-15:30 **Antoine Brault** (MMMI) - Effect of nirsevimab on hospitalisations for respiratory syncytial virus bronchiolitis in France, 2023&24: a modelling study

15:30-16:00 *Coffee break*

Session 4 (Chairs: Samuel Alizon and Constanze Ciavarella)
16:00-16:25 **Louis Colliot** (CIRB) - Modelling the HIV epidemic in France using virus genomic data
16:25-16:50 **Amber Kunkel** (SpF) - A new approach to estimating HIV incidence and the size of the undiagnosed HIV+ population in France, accounting for migration

Session on Funding opportunities of ANRS-MIE (Chairs: Amandine Véber and Nathanaël Hoze)

16:50-17:00 Presentation of the laureates of the 2024 ANRS-MIE PhD scholarships

Bastien Brebel (IAI) - Combining interactomics and artificial intelligence in the fight against emerging viral threats

David Nahmani (LAGA) - Mathematical modeling and analysis of heterogeneities in arboviroses control techniques

17:00-17:20 Presentation of funding opportunities of ANRS-MIE and questions (Zélie Godin & Inmaculada Ortega Perez, ANRS-MIE)

17:30-19:00 **Poster session**

19:45 - Conference Dinner at La Réserve, 36-38 rue de la Visitation, Rennes.

Friday 15 NOV.

Session 5 (Chairs: Lulla Opatowski and Benjamin Faucher)

9:00-9:25 **Eugenio Valdano** (IPLESP) - Estimates of the reproduction ratio from epidemic surveillance may be biased in spatially structured populations

9:25-9:50 **Kacem Lefki** (LAMA) - An infinite dimensional metapopulation SIS model with generalized incidence rate

9:50-10:15 **Lucille Calmon** (EPIcx) - Preserving friendships in school contacts: an algorithm to construct synthetic temporal networks for epidemic modelling

10:15-10:40 **Maylis Layan** (EMAE/MESuRS) - Modeling the nosocomial transmission of respiratory infections by coupling close-proximity interactions and aerosol-mediated long-distance transmission routes

10:40-11:10 *Coffee break*

Session 6 (Chairs: Mélanie Prague and Camille Schneider)

11:10-11:15 **Presentation of the new permanent researchers**

11:15-11:40 **Adrien Mitard** (IAME/SISTM)- Modelling antibody levels impact on infection and SARS-CoV2 replication

11:40-12:05 **Nicolas Boespflug** (BPH/VRI)- A mechanistic model of initial and persisting antibody response following Ebola vaccination in the PREVAC trial

12:05-12:30 **Younjung Kim** (IPLESP)- Modelling the life cycle dynamics of medically important ticks: insights from *Ixodes ricinus* in Alsace, France

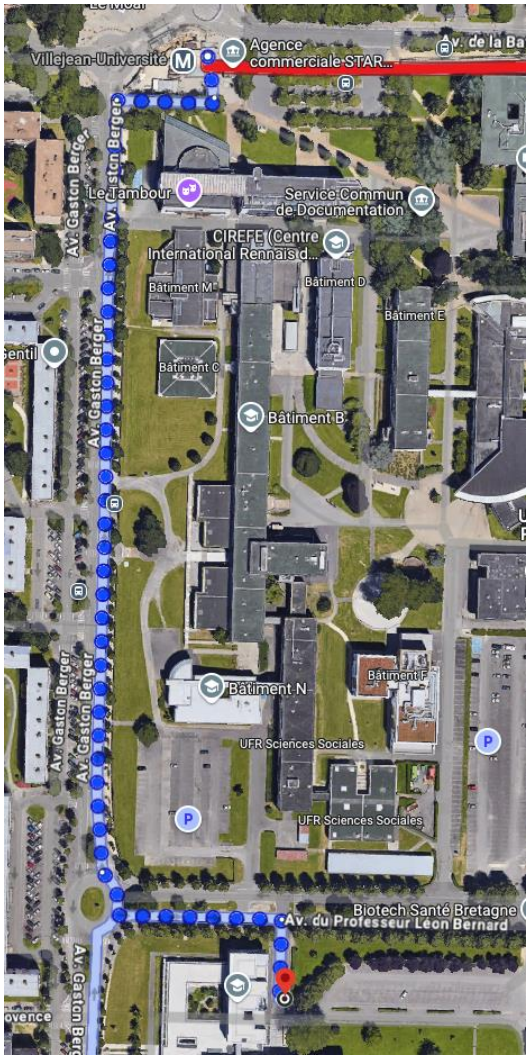
12:30-14:00 *Lunch break*

End at 14:00

Access to the meeting

The meeting will take place in the main building of EHESP located at 15 avenue du Pr. Léon-Bernard in Rennes, in the Simone Veil amphitheater.

The easiest access from Rennes train station is by metro line A towards Kennedy, stopping at Villejean-Université. Once you arrive at the station, you will only have a 5-minute walk (600m) straight south along Avenue Gaston-Berger.



The bus line C4 is also available for those of you wanting to avoid the subway.

The details of the route are available on this link or this QR code:

A mechanistic model of initial and persisting antibody response following Ebola vaccination in the PREVAC trial

Nicolas Boespflug[1,2], Marie Alexandre[1,2], Abdoul Habib Beavogui[3], Seydou Doumbia[4], Mark Kieh[5], Bailah Leigh[6], Samba O Sow[7], Rodolphe Thiébaut[1], Yves Lévy[8], Yazdan Yazdanpanah[9], Laura Richert[1], Edouard Lhomme[1], and Mélanie Prague[1]

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5: Partnership for Research on Ebola Virus in Liberia (PREVAIL) – Libéria
6: College of Medicine and Allied Health Sciences (COMAHS) – Sierra Leone
7: Centre pour le Développement des Vaccins, Ministère de la Santé et du Développement Social du Mali – Mali
8: Vaccine Research Institute [Créteil, France] – Inserm U955; Hopital Henri Mondor; Univ. Paris-Est; VRI; – France
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Antibody response dynamics following Ebola vaccination remain incompletely under- stood, particularly regarding the continuum of initial induction to long-term persistence. This study developed a mechanistic NLME model of B-cell stimulation post-vaccination able to infer antigen presentation kinetics and propose an identifiable model based solely on anti- ZEBOV IgG levels.

This study was based on the PREVAC randomized placebo-controlled trial (NCT02876328), which enrolled healthy adults and children to evaluate the safety and immune responses of three vaccine strategies: Ad26.ZEBOV followed by MVA-BN-Filo 56 days later (the Ad26– MVA group, 799 participants), rVSVΔG-ZEBOV-GP followed by placebo 56 days later (the rVSV group, 802 participants), and rVSVΔG-ZEBOV-GP followed by rVSVΔG-ZEBOV- GP 56 days later (the rVSV–booster group, 399 participants).

Our model was modified from Clairon et al. (2022, PLOS Comp. Biol.), which assumes that antigen stimulates the differentiation of naive B cells into long-lived and short-lived antibody-secreting cells. The two groups were modelled following similar pipelines. We compared vaccine exposure using a decreasing exponential or a bell-shaped curve. Parameters were derived from literature on vaccine viremia and distribution or estimated using data-driven approaches.

For both vaccine groups, a bell-shaped curve best described the dynamics according to model information criterion (BICc) indicating a long antigen presentation regardless of replication competence. Longer presentation times were found for rVSV (half-life t1/2=6.9 days at first dose and t1/2=0.07 days at second dose) than for Ad26–MVA (t1/2=2.77 days for Ad26.ZEBOV and t1/2=1.39 days for MVA-BN-Filo). Overall fits showed good adjustment but usually underestimated peak antibody concentrations, especially after first injection or in the highest responders.

While our model is one of the first attempts to better understand antigen presentation ki- netics, this study shows limitations due to limited observation of B cell responses. This work opens the door to detection of additional markers that could be measured to enhance identifiability.

Effect of nirsevimab on hospitalisations for respiratory syncytial virus bronchiolitis in France, 2023–24: a modelling study

Antoine Brault[1], Isabelle Pontais[2] , Vincent Enouf[5,7,8], Christine Debeuret[3], Emma Bloch[8], Juliette Paireau[1,4], Marie-Anne Rameix-Welti[5,6,7], Michael White[8], Gaëlle Baudemont[8], Bruno Lina[9], Isabelle Parent Du Châtelet[4], Jean-Sébastien Casalegno[8], Sophie Vaux[4], and Simon Cauchemez[1]

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Background

Respiratory syncytial virus (RSV) is a major cause of hospitalisations and deaths among infants worldwide. France was one of the first countries to implement a national programme (beginning on Sept 15, 2023) for administration of nirsevimab, a single-dose long-acting monoclonal antibody treatment, to infants born on or after Feb 6, 2023, to prevent lower respiratory tract infection caused by RSV. We aimed to estimate the effectiveness of nir- sevimab and the number of hospitalisations averted in children younger than 24 months in real-world settings.

Methods

In this modelling study, we developed an age-structured deterministic model characteris- ing RSV transmission as well as plausible scenarios for the administration of nirsevimab doses based on maternity ward and community pharmacy supply data. We retrospectively estimated nirsevimab effectiveness in infants younger than 24 months during the 2023–24 RSV season in France (excluding overseas territories) and the number of averted hospital- isations for RSV bronchiolitis occurring after emergency department visits, by calibrating the model to hospital and virological surveillance data from Aug 21, 2017, to Feb 4, 2024, alongside serological data from a previous cross-sectional study. To assess the robustness of our estimates, we conducted sensitivity analyses in which we modified our assumptions about the number of doses administered, the reconstruction of the number of RSV-associated hospitalisations for bronchiolitis, the duration of maternal and post-infection immunity to RSV, and the number of contacts in children aged 0–2 months.

Findings

We estimated that nirsevimab administration prevented 5800 (95% credible interval 3700– 7800) RSV-associated hospitalisations for bronchiolitis after emergency department visits among children younger than 24 months, including 4200 (2900–5600) hospitalisations among those aged 0–2 months, between Sept 15, 2023 (the date nirsevimab was introduced), and Feb 4, 2024-a 23% (16–30) reduction in the total number of hospitalisations and a 35% (25–44) reduction in the 0–2 months age group, compared with the scenario without administration. In our baseline scenario, in which we estimated that 215000 doses of nirsevimab were admin- istered by Jan 31, 2024, the estimated effectiveness against RSV-associated hospitalisations for bronchiolitis was 73% (61–84), corresponding to one hospitalisation averted for every 39 (26–54) doses administered. In sensitivity analyses, nirsevimab remained effective against RSV-associated hospitalisations for bronchiolitis after emergency department attendance.

Interpretation

Our findings show that nirsevimab administration campaigns could effectively reduce the RSV-related hospital burden of bronchiolitis in children younger than 24 months.

Combining interactomics and artificial intelligence in the fight against emerging viral threats

Bastien Brebel[1,2,3,4,5], Marc Vidal[3,4,5], and Caroline Demeret[1]

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2: École Doctorale Bio Sorbonne Paris Cité – Université Paris Cité – France
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5: Department of Cancer Biology, Dana-Farber Cancer Institute – États-Unis

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This project develops an innovative pipeline combining high-throughput interactomics and artificial intelligence to rapidly analyze emerging viral threats. Our interactomics approach comprehensively maps virus-virus and virus-host protein interactions at scale. We then leverage AI, including protein and genomic language models, to predict viral protein functions and prioritize critical interactions. Machine learning algorithms help design pep- tides to disrupt these key interactions. By integrating large-scale interaction data with AI-driven predictions, we aim to accelerate therapeutic responses to future pandemics, even before traditional culture models are available

Preserving friendships in school contacts: an algorithm to construct synthetic temporal networks for epidemic modelling

Lucille Calmon[1], Elisabetta Colosi[1,2], Giulia Bassignana[1], Alain Barrat[3], and Vittoria Colizza[1,4]

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4: Department of Biology, Georgetown University – États-Unis

High-resolution temporal data on contacts between hosts provide crucial information on the mixing patterns underlying infectious disease transmission. Publicly available data sets of contact data are however typically recorded over short time windows with respect to the duration of an epidemic. To inform models of disease transmission, data are thus often re- peated several times, yielding synthetic data covering long enough timescales. Looping over short term data to approximate contact patterns on longer timescales can lead to unrealistic transmission chains because of the deterministic repetition of all contacts, without any renewal of the contact partners of each individual between successive periods. Real contacts indeed include a combination of regularly repeated contacts (e.g., due to friendship relations) and of more casual ones. In this paper, we propose an algorithm to longitudinally extend contact data recorded in a school setting, taking into account this dual aspect of contacts and in particular the presence of repeated contacts due to friendships.

To illustrate the interest of such an algorithm, we then simulate the spread of SARS-CoV-2 on our synthetic contacts using an agent-based model specific to the school setting. We compare the results with simulations performed on synthetic data extended with simpler algorithms to determine the impact of preserving friendships in the data extension method. Notably, the preservation of friendships does not strongly affect transmission routes between classes in the school but has a clear impact on the infection pathways between individual students. Our results moreover indicate that gathering contact data during two days in a population is sufficient to generate realistic synthetic contact sequences between individuals in that population on longer timescales. The proposed tool will allow modellers to leverage existing contact data, and contributes to the design of optimal future field data collection.

Impact of age and intra-household non-pharmaceutical interventions on the SARS-CoV-2 transmission in French households – comparison of alpha and omicron variants

Sophie Chervet[1,2,3], Maylis Layan[1,3,4], Sylvie Behillil[5,6], Naïm Bouazza7, Nelly Briand7, Agnès Delaunay-Moisan[8], Flora Donati[6,9], Vincent Enouf[6,10,11], Marianne Leruez-Ville[12,13], Faheemah Padavia[7], Chloé Sturmach[6], Simon Cauchemez[14], Solen Kernéis[2,15], Jérémie Guedj[2], Sylvie Van Der Werf[16], Isabelle Sermet-Gaudelus[17,18], and Lulla Opatowski[1,3]

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13: URP FOETUS – Université Paris Cité – France
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Households play an important role in community transmission of respiratory viruses. However, the implementation of non-pharmaceutical interventions at home is a challenge, and their usefulness is poorly informed due to lack of data. Here, we study the impact of age, symptoms and intra-household non-pharmaceutical interventions on the transmission of SARS-CoV-2 in French households, by analysing longitudinal data collected from households over the periods of circulation of variants alpha and omicron (PedCovid study). Households were enrolled after identification of a paediatric confirmed SARS-CoV-2 infection, and fol- lowed up for 45 days. Visits included PCR tests, questionnaires informing on age, symptoms and protective measures implemented in the household (isolation, mask wearing, surface disinfection). We developed a dynamic transmission model accounting for these individual characteristics and used Bayesian inference with data augmentation to estimate person-to- person risks of transmission. A total of 128 and 54 households were recruited during the alpha and omicron periods, respectively, representing 535 and 234 individuals, respectively. The overall median age of participating children and teenagers

was 9 years old (IQR=5-12.25). Estimates of relative infectivity and susceptibility were found to vary by age, symptoms, and implemented measures during the alpha period. Symptomatic infants (< 6 years old) were 0.36 times (95% CrI: 0.13-0.78) less infectious than symptomatic adults, and all asymp- tomatic individuals were less infectious than symptomatic adults. During the same alpha period, we estimated that strict confinement of an infected individual reduced transmission by a factor 0.53 (95% CrI: 0.24-0.95). By opposition, we did not find any effect of surface disinfection or social distancing and mask wearing on transmission. Interestingly, no impact of age, symptoms or protective measures was found during the omicron period. This study highlights different transmission patterns between alpha and omicron periods in France and provides insights into the global transmission of respiratory viruses in households.

Modelling the HIV epidemic in France using virus genomic data

Louis Colliot[1], Samuel Alizon[2], and Laurence Meyer[3,4]

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Every time the Human Immunodeficiency Virus (HIV) replicates within a cell, errors can creep into its genome. These mutations make it possible to track an epidemic, because the more similar two virus sequences are, the closer the two individuals who bear them are likely to be in the chain of transmission. For more than twenty years, the field of phylodynamics has been analyzing this type of data to estimate, for example, epidemic growth rates or dispersion rates between regions. The SARS-CoV-2 pandemic illustrated the power of these methods, which remain marginal in France where routine sequencing performed during HIV screening is rarely used for epidemiological surveillance. However, this method is adapted to the HIV epidemic, it could inform us about the presence of clusters or the scale of the 'hidden' epidemic, which, by definition, is difficult to estimate because it is not screened. Thanks to the collaboration with leading national clinical virology teams, we analyze data from French cohorts, namely the national PRIMO cohort or the HIV-OE cohort at Montpellier University Hospital. Preliminary results allow us to place the French epidemic in a global context and to successfully characterize homogeneous clades for the most common circulating subtype (HIV1-B). This better understanding of the structure of the French HIV epidemic will help guide future phylodynamic analyses to estimate variations in incidence and prevalence in overexposed populations over time. In particular, we will focus on assessing the proportion of the epidemic that is hidden to optimize public health policies.

Epidemiological modeling to assess early response strategies to cholera epidemics: a case study for humanitarian actors in Cameroon

Pascal Crépey[1], Anne-Laure Maillard[2], Mélanie Droogleever Fortuyn , Annemieke Brouwer, and Marc Van Den Homberg

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2: Santé publique France – Direction des régions Cellule région Bretagne – Rennes, France

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Cholera is an acute diarrheal infection caused by ingesting food or water contaminated with Vibrio cholerae. It is estimated to affect 1.3 to 4 million people per year, causing 21,000 to 143,000 deaths. This burden is under-reported and mainly affects developing countries with sub-optimal surveillance systems. The WHO Global Task Force on Cholera Control aims to eliminate cholera by 2030 in 20 countries, including Cameroon. Cameroon has experienced cholera epidemics since 1971. From mid-June 2022 to mid-June 2023, 19,087 cases of cholera were reported, with 1,880 confirmed cases and 450 deaths. Humanitarian actors are heavily involved in the response to cholera epidemics. An early action protocol is being drawn up to enable the Cameroon Red Cross to anticipate the spatial dissemination of cholera outbreaks. It aims to trigger the necessary funding based on forecasts. To this aim, we developed a stochastic meta-population epidemiological model to reproduce the cholera spreading dynamics. Due to constraints on the availability of epidemiological data and population flows, we divided the country into patches corresponding to administrative areas relevant to health authorities. Those patches are then connected by directed population flows whose strength is estimated with a gravity model. We used a Bayesian framework to infer propagation parameters consistent with recent cholera epidemics, allowing us to generate possible epidemiological scenarios. The model can then assess the impact of various prevention strategies, from targeted vaccination to water chlorination. This work paves the way for new approaches enabling real-time epidemiological modeling and dynamic adaptive policy pathways to inform public health decision-making.

What modelling can bring to the search for the origin of the Covid-19 pandemic

Florence Debarre[1]

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When and where did the Covid-19 pandemic start ? I propose to give an overview of the way in which modelling work can shed light on these questions, and how different approaches bring – or not – the same answers, while underlining the limits of the different models and of available data.

Transformers for EpiDemiological DYnamics: from genomic data to epidemiological parameters

Vincent Garot[1,2], Luca Nesterenko[3], Luc Blassel[1,4], Anna Zhukova[5], Samuel Alizon[2], and Laurent Jacob[1]

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The way a virus spreads leaves footprints in its genome. Phylodynamics leverages these footprints to estimate epidemiological parameters from collected virus genetic data. The estimation is typically done in a likelihood-based framework. The epidemiological process is modeled on a virus transmission tree. This tree is approximated by time-scaled phylogenetic trees reconstructed from virus sequences. However, as the epidemiological models become more realistic, their complexity increases, and the likelihood might become intractable, impeding the use of standard likelihood-based inference methods.

We introduce Teddy, a likelihood-free inference method where likelihood computations are replaced by data sampling from the epidemiological model. More precisely, we use this data to learn a function that takes observed data (dated virus sequences) and returns a posterior distribution of the epidemiological parameters given the data. Our function is parameterized by a neural network, with self-attention layers to handle permutation invariances among sequences and positional embeddings to incorporate the dates. The output contains an estimation of the epidemiological parameters and a measure of uncertainty in the form of credible intervals.

Under the common and tractable birth-death model on simulated data and early COVID data, the inference obtained by Teddy matches the one obtained by BEAST, a state-of-the-art Bayesian inference method relying on MCMC. Unlike BEAST, however, Teddy does not require tree reconstruction or likelihood evaluation. We also show that model mispecifications have the same effect on Teddy and BEAST. These results are a proof of concept and suggest that Teddy may allow inference under models where likelihoods are intractable and BEAST could not be used.

Transfer learning to inform the spread of other respiratory viruses : Application to Influenza using COVID19

Suprabhath Kalahasti[1], Eugenio Valdano[1,2], and Pierre-Yves Boëlle[1,3]

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The COVID-19 pandemic contributed to the generation of surveillance data at a larger scale than what was done traditionally by public health systems. Case counts, hospitalization rates, and deaths coupled with data on human behaviour and mobility patterns were accessible to enable swift evidence-based public health responses. These datasets, however, are typically rarely re-usable: they describe epidemic and human dynamics for a specific disease, a specific population, and a point in time. It is thus unclear how to extract information from those data that can be extrapolated and generalized to other public health emergencies for which fewer data, or data at a lower resolution, might be available. Transferring knowledge using traditional models is hard: it is conceivable that, for instance, epidemic waves of COVID-19 and influenza might share some similarities - they are both directly transmitted, respiratory pathogens. Analogously, epidemic waves of the same disease in neighbouring regions might also be similar. Quantifying and exploiting this similarity using traditional models requires manual identification of features and explicit fit to the data to allow/enable the transfer of knowledge. These assumptions may be arbitrary and unjustified.

In this project, we aim to overcome this by using data from the COVID-19 pandemic to inform and learn the dynamics of epidemics caused by other respiratory pathogens. The scientific goals of the projects entail:

- Use model agnostic metal learning (MAML) to enable the transfer of predictability between different contexts.

- Combine deep learning and mathematical modelling approaches to improve the knowledge and predictability of seasonal respiratory airborne epidemics and generate reliable forecasts.

- Using an online survey collected from participants across the globe during the wartime pandemic period (2020-2022), use unsupervised learning approaches and identify subgroups of the population in terms of similar behavioural patterns and develop risk profiles to parametrize mathematical models and build scenarios of disease spread.

Modeling the life cycle dynamics of medically important ticks: insights from Ixodes ricinus in Alsace, France

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Ixodes ricinus is the primary vector for Lyme disease and tick-borne encephalitis across Europe. To better understand the environmental and ecological drivers of its population dynamics, we collected monthly tick density data over ten years (2013–2022) in four sites in Northeast France, where *I. ricinus* has established, and developed and fitted a mathematical model using a Bayesian approach. Our model estimated oviposition, hatching, and moulting rates across a range of temperature or saturation deficit, as well as questing and vertebrate host contact rates. Furthermore, we showed the importance of diapause in reproducing the observed seasonal population dynamics. Finally, model projections indicated a significant decrease in *I. ricinus* abundance over the next 20 years under several climate change scenarios. This study elucidates *I. ricinus* population dynamics in Northeast France, provides foundations for developing models of *I. ricinus*-borne pathogen transmission, and is adaptable to other Ixodidae populations of public health significance.

A new approach to estimating HIV incidence and the size of the undiagnosed HIV+ population in France, accounting for migration

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Background:
Existing approaches to estimating HIV incidence and the size of the undiagnosed HIV+ pop- ulation do not properly account for pre-migration infections. We developed a new method to address this limitation and tested the method's performance on simulated and real data from the French HIV surveillance system.

Methods:
First, we applied a pre-existing Bayesian model based on CD4, clinical stage, and previ- ous negative test date to estimate individual-level posterior distributions of infections times for each person newly diagnosed with HIV from 2012 to2023. Pre- and post-migration infections were separated by comparing the date of arrival in France with the estimated date of infection. To estimate yearly HIV incidence in France, draws from the posterior distribu- tions of infection times were combined with methods previously developed to estimate AIDS reporting delays. A similar approach was used to estimate the number of people arriving in France with undiagnosed HIV infection per year. To estimate the size of the undiagnosed HIV+ population at the end of 2023, we added up the number of still-undiagnosed HIV incident and infected and undiagnosed migrants each year from 2012-2023, and projected forward from 2024-2042 the yearly number of diagnoses among people infected prior to 2012.

Results:
On simulated data, the model produces estimates of HIV incidence and the size of the undiagnosed HIV+ population that are similar to the true simulated values. On HIV surveil- lance data, our new estimates of HIV incidence and the size of the undiagnosed population in France are understandably lower than previous estimates that did not account for pre- migration infections.

Discussion:
The proposed methods could improve estimates of key HIV epidemic indicators in France and other countries with substantial rates of pre-migration infections.

Modeling the nosocomial transmission of respiratory infections by coupling close-proximity interactions and aerosol-mediated long-distance transmission routes

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Nosocomial transmission of respiratory infections represent an important public health issue for hospitals, with a high risk of transmission affecting both patients and healthcare workers. It is commonly admitted that respiratory pathogens are transmitted through large droplets, emitted while speaking or coughing, which can lead to short-range transmission dur- ing close-proximity interactions, but also through small size aerosols that remain suspended in the air and may cause long-range transmission. Better characterizing the respective roles of close contact and long-distance aerosol transmissions in epidemic dynamics is essential to design effective control strategies. Yet, very few models combine both transmission routes. We developed an agent-based stochastic model of respiratory pathogen transmission in a hospital ward accounting simultaneously for close-contact and aerosol-mediated transmission. We informed our model with real close-proximity interaction data collected in an intensive care unit during the first wave of the SARS-CoV-2 pandemic. Using simulations, we explored five scenarios illustrating a range of respiratory pathogens with different person-to-person transmission rates and individual shedding rates of pathogen-laden aerosols. Parameter val- ues were chosen so that the overall average secondary attack rate (SAR) remained between 20% and 30%. We found that epidemic dynamics (epidemic duration, peak timing, epidemic curves) were overall comparable, with no effect of the transmission scenario. However, when the contact rate increased from 0.25 to 1.5 per day while the shedding rate decreased from 20 to 5 per day, the relative contribution of the contact transmission route increased from 10 to 50%, the SAR among patients increased by 50%, and the SAR among paramedical staff decreased by 13%. In scenarios with higher transmission through contacts, patients were more represented among secondary cases. Our results suggest that considering more precisely physical mechanisms involved in transmission could allow us to adapt intervention measures depending on transmission route relative contributions.

An infinite dimensional metapopulation SIS model with generalized incidence rate

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The SIS epidemic model, first introduced by Kermack and McKendrick, models the spread of an epidemic where infected individuals recover with no immunity, and thus are susceptible again to the disease. This model is usually used for gonorrhea. Many ways to generalize the basic Kermack-McKendrick SIS exist in the literature, we focus in this talk on two of them: - Adding heterogeneity to the model. One can assume that the spreading and recovery rates depend on some parameters, for instance some specific health conditions, geographic position of the individuals,

Considering another model for the infection rate than the law of mass-action. It is usually assumed that the infection rate in a population with S susceptible individuals and I infected individuals is proportional to SI. This assumption does not always fit epidemiological data.

The model we propose in this talk unites all these two aspects. Using some elements of operator theory, we prove that, for any initial condition, the proportion of infected individuals converges to an endemic equilibrium.

Modelling antibody levels impact on infection and SARS-CoV2 replication

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Context and objectives:
Despite unprecedented efforts to tackle a pandemic, SARS- CoV-2 is still circulating at a high rate. Part of the population remains at risk. Therefore, establishing Correlates of Protection against infection but also transmission is crucial. Moreover, we still lack comprehension on the differences between protections offered by vaccination (mono or bivalent) or infection especially in the Omicron context. We addressed those questions using a primate study specifically designed to study those different immunological backgrounds when challenged with Omicron BQ.1.1. Our main objective is to describe the retroactive loop between virus and antibodies. Then, we wish to investigate neutralization and binding levels as mechanistic correlates of protection against infection and transmission as high viral load is associated with higher transmission (1).

Methods and Results:
We used non-linear mixed effect models estimated through likelihood optimization using the SAEM algorithm. We used a target cell limited model to characterize the viral load of infected animals (2). We added an antibody response model where the B cell replication is activated either through vaccination or directly by the modelled viral load. They produce binding antibodies that form immune complexes with free virions inducing a quicker elimination (3). A fraction of them is fully neutralized and can- not infect. Our model also includes antibody-dependent cellular cytotoxicity to explain the inter-individual variability in the elimination of infected cells.
We used our model to propose neutralisation as a Correlate of Protection. We simulated human-like infections depending on the neutralisation at baseline to determine the thresh- old needed to prevent detectable viral load. We were also able to describe its effect on the reduction of the Secondary Attack Rate (4).

Conclusion:
We were able to fit jointly viral load, infectious titers and antibodies dynamics in various groups with very few covariates. Our model could then be used to analyze other complex designs.

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Mathematical modeling and analysis of heterogeneities in arboviroses control techniques

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Dengue fever, zika fever, chikungunya, yellow fever and other arboviruses are transmitted by mosquitoes of genus Aedes, now common from the equatorial and tropical regions to the temperate zones. Except for yellow fever, there exist neither treatment nor vaccine for these diseases, and in such conditions, the main method to control them is to focus on the popula- tion of mosquitoes. The classical control methods, based on insecticides, induce resistance, which reduces their own efficiency, and are detrimental to the environment due to their non- specific action. Among others, the biological control methods, aiming at reducing the size or the vectorial capacity of the wild population, has recently gathered much attention. Sterile Insect Technique is one of them, allowing to reduce or eradicate a wild population thanks to the release of a large amount of sterilized male mosquitoes. Infection by the bacterium Wolbachia is presently another valuable track. This bacterium induces an important decrease in the vectorial capacity of the infected mosquitoes, which seems sufficient to eradicate dengue fever epidemics. In addition, this approach has the advantage to offer robustness against reinvasion, due to the stability of the fully infected equilibrium.

From a mathematical point of view, these two techniques have been widely studied when the environment is assumed to be homogeneous. However, it is clear that environments are not homogeneous neither in space nor in time. The aim of this project is to investigate the heterogeneous situation. Such a study may have an important impact for practical applications. Indeed, it is well known that the dynamics of the mosquito population strongly depends on the season and on the resources available in the environment. Therefore, the strategy should be adapted and optimized to take into account these variations.

The subject of this PhD project is to study qualitatively and quantitatively the effects of seasonal variations and of spatial heterogeneities into the success of control techniques as population replacement by Wolbachia infected mosquitoes or the sterile insect technique.

The following general issues will be considered, in adequate mathematical settings:

Given a seasonal variation, when is the best period of the year to implement the release of Wolbachia-infected mosquitoes or the release of sterilized males? And how to design these releases with respect to these seasonal variations?How may spatial heterogeneities influence the success of replacement strategy and of sterile insect technique? Where should we implement the releases to have the best efficiency?

Investigating spatial patterns of the 1892 Cholera epidemic in France

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Background & Aims of Study
During the XIXth century, Europe faced major Cholera outbreaks, resulting in a high number of deaths and a significant burden on the population. France experienced four Cholera outbreaks during this period, with a major epidemic occurring in 1892. In response, the government deployed medical doctors nationwide to conduct extensive investigations and implement control measures to mitigate the spread of the disease. Extensive data was collected during the outbreak investigation. Here, we develop a statistical mathematical model and employ modern techniques to characterize the drivers of the spatial patterns of the 1892 Cholera epidemic.

Methods & Results
The 1892 dataset represents a rare and comprehensive record of a historical epidemic, including individual-level data for 4,550 reported deaths with variables such as commune and date of death, duration of illness, sex, and age. To understand the observed spatial patterns of spread, we developed city-to-city transmission statistical models inspired by prior research (1). These models incorporate different explanatory variables, such as distances between cities, sizes of the population in each city and changes in the transmission rate over time allowing us to quantify the drivers of spatiotemporal dynamics of the cholera epidemic. Human population movements between communes in France were reconstructed by using a density dependent gravity model formulation.

Parameter space was explored using Bayesian Monte Carlo Markov Chain methods.

Spatial cluster analysis of infected communes suggested that major French ports at the time, acting as hubs of transportation and commerce, played a critical role in the 1892 cholera spread. To account for this feature, we extended our model to incorporate cholera reintroduction via ports. Results revealed that the model accounting for reintroductions from outside France better explain the epidemic's dynamics. Further improvement was achieved by including a higher intensity of transmission from ports to their surrounding communes within a 10 km buffer zone.

Conclusions
This study provides a robust framework for characterizing the spatiotemporal spread of the 1892 cholera epidemic in France and suggests that French ports were major focal points for the introduction and transmission of the pathogen in France. The methodology developed here is adaptable to a broad range of epidemics and invasive processes in epidemiology, accounting for diverse transmission routes. For instance, this approach could be extended to analyze the dispersal patterns of vectors, such as mosquitoes, across Europe.

Epidemiological interactions between Influenza and Respiratory Syncytial Viruses

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Background

Temperate areas experience winter peaks of Influenza and Respiratory Syncytial Virus (RSV) infection, with high burdens of hospitalisation. Partial cocirculation provides op- portunities for co-infection which has been associated with higher severity. In vitro studies demonstrated biological interaction between these viruses, and there is growing population evidence for the inhibition of one virus by the other. Many possible mechanisms have been proposed including resource competition, immune activation and behaviour change, but there is limited evidence to differentiate between them.

Methods

We developed a deterministic compartmental model for the simultaneous transmission of the two viruses, incorporating five possible mechanisms of interaction (modified susceptibil- ity, transmissibility and hospitalisation rates, a refractory period following infection, and a false-positive period following infection). Using Bayesian inference, we fit the model to data from 2014-2020 on weekly numbers of detections of Influenza, RSV or both, among children hospitalised with acute respiratory illness in Valencia, Spain. We estimated the size of each potential interaction, and its effect on burden of disease.

Results

We found positive evidence for inhibition between Influenza and RSV detectable at the population level. In the two best-supported models, current infection with Influenza reduced susceptibility to RSV by 100% (95% credibility interval 56-100%) or reduced the risk of hospitalisation upon infection with RSV by 90% (55-100%). Relative to a model with no interaction, both mechanisms decreased total cases recorded by˜1% but had a much larger effect on the number of recorded co-detections, with reduced susceptibility causing a 31% decrease (17-31%) and reduced hospitalisation a 59% decrease (36-66%).

Discussion

We leveraged long-term data from a hospital network to identify and quantify mechanisms of interaction between Influenza and RSV. These have implications for the risk of co-infection as well as for the quantitative effect of Influenza and RSV vaccination on the overall burden of respiratory infections.

Teleworking and health in an epidemic context: contrasting the infectious and non-communicable diseases perspectives

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Recent years have seen a significant shift towards teleworking. While this escalating, practice can reduce the infection risk for workers during an epidemic, its health impact also encompasses non-communicable diseases (NCDs) such as musculoskeletal disorders or mental health. However, the link between teleworking frequency and NCD risk is unclear. In this study, we aimed to unravel the intricate interplay between teleworking, infectious disease (ID) transmission, and NCD risk, to quantify how these factors could affect a potential optimal teleworking frequency with regards to health outcomes. First, we conducted a rapid review to identify possible exposure-response relationships between teleworking and NCD risk. Then, we designed a mathematical model of ID transmission and NCD acquisition in a medium-sized company. We simulated infection dynamics over a three-month epidemic wave. On weekdays, employees were either physically present at the workplace, with potential exposure to infectious colleagues, or engaged in telework, facing a reduced community-based risk. We compared the results obtained by our model when using different teleworking frequencies and exposure-response curves, to contrast both ID and NCD risks in relation to the extent of telework engagement.

From the literature, we found diverging evidence for the shape of the exposure-response relationship indicating that, depending on the NCD considered, the risk incurred by teleworking may peak at either low, intermediate or high teleworking frequency. Depending on the chosen shape of this relationship and frequency of teleworking, we observed an individual and collective benefit-risk balance between a reduction in ID transmission and a potentially increased burden of NCD.

By acknowledging the dual facets of both infectious and non-communicable health outcomes, our study emphasises the need for a holistic approach when formulating strategies for ID prevention, ensuring that the societal and health impacts of such interventions are comprehensively assessed.

Estimates of the reproduction ratio from epidemic surveillance may be biased in spatially structured populations

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Accurate estimates of the reproduction ratio are crucial for projecting the evolution of an infectious disease epidemic and for guiding the public health response. Here we prove that estimates of the reproduction ratio based on inference from surveillance data can be inaccurate if the population comprises spatially distinct communities, as the space–mobility interplay may hide the true evolution of the epidemic from surveillance data. Consequently, surveillance may underestimate the reproduction ratio over long periods, even mistaking growing epidemics as subsiding. To address this, we use the spectral properties of the matrix describing the spatial epidemic spread to reweight surveillance data. We propose a correction that removes the bias across all epidemic phases. We validate this correction against simulated epidemics and use COVID-19 as a case study. However, our results apply to any epidemic in which mobility is a driver of circulation. Our findings may help improve epidemic monitoring and surveillance and inform strategies for public health responses.

Reference:
Birello et al, Nat Phys Physics 20, 1204–1210 (2024) doi:10.1038/s41567-024-02471-7; free-access link: https://rdcu.be/dFJgn

POSTERS

Maria Alexa (EMAE, Institut Pasteur), Modelling the acquisition of extended-spectrum beta-lactamase-producing enterobacteriaceae (ESBL-PE) in neonates in Madagascar households.

Claudio Ascione (INSERM / Sorbonne Université), How floods may affect the spatial spread of respiratory pathogens: the case of Emilia-Romagna, Italy in May 2023.

Andrée Barnier (MaiAGE, INRAE), Spatial random graph models for the spread of pathogens through commercial animal movements.

Gaëlle Baudemont (Institut Pasteur), Applying sero-catalytic models to multiplex serological data for more accurate assessment of malaria.

Shrichand Bhuria (University of Leeds), Modelling early epidemic dynamics using stochastic birth-death processes: insights from analytical solutions and Gillespie simulation.

Paolo Bosetti (MMMI, Institut Pasteur), Modelling the impact of a quadrivalent ACWY meningococcal vaccination and vaccination targeting serogroup B in France.

Auguste Caen (MiaGE, INRAE / EMRA Institut Pasteur), Identifiability and calibration of the generalized Lotka-Volterra model using microbiota frequency data.

Francesco Camaglia (MMMI, Institut Pasteur), Modeling household transmission dynamics: integrating contact-based and long range mechanisms through flexible analytical tools.

Thomas Cortier (MMMI, Institut Pasteur), Individual SARS-CoV-2 RNA viral load is strongly associated to the risk of transmission within households.

Lina Cristancho-Fajardo (MMMI, Institut Pasteur), Assessing SARS-CoV-2 transmission in African households from the reanalysis of serosurveys.

Elise Hodbert (Nantes Université / MESuRS, CNAM), Exploring multiresistance patterns in community-acquired E. coli UTI with machine learning.

Quentin Leclerc (MESuR, CNAM / EMRA, Institut Pasteur), Modelling the indirect impact of vaccination against viruses on antibiotic resistance.

Aurélien Maurin (MESuRS, CNAM / PACRI, Institut Pasteur), Impact of amoxicillin shortage on pneumococcal resistance and IPD in children: evaluation of antibiotic allocation strategies in European countries.

Hugo Martin (RSMS / EHESP) Less effective but individually less costly prophylactic measures can reduce disease prevalence in a simple epidemic model accounting for human behaviour.

Noé Ochida (MMMI, Institut Pasteur), Reconstructing the history of circulation of CCHFV and RVFV in Senegal and Cameroon from serological data.

Juliette Paireau (MMMI, Institut Pasteur), Integrating information from historical data into mechanistic models for influenza forecasting.

Camille Schneider (EMAE, Institut Pasteur), Understanding antibiotic resistance transmission within and between humans in Klebsiella pneumonia and Escherichia coli – a theoretical modelling study.

Matthew Shin (MMMI, Institut Pasteur), Improving on existing methods to smooth time series data in real time.

Ilona Eveline Suhanda (IPLESP, INSERM), Evaluating the potential of computerized decision support system (CDSS) to improve knowledge of Lyme borreliosis occurrence in France.

Olivier Supplisson (CNRS), Do flood events predict dengue hospital admissions? A Bayesian thinned log-Gaussian Cox Process approach using dengue admissions to the reference hospital for infectious diseases in southern Vietnam, 2015.

Boxuan Wang (IPLESP, INSERM), Optimising HIV pre-exposure prophylaxis eligibility in men who have sex with men in 10 European countries: a modelling study and cost-effectiveness analysis.