Agent-Based Modeling of Early-Stage Influenza

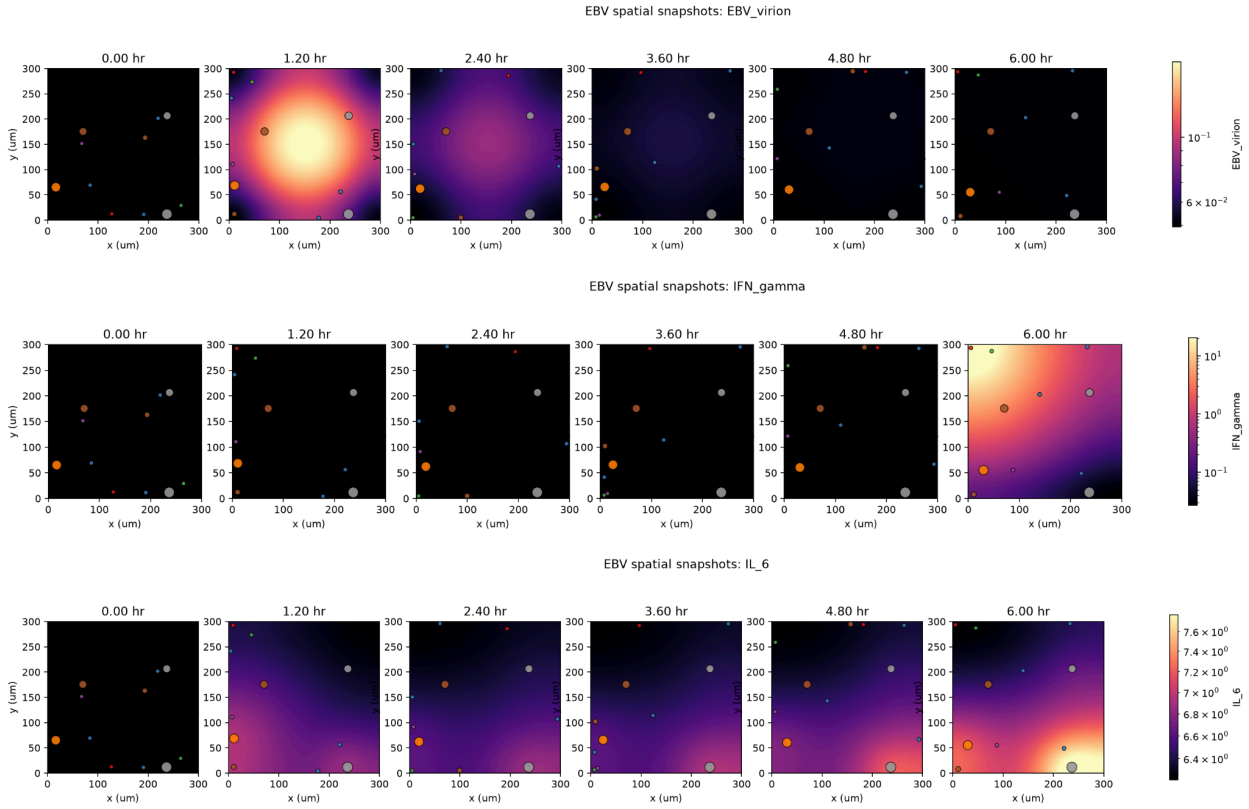
By: Laith AlSenan
laith.alsenan@kaust.edu

This report discusses my work in the ABM of the influenza project. The project has four subtracks: Multiagent, Influenza Modeling, EBV/HIV Modeling, and Vault Lab. My specific subtrack in EBV/HIV modeling. In this report, I'll discuss how this relates to the main project, what I did in my subproject, and the results.

My work in the HIV/EBV subtrack is to extend the multi-agent framework and influenza ABM structure into a lymphoid tissue setting, where chronic viral infections like EBV and HIV interact with immune and stromal cells in a very different environment than the lung. The goal is not just to make a separate EBV model, but to test whether the same agent-based modeling structure can be adapted across diseases while still preserving disease-specific biology.

I helped build the foundation for an EBV spatial agent-based model by defining key cell processes, like infected B cells, CD4 and CD8 T cells, T follicular helper cells, dendritic cells, macrophages, and stromal support cells. I also connected these cells through diffusible cytokine and chemokine fields, pairwise interaction tests, validation steps, and simulation outputs. The goal was to adapt the existing influenza modeling workflow to a new viral system, enabling EBV-specific immune interactions to be studied in a spatial lymphoid tissue environment.

Here are a few examples of results from a 6-hour simulation, which I will briefly show and explain.



EBV MVP simulation time-series outputs. Time-series plots summarize the major dynamics from the EBV spatial simulations, including viral diffusion, changes in cytokine/chemokine fields, immune-cell interactions, and agent-state transitions over time. Together, the three plots show how the MVP framework tracks EBV spread and multicellular immune signaling across simulation runs.

The EBV MVP simulation shows that the model can integrate viral diffusion, immune-cell behavior, stromal-cell support, and cytokine/chemokine signaling within a single spatial lymphoid tissue environment. The results indicate that EBV virions spread through the simulated tissue while immune and stromal cells generate signaling fields and interaction events. Overall, these outputs suggest that the framework serves as a foundation for studying how EBV spreads, how immune surveillance and tissue organization may interact in lymphoid tissue, and how these interactions may vary across lymphoid tissues.

Although there has been good progress, there is still a lot to go. For example, these are MVP models, not complete models; cells that haven't been included yet could make a difference in the results, and many parameters are estimated, with only a few supported by concrete literature citations. Also, working on the HIV model has been slow. The EBV repo is much more advanced.

In conclusion, the HIV model is still a work in progress, and the EBV model is working and can produce results, but it is still a long way from being complete. This shows that the agent-based approach can be extended to diseases beyond influenza and opens a pathway to greater understanding of disease dynamics, which can help develop cures and treatments for many diseases.