# A Spatiotemporal Model of T-Cell Population Dynamics within a Lymph Node

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## ABSTRACT

Well-mixed models overlook how LN architecture shapes immunity. We built a 3D reaction–advection–diffusion model of activated and memory T cells on a realistic LN mesh. Seeding a small precursor pool in a follicle near the paracortex generates sharp local gradients while reproducing the expansion peak and contraction dynamics reported by De Boer et al. (2003).

**Key findings**
**Expansion peak:** Localized proliferation matches observed rise and fall of T-cell counts.
**Spatial insight:** Strong gradients between proliferation zones and periphery reveal hidden mechanisms.
**Systemic robustness:** Total counts remain insensitive to seed location; global kinetics dominate.

## INTRODUCTION

**Why LNs?** Sites of T-cell activation, proliferation, trafficking, death, and memory — spatial context shapes immunity. (**Fig. 1**)

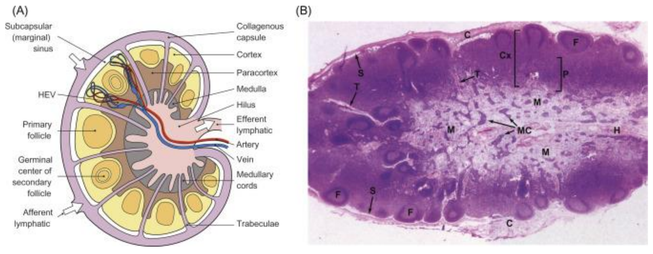


**Fig. 1: Labeled Human Lymph Node Structure**

**What's missing?** ODEs lose spatial detail; imaging is local/short-term → need whole-node, time-evolving spatial model.

**Our refinement:**
3D reaction–advection–diffusion model on LN mesh, calibrated to expansion–contraction–memory kinetics.

**Key insight:**
Follicle "hot zones" drive steep local gradients while systemic totals remain robust.

## METHODOLOGY

**Framework:** 3D spatiotemporal PDE model of T-cell dynamics in LN geometry

**Activated cells:**

$$\frac{\partial A}{\partial t} = \nabla \cdot (D_A \nabla A - \vec{v} A) + \rho(t) A - \delta_A(t) A - rA$$

Diffusion($D_A \nabla A$), directed flux($\vec{v} A$), proliferation ($\rho$), death ($\delta_A$), and differentiation ($r$).

**Memory cells:**

$$\frac{\partial M}{\partial t} = \nabla \cdot (D_M \nabla M - \vec{v} M) + rA - \delta_M M$$

Diffusion($D_M \nabla M$), directed flux($\vec{v} M$), proliferation ($\rho$), death ($\delta_A$), and differentiation ($r$).

**Parameters:**

Table 1: Model Parameters

| Symbol | Meaning | Value (units) | Notes |
|---|---|---|---|
| $\rho$ | Proliferation rate (activated) | 1.236 /day | Drives clonal expansion |
| $\delta_A$ | Baseline death (activated) | 0.05 /day | Pre-contraction turnover |
| $\alpha$ | Contraction boost (activated) | 0.2 /day | Extra death post-peak |
| $\delta_M$ | Death rate (memory) | 0.0007 /day | Ensures long-lived memory |
| $r$ | Differentiation $A \to M$ | 0.0191 /day | Memory formation rate |
| $A_0$ | Initial activated cells | 24 | Seeded in one follicle |
| $T_{\text{peak}}$ | Time of expansion peak | 6.5 days | Matches canonical kinetics |
| $D_A$ | Diffusion (activated) | 0.1 | Limited spread from hot zone |
| $D_M$ | Diffusion (memory) | 1.09 | Broad dispersal in LN |
| $\vec{v}$ | Advection field | fixed | Mesh-derived, time-invariant |

## RESULTS

**Spatial gradients:** The model predicts extreme heterogeneity in T-cell density. Proliferation in the paracortex generates multi–order-of-magnitude differences compared to the LN periphery (**Fig. 2**).

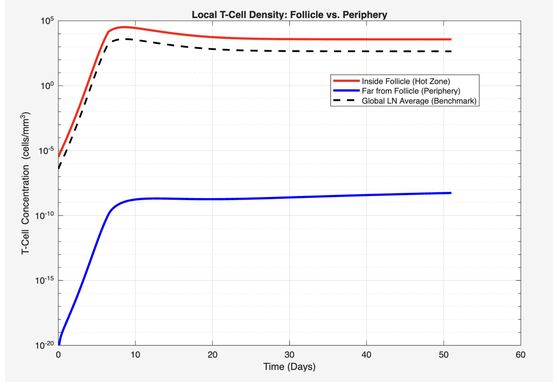


**Fig. 2: Local T-Cell Density: Follicle vs. Periphery**

**Systemic robustness:** Despite local heterogeneity, the total T-cell count over time is nearly identical across different paracortical seed locations (**Fig. 3**).

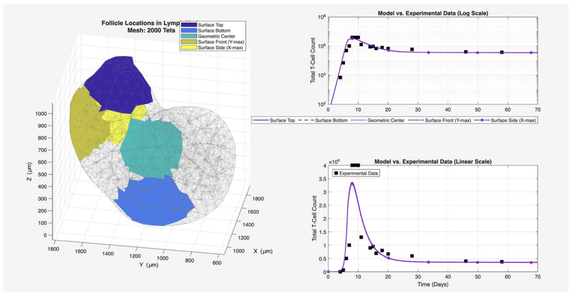


**Fig. 3: T-cell count over different paracortical seed locations**

**Comparison with non-spatial models:** The spatial PDE framework smooths systemic dynamics compared to ODE models, better matching physiological data (**Fig. 4**).

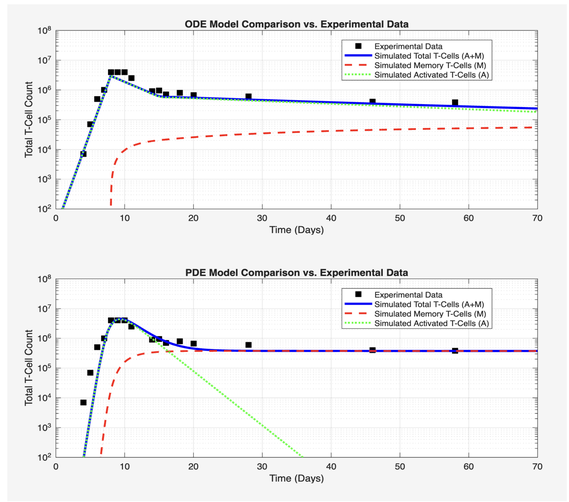


**Fig. 4: Spatial PDE model vs. Legacy ODE model**

## CONCLUSION

- **Spatial PDE model:** uncovers steep local T-cell gradients hidden in bulk data.
- **Systemic totals:** robust, largely independent of seeding.
- **Drivers:** proliferation, death, and transport dominate organ-level dynamics.
- **Framework:** quantitative link between LN microstructure and immune response.

## Discussions

**Local vs systemic:** Proliferation near paracortical follicles creates steep local gradients, even while systemic totals stay stable.

**Predictions:**
• Proliferation zones of T-cell density near follicles
• Seeding location shifts local patterns, not totals
• Transport processes shape contraction and dispersal

**Next steps:** Validate hot zones with intravital imaging or spatial transcriptomics, probe transport dynamics, and extend to other cell types.

**Takeaway:** LN architecture shapes immunity; our model provides a quantitative tool to uncover spatial dynamics **(Fig. 5).**

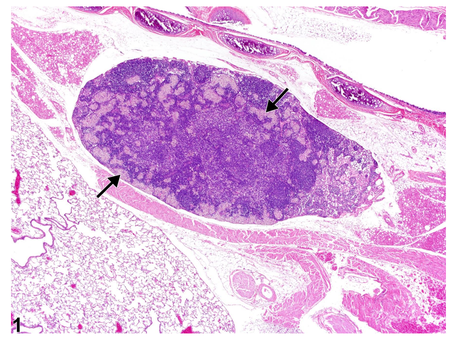

**Fig. 5: Mouse Lymph Node Structure**

## ACKNOWLEDGEMENTS & REFERENCE

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**Reference:**
De Boer, R.J., Homann, D., Perelson, A.S. *Different Dynamics of CD4⁺ and CD8⁺ T Cell Responses During and After Acute Lymphocytic Choriomeningitis Virus Infection. Journal of Immunology* 171(8): 3928–3935 (2003). doi:10.4049/jimmunol.171.8.3928