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Coagulation-fragmentation dynamics in macrophage populations

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Inflamed tissues are densely populated with macrophages that influence the balance between inflammation amplification and resolution. Macrophages that accumulate immunogenic substances such as cholesterol (in atherosclerosis) and uric acid (in gout) become pro-inflammatory and drive inflammatory responses that never resolve.

I use *in vitro* experiments to show that substances dynamically accumulate within macrophage populations via a coagulation-fragmentation process. This informs a coagulation-fragmentation equation that models macrophage accumulation of substances *in vitro*. This model generalises to a non-local PDE model of macrophage populations during tissue homeostasis and inflammation. Model analysis shows that immunogenic substances coalesce to extraordinarily large quantities inside macrophages during tissue inflammation but not homeostasis.

Atherosclerosis is considered as a specific example. The model is used to understand the dynamics of lipid accumulation inside macrophages that populate the inflamed artery wall. Our model can simply explain several complex hallmarks of atherosclerosis disease progression such as the formation of lipid-loaded macrophage foam cells and extracellular pools of necrotic debris.

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