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Parameter estimation for a model of peritonitis focusing on the sequential immune cell response

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Macrophages can be activated to a more inflammatory M1 phenotype or to an M2-like phenotype which promotes the resolution of inflammation. Problems with this phenotypic switch can result in a population imbalance that leads to chronic wounds or disease. We have developed a model for the sequential influx of immune cells in the peritoneal cavity in response to a bacterial stimulus that includes macrophage polarization. With this model we are able to reproduce the expected timing of sequential influx of immune cells and mediators in a general inflammatory setting. Weighted least-squares parameter estimates were obtained for this data (scaled) using trust region optimization in logarithmic parameter space. We then explore local structural and practical identifiability of the proposed model *a posteriori,* and obtain an identifiable subset of parameters for simulation of treatments.