The purpose of this project was to examine the influence of parenteral administration of interleukin-1, a cytokine with diverse biological activities, on antibacterial resistance in a laboratory rodent model. We first documented that intraperitoneal injection of minute quantities (0.1–1.0 ng per mouse) of interleukin-1 resulted in a rapid influx of inflammatory neutrophils. Neutrophil accumulation did not result from contamination of the interleukin-1 with bacterial lipopolysaccharide, nor was it abrogated by treatment with indomethacin, an inhibitor of prostaglandin synthesis. We also observed a small but significant increase in the number of inflammatory macrophages at later timepoints. We went on to show that prophylactic or concomitant administration of interleukin-1 (0.17 ug per mouse) significantly enhanced the resistance of recipient mice to a challenge infection with the facultative intracellular pathogen *Listeria monocytogenes*. Protection was not caused by contaminating bacterial lipopolysaccharide. Interleukin-1 mediated protection was associated with a rapid burst of serum colony-stimulating activity.
Current experiments are comparing the separate and combined effects of interleukin-1 and other cytokines on antibacterial resistance. We also will examine the influence of in vivo and in vitro administration of interleukin-1 on leukocyte function. These observations suggest that pretreatment with interleukin-1 might be beneficial for those at increased risk of bacterial infection. This work has resulted in the following publications:

