The existence of intracellular rickettsiae requires entry, survival, and replication in the eukaryotic host cells and exit to initiate new infection. While endothelial cells are the preferred target cells for most pathogenic rickettsiae, infection of monocytes/macrophages may also contribute to the establishment of rickettsial infection and resulting pathogenesis. We initiated studies to characterize macrophage-\textit{Rickettsia akari} and -\textit{Rickettsia typhi} interactions and to determine how rickettsiae survive within phagocytic cells. Flow cytometry, microscopic analysis, and LDH release demonstrated that \textit{R. akari} and \textit{R. typhi} caused negligible cytotoxicity in mouse peritoneal macrophages as well as in macrophage-like cell line, P388D1. Host cells responded to rickettsial infection with increased secretion of proinflammatory cytokines such as interleukin-1beta (IL-1beta) and IL-6. Furthermore, macrophage infection with \textit{R. akari} and \textit{R. typhi} resulted in differential synthesis and expression of IL-beta and IL-6, which may correlate with the existence of biological differences among these two closely related bacteria. In contrast, levels of gamma interferon (IFN-gamma), IL-10, and IL-12 in supernatants of infected P388D1 cells and mouse peritoneal macrophages did not change significantly during the course of infection and remained below the enzyme-linked immunosorbent assay cytokine detection limits. In addition, differential expression of cytokines was observed between \textit{R. akari}- and \textit{R. typhi}-infected macrophages, which may correlate with the biological differences among these closely related bacteria.