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Chlamydia pneumoniae inhibits activated human T lymphocyte proliferation by the induction of multiple apoptotic pathways

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Chlamydia pneumoniae is an omnipresent obligate intracellular bacterial pathogen that infects numerous host species. Infections of humans are a common cause of community acquired pneumonia but have also been linked to chronic diseases such as atherosclerosis, Alzheimer's disease, and asthma. Persistent infection and immune avoidance are believed to play important roles in the pathophysiology of *C. pneumoniae* disease. We found that *C. pneumoniae* organisms inhibited activated but not inactivated human T cell proliferation. Inhibition of proliferation was pathogen-specific, multiplicity of infection dependent that required chlamydial entry but not *de novo* protein synthesis. *C. pneumoniae* T cell inhibitory activity was resistant to ultraviolet irradiation but sensitive to heat implicating pathogen surface components or secreted molecules as inhibitory effectors. Activated $CD4^+$ and $CD8^+$ T cells were equally sensitive to *C. pneumoniae* anti-proliferative effectors. The *C. pneumoniae* anti-proliferative effect was linked to T cell death associated with caspase 1, 8, and 9 activation; indicating that multiple cellular death pathways were generated following pathogen T cell interactions. Collectively, these findings are consistent with the conclusion that *C. pneumoniae* could induce a local T cell immunosuppression and inflammatory response revealing a possible host-pathogen scenario that would support both persistence and inflammation.

Keywords: Persistent infection, Immunosuppression, Inflammation, Cellular death