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Host factors and genetic susceptibility for infections due to intracellular bacteria and fastidious organisms

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Abstract. While genetic polymorphisms play a paramount role in tuberculosis (TB), less is known about their contribution to the severity of diseases caused by other intracellular bacteria and fastidious microorganisms.

We searched electronic databases for observational studies reporting on host factors and genetic susceptibility for infections caused by intracellular fastidious bacteria published up to May 30, 2014.

Contribution of genetic polymorphisms was documented for TB. These include genetic defects in the mononuclear phagocyte/T helper cell type 1 (Th1) pathway contributing to disseminated TB disease in children and genome-wide linkage analysis (GWAS) in reactivated pulmonary TB in adults. Similarly, experimental studies supported the role of host genetic factors in clinical presentation of illnesses resulting from other fastidious intracellular bacteria. These include IL-6 -174 G /C or low mannose-binding (MBL) polymorphisms, which are incriminated in chronic pulmonary conditions triggered by *C. pneumonia*, type 2-like cytokine secretion polymorphisms, which are correlated to various clinical patterns of *M. pneumoniae* infections and genetic variation in the NOD2 gene, which are indicators of tubal pathology resulting from to *Chamydia trachomatis* infections.

Monocytes/macrophages migration and T lymphocytes recruitment defects are corroborated to ineffective granuloma formation observed among patients with chronic Q fever. Similar genetical polymorphisms have also been suggested for infections caused by *T. whipplei* although not confirmed yet.