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The chlamydiae are Gram negative obligate intracellular bacteria with a complex developmental cycle comprising a metabolically less active infectious stage the elementary body EB and a metabolically more active stage the reticulate body RB They are responsible for many acute and chronic diseases in humans and animals In order to play a causative role in chronic diseases chlamydiae would need to persist and to re activate within infected cells tissues for extended periods of time Persistence in vitro is defined as viable but non cultivable chlamydiae involving morphologically enlarged aberrant and nondividing RBs termed aberrant bodies AB In vitro alterations of the normal developmental cycle of chlamydiae can be induced by the addition of Interferon IFN tumor necrosis factor α TNF α and penicillin G exposure as well as amino acid or iron deprivation monocyte infection and co infection with viruses In vivo key questions include whether or not ABs occur in infected patients and animals and whether such ABs can contribute to prolonged chronic inflammation fibrosis and scarring through continuing stimulation of the host immune system known from diseases such as trachoma pelvic inflammatory disease reactive arthritis and atherosclerosis To date the direct causal role in the pathogenesis of chlamydial infection and persistence in vivo has been questioned since there was no tractable animal model of chlamydial persistence so far A very recent study was able to establish an experimental animal model of in vivo persistence when *C muridarum* vaginally infected mice were gavaged with amoxicillin Amoxicillin treatment induced *C muridarum* to enter the persistent state in vivo Recent in vivo data from patients indicate that viable but non infectious developmental stages are present in the genital tract of chronically infected women and that the gastrointestinal tract might be a reservoir for persistent chlamydial infections at other sites

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