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ORIGINAL ARTICLE

# *Chlamydia pneumoniae* and *Helicobacter pylori* IgG seropositivities are not predictors of osteoporosis-associated bone loss: a prospective cohort study

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**Abstract** The potential link between infection with *Chlamydia pneumoniae* or *Helicobacter pylori* and osteoporosis has not been investigated in population-based longitudinal studies. A total of 250 healthy postmenopausal women who participated in a prospective cohort study were evaluated for IgG antibodies directed against *C. pneumoniae* and *H. pylori*, osteoprotegerin (OPG), the receptor activator of nuclear factor kappa B ligand (RANKL), CrossLaps, and osteocalcin. Bone mineral density (BMD) was measured at the femoral neck and lumbar spine at baseline and at follow-up 5.8 years later. There were no significant differences in age-adjusted bone turnover markers, OPG, RANKL, the RANKL/OPG ratio, and BMD between the *C. pneumoniae* and *H. pylori* IgG seropositive and seronegative subjects ($P > 0.05$). Neither *C. pneumoniae* nor *H. pylori* IgG seropositivity was associated with age-and body mass index-adjusted BMD at the femoral neck and lumbar spine or bone loss at the 5.8-year follow-up. In logistic regression analysis, neither *C. pneumoniae* nor *H. pylori* IgG seropositivities predicted incident lumbar or spine osteoporosis 5.8 years later. In conclusion, neither *C. pneumoniae* nor *H. pylori* IgG seropositivity was associated with bone turnover markers, the RANKL/OPG ratio, BMD, or bone loss in postmenopausal women. In addition, chronic infection with *C. pneumoniae* or *H. pylori* did not predict incident osteoporosis among this group of women.

**Keywords** *Chlamydia pneumoniae* · *Helicobacter pylori* · Bone mineral density · Osteoporosis

## Introduction

*Chlamydia pneumoniae* is an obligate intracellular human respiratory pathogen that contributes to a wide spectrum of clinical presentations, including atherosclerosis [1]. This bacterium is able to survive in host cells and can affect chronic processes, such as atherosclerosis, through augmentation of the inflammatory system, signaling pathways, and oxidative stress [2]. Thus, it is plausible to consider a contributory role for *C. pneumoniae* infection in accelerated bone loss. Indeed, in vitro and in vivo studies have shown that infection with *C. pneumoniae* produces potentially inflammatory and bone resorptive cytokines and chemokines [3, 4]. Very recently, it has been reported that chlamydial DNA was found in osteoporotic bone tissue [5].

The association of *Helicobacter pylori* seropositivity with a variety of extradigestive manifestations, such as cardiovascular, immunological, and various other pathologies, has been suggested [6]. However, the link between *H. pylori* and these extradigestive manifestations is

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