

can reduce the BMD of the femoral neck (95% CI: 0.04~0.19,  $p = 0.002$ ), but cannot reduce the BMD of the lumbar spine (95% CI: -0.04~0.11,  $p = 0.346$ ).

**Conclusions.** Summarizing the articles and results analysis suggests that EH can have a negative effect on BMD, for different parts of bone, the degree of reduction is different. Furthermore, the reduction level of BMD can vary for different regions and populations.

**Key words:** association, essential hypertension, bone mineral density, meta-analysis

## 179. THE ROLE OF TRIGGER INFECTIONS IN REACTIVE ARTHRITIS

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**Introduction.** Reactive Arthritis(ReA) is an immune-mediated synovitis resulting from slow bacterial infections and showing intra-articular persistence of viable nonculturable bacteria and/or immunogenetic bacterial antigens synthesized by metabolically active bacteria residing in the joint and/or elsewhere in the body. Reactive arthritis is known to be triggered by a bacterial infection, particularly of the genitourinary (*Chl. trachomatis*, *Neisseria gonorrhoea*, *Mycoplasma hominis*, and *Ureaplasma urealyticum*) or GI tract (*Salmonella enteritidis*, *Shigella flexneri*, and *disenteriae*, *Yersinia enterocolitica*, *Campylobacter jejuni*, *Cl.difficile*). The incidence is about 2% to 4% after a urogenital infection mainly with *chlamydia trachomatis* and varies from 0% to 15% after gastrointestinal infections with *Salmonella*, *Shigella*, *Campylobacter*, or *Yersinia*.

**Aim of the study.** To identify the most common infections that lead to the reactive arthritis and to highlight the pathogenetic mechanisms of action, which would help to improve the treatment tactic.

**Materials and methods.** The relevant articles on the topic were taken from the databases NCBI, PubMed, Medline, and ScienceDirect .

**Results.** Reactive arthritis is an immune-mediated syndrome triggered by a recent infection. It is hypothesized that when the invasive bacteria reach the systemic circulation, T lymphocytes are induced by bacterial fragments such as lipopolysaccharide and nucleic acids. These activated cytotoxic-T cells then attack the synovium and other self-antigens through molecular mimicry. This is supported by the evidence of *Chlamydia trachomatis* and *C pneumoniae* ribosomal RNA transcripts, enteric bacterial DNA and bacterial degradation products in the synovial tissue and fluid. It is believed that anti-bacterial cytokine response is also impaired in reactive arthritis, resulting in the decreased elimination of the bacteria.

**Conclusions.** Current evidence supports the concept that reactive arthritis (ReA) is an immune-mediated synovitis resulting from slow bacterial infections and showing intra-articular persistence of viable, nonculturable bacteria and/or immunogenetic bacterial antigens synthesized by metabolically active bacteria residing in the joint and/or elsewhere in the body. The mechanisms that lead to the development of ReA are complex and basically involve an interaction between an arthritogenic agent and a predisposed host. The way in which a host accommodates to invasive facultative intracellular bacteria is the key to the development of ReA. The details of the molecular pathways that explain the articular and extra-articular manifestations of the disease are still under investigation.

**Key words:** bacterial infection, trigger, reactive arthritis