# **Pediatric Rheumatology**



Poster presentation

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# The influencing of environmental and genetics factors on bone metabolism in juvenile idiopathic arthritis children

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## **Background**

Bone mineralization losses depend on not only inflammatory aggression and complication of arthritis therapy or presence of whole numbers of genetic factors, influencing on bone metabolism, inflammation, immune system's functions and the therapy effectiveness.

The aim of our study was to research bone metabolism status depending on molecular markers and inflammatory activity in JIA children.

# Materials and methods

We included 184 JIA children, 77 boys and 112 girls. Bone mineralization was detected by dual-energy X-ray absorptiometry of lumbar spine  $L_1$ – $L_4$ . Bone biochemical markers were osteocalcine, C-terminal telopeptides, parathyroid hormone (PTH), Ca, Ca<sup>++</sup>, P, total alkaline phosphatase (TAP) activity. We've detected *ApaI-*, *TagI-*, *BsmI*-restriction length polymorphism assay of vitamin D (*VDR*) receptor gene, *Hind III* osteocalcine gene, *Sp I* type I collagen I $\alpha$  chain (*Coll \alpha I*), *BclI* glucocorticoid receptor gene (*GCR*).

#### Results

Low bone mineral density (LBMD) for age was detected then Zscore < -2 SD in 36 children, 18 girls and 18 boys. Girls with LBMD had lower height and weight, earlier age of arthritis onset and higher clinical and paraclinical arthritis activity parameters, higher osteocalcine and lower PTH. Children, who received glucocorticoids had lower BMD-Zscore, Ca in boys and lower BMC, BMD, BMD-Zscore, Ca, P, TAP activity in girls. Boys with normal BMD had frequently GG genotype *Bcl I GCR*. We have revealed positive correlation A allele *ApaI VDR* and negative correlation B allele BsmI *VDR* with BMD.

### **Conclusion**

We have detected opposite changes of bone mineralization between boys and girls, due to large numbers of environmental and genetics factors.