

Introduction

- Rheumatoid arthritis (RA) is a chronic autoimmune disease that is characterized by local and systemic bone destruction.
- Dysfunction in mineral homeostasis can contribute to systemic osteoporosis.
- Parathyroid hormone (PTH) regulates plasma calcium levels
- Fibroblast growth factor 23 (FGF-23) was recently implicated in regulation of phosphate levels.

Aim of study

To assess the state of mineral homeostasis in RA patients.

Methods

Serum and plasma samples were collected from RA patients and self-reported healthy donors. Levels of creatinine, calcium, phosphate, and parathyroid hormone (PTH) were assessed using standard blood biochemistry. In 91 plasma samples from RA patients and 31 samples from control individuals, the expression of FGF-23 was assessed using human FGF-23 ELISA.

Demographics

	RAs	Controls
Gender (F/M)	61/30	7/6
Age	61 ± 10 years	65 ± 7 years

Results

Figure 1. Plasma levels of calcium and phosphate were significantly increased in RA patients

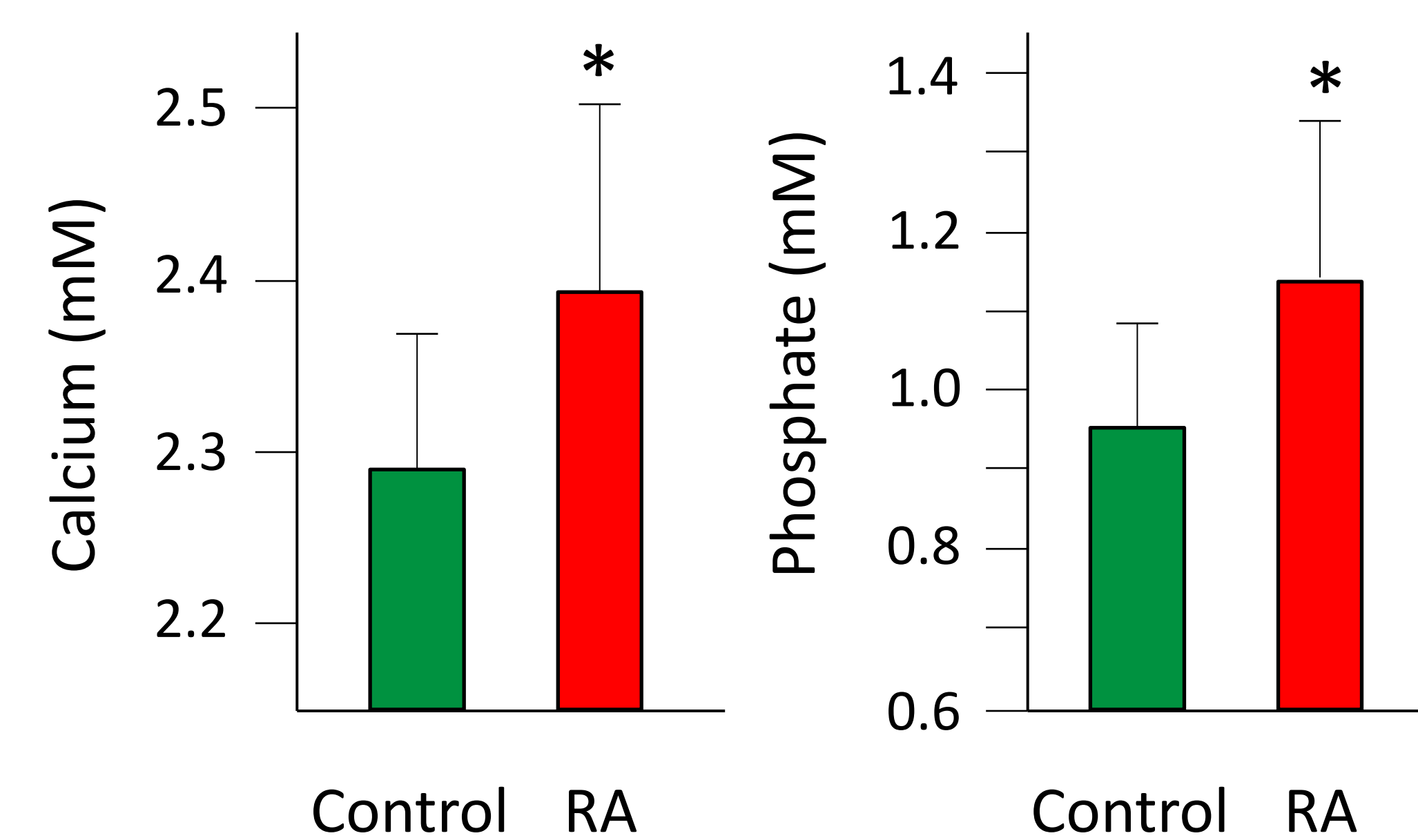


Figure 2. Plasma Creatinine levels were significantly increased in RA patients

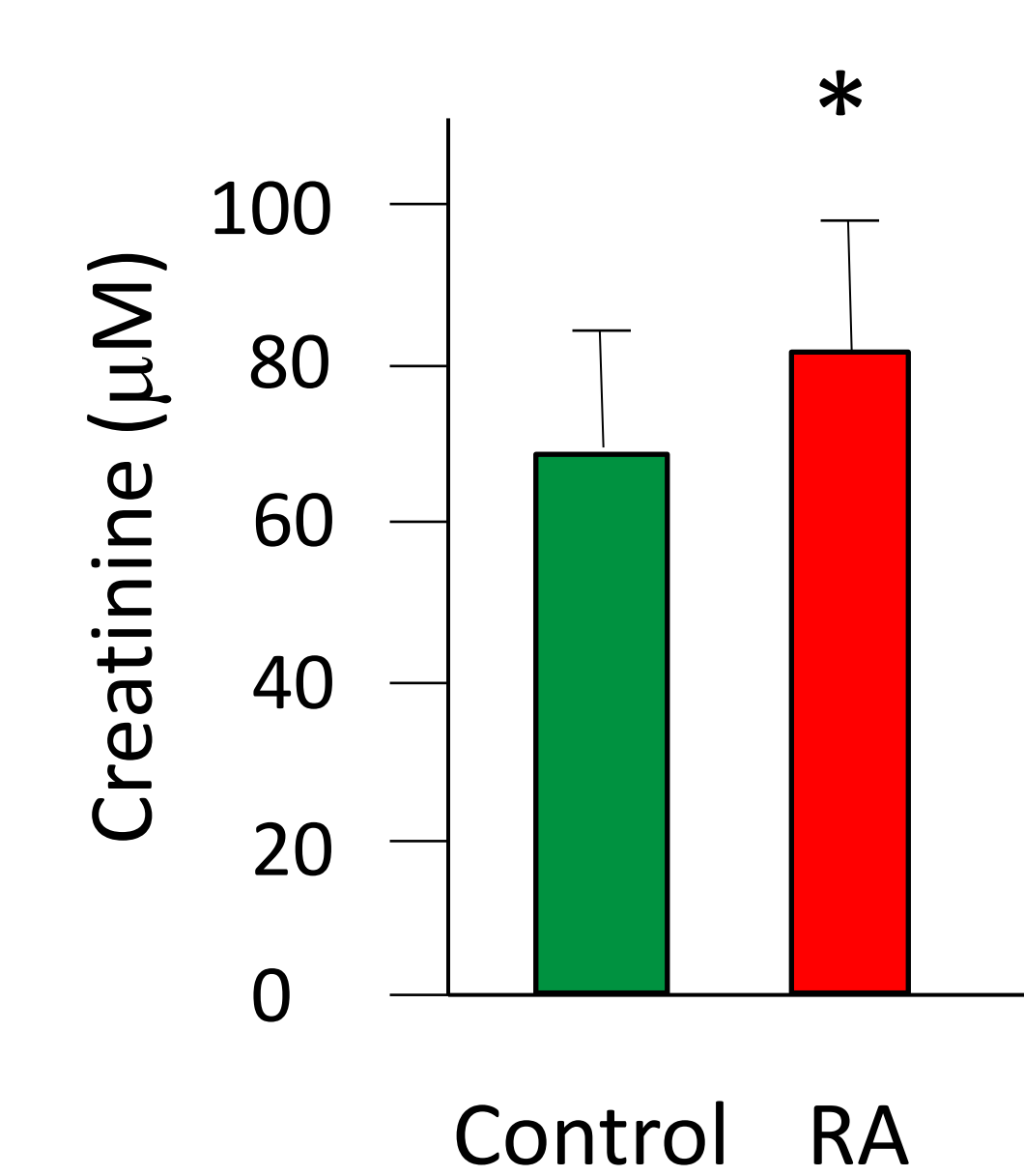


Figure 3. PTH levels were significantly increased in RA patients

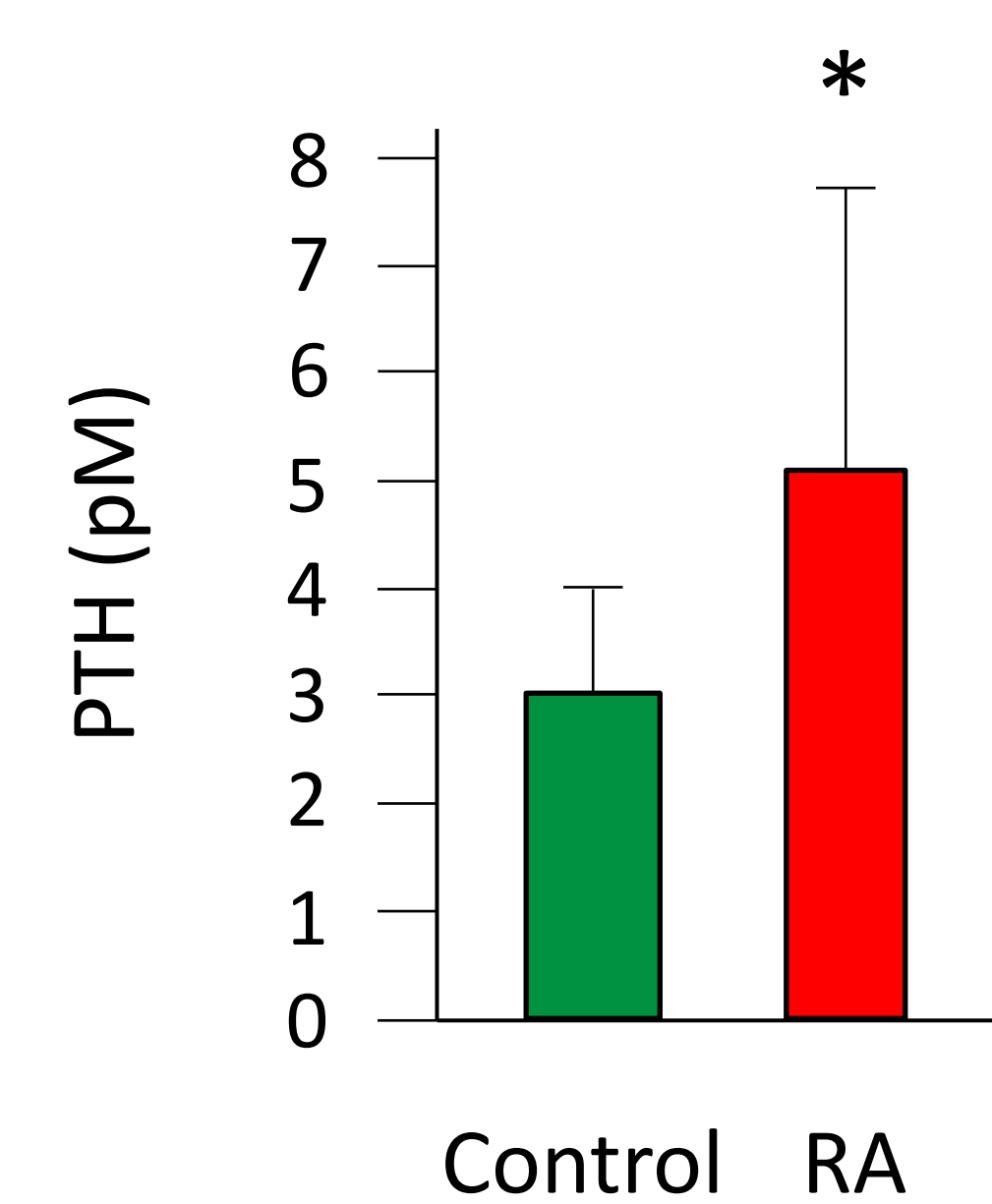
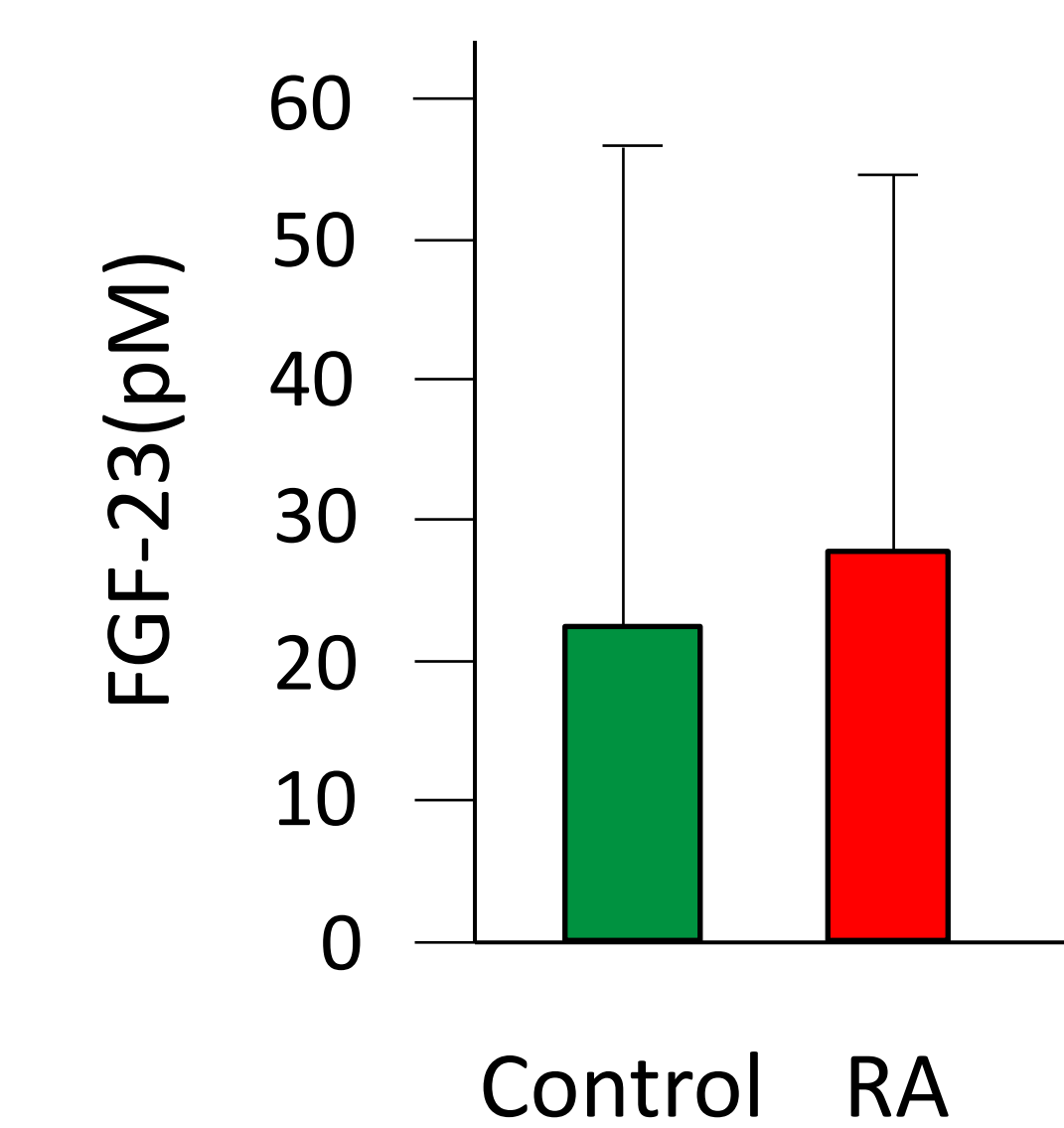


Figure 4. FGF-23 levels were similar in control group and RA patients



Summary and conclusions

- RA patients exhibited significant increase in plasma calcium levels
- RA patients exhibited significant increase in plasma phosphate levels
- Creatinine levels were significantly increased in RA patients, indicating mild kidney dysfunction
- RA patients exhibited significantly higher levels of PTH, but no significant differences in FGF-23 levels
- Thus, RA patients demonstrate significant imbalance in mineral homeostasis, in particular high levels of PTH, which may contribute to generalized bone loss in RA patients.

IODA - In vitro Osteoclast Differentiation in Arthritis

The work is a part of a large, multi-institution, and multi-year project called IODA. Our long-term goals are:

- ✦ establish osteoclastic parameters strongly related to presence and severity of bone and joint destruction
- ✦ determine whether these biomarkers could be used as predictors of patient progression and responsiveness to different treatment regimens
- ✦ identify new targets for development of antiresorptive therapies specifically aimed at arresting inflammation-induced bone loss.

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