**Objectives:** We wished to assess volumetric bone mineral density (BMD) by forearm QCT in conjunction with dual-energy X-ray absorptiometry (DXA) and bone biomarkers in RA and AS.

**Methods:** Forty RA and AS patients treated with etanercept (ETN) or certolizumab pegol (CZP) were included in a 12-month follow-up study. Peripheral QCT and DXA BMD were determined. Bone biomarkers, such as PTH, osteocalcin, RANKL, 25-hydroxyvitamin D (VITD), P1NP, CTX, sclerostin, DKK-1 and cathepsin K (CATHK) were assessed by ELISA.

**Results:** There was no further bone loss during anti-TNF treatment. Volumetric and areal BMD showed significant correlations with each other (p<0.05). Total QCT BMD after 12 months was inversely determined by disease activity at baseline in the full cohort (p=0.030). Cortical BMD was negatively determined by baseline disease activity (p=0.005) and CATHK (p=0.025). In RA, VITD-0 determined QTTRABMD-12 (p=0.005). In the full cohort, the one-year change in QTTRABMD was related to TNF inhibition together with VITD-0 (p=0.031). Therapy and lower CATHK determined OCORTBMD changes (p=0.006). In RA, treatment together with VITD-0 (p<0.01) or CATHK-0 (p=0.002), while in AS, treatment together with RANKL-0 (p=0.05) determined QT BMD changes.

**Conclusion:** QCT confirmed that biologics may attenuate bone loss. Disease activity, CATHK, RANKL and VITD may predict the effects of anti-TNF treatment on volumetric BMD changes. There may be differences between RA and AS in this respect.

**Acknowledgements:** This research was supported by Hungarian National Scientific Research Fund (OTKA) grant No. K 105073 (H.P.B. and Z.S.); by the European Union and the State of Hungary and co-financed by the European Social Fund in the framework of TAMOP-4.2.4-A/2-11/1-2012-0001 ‘National Excellence Program’ (Z.S.); by the European Union grant G1iT0-2.3.2-15-2016-00050 (Z.S.); and by the Pfizer Investigator Initiated Research Grants no. WS1955414 and WS1696450 (Z.S.).

**Disclosure of Interests:** Balázs Juhász: None declared, Katalin Gulyás: None declared, Agnes Horváth: None declared, Edit Végh: None declared, Anita Puszta: None declared, Agnes Szentpetery: None declared, Zsófia Pethő: None declared, Nóra Bodnár: None declared, Attila Harman: None declared, Levente Bodoki: None declared, Harjit Pal Bhattoo: None declared, Eva Szekanecz: None declared, Katalin Hodosi: None declared, Andrea Domjan: None declared, Szilvia Szamosi Speakers bureau: Roche, Csaba Horváth: None declared, Sándor Szántó Speakers bureau: Abbvie, MSD, Novartis, Consultant of: Abbvie, Novartis, Gabriella Szücs Speakers bureau: Roche, Boehringer, Acteon, Sager, Consultant of: Acteon, Boehringer, Hennie Raterman: None declared, Willem Lems Speakers bureau: Pfizer, Amgen, Lilly, UCB, Galapagos, Consultant of: Pfizer, Amgen, Lilly, UCB, Lilly, Novartis, Pfizer, Zoltán Szekanecz Speakers bureau: Pfizer, Roche, Abbvie, Novartis, Lilly, Sanofi, Consultant of: Pfizer, Abbvie, Novartis, Grant/research support from: Pfizer, UCb.

**DOI:** 10.1136/annrheumdis-2021-eular.19519

---

**T315 SUPPRESSES OSTEOGENIC DIFFERENTIATION IN SAOS-2 CELLS BY INHIBITING PHOSPHORYLATION OF AKT**

**Z. Huang1, X. Huang1, Y. Huang1, Z. Li1, Q. Huang1, T. Li1.** Guangdong Second Provincial General Hospital, Department of Rheumatology and Immunology, Guangzhou, China.

**Background:** New bone formation is common in the late stage of various inflammatory arthritis, while osteoblasts play a vital role in this process. Activation of PI3K/Akt pathway promotes the differentiation and enhances the function of osteoblasts [1]. T315 is a novel small molecule drug, which may induce apoptosis and suppress the expression of cellular markers of chronic lymphocytic leukemia cells by disrupting PI3K/Akt pathway [2]. However, the lack of study focuses on the influence of T315 on the other cells, except tumor cell lines.

**Objectives:** We aimed to assess the effect of T315 on human osteoblast-like Saos-2 cells, while its potential mechanism in PI3K/Akt pathway was evaluated as well.

**Methods:** (1) Saos-2 was stimulated with an osteogenic reagent which contained L-ascorbic acid, β-glycerophosphoric acid, and dexamethasone. The concentration of T315 was adjusted to 0μg/ml, 1μg/ml, and 2μg/ml in the culture medium. (2) Alizarin red stain and alkaline phosphatase (ALP) stain were performed at d0, d7, d14, and d21 after being treated with T315. (3) Cellular protein concentration of T315 was adjusted to 0μg/ml, 1μg/ml, and 2μg/ml in the culture. (4) Cellular protein concentration of T315 was adjusted to 0μg/ml, 1μg/ml, and 2μg/ml in the culture-glycerophosphoric acid, and dexamethasone. The concentration of ALP accordingly (Figure 1D). (5) T315 did not alter pPI3K, but it inhibited the phosphorylation of Akt (Figure 1F). (4) T315 did not alter pPI3K, but it inhibited the phosphorylation of Akt (Figure 1G). (H). (5) Runx2 was reduced because of the greater dose or longer incubation time with T315 (Figure 1).

**Conclusion:** T315 inhibits the differentiation of osteoblasts through inhibiting the phosphorylation of Akt. Surprisingly, pPI3K seldom changes in this process, so its detailed mechanism should be investigated in further.

**REFERENCES:**


---

**LEAST SIGNIFICANT CHANGE IN BONE DENSITOMETRY IN PATIENTS WITH OBESITY**

**D. Claire1, M. Geoffroy1, L. Kanagaratnam2, C. Isabelle1, A. Hittinger1, B. Loïs1, C. Clément1, J. H. Salmon1.** Centre Hospitalier Universitaire de Reims, Rheumatology, Reims, France; Centre Hospitalier Universitaire de Reims, Unité d’aide méthologique, Reims, France.

**Background:** Dual energy x-ray absorptiometry is the reference method to measure bone mineral density (1). Loss of bone mineral density is significant if it exceeds the least significant change. The threshold value used in general population is 0.03g/cm² (2). Patients with obesity are known for having a higher bone mineral density due to metabolism and physiopathology characteristics (3,4).

**Objectives:** The aim of our study was to determine the least significant change in bone densitometry in patients with obesity.

**Methods:** We conducted an interventional study in 120 patients with obesity who performed a bone densitometry. We measured twice the bone mineral density at the lumbar spine, the femoral neck and the total hip in the same time (5,6). We determined the least significant change in bone densitometry from each pair of measurements, using the Bland and Altman method. We also determined the least significant change in bone densitometry according to each stage of obesity.

**Results:** The least significant change in bone densitometry in patients with obesity is 0.049g/cm² at the lumbar spine, 0.069g/cm² at the femoral neck and 0.06g/cm² at the total hip.