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ANNOUNCEMENT

Journal of Bone and Mineral Metabolism Best Paper Award 2008

The *Journal of Bone and Mineral Metabolism* Best Paper Award was established in 2008. **Candidates for the award must be members of the Japanese Society for Bone and Mineral Research**, and the winner is honored at the Society's Annual Meeting.

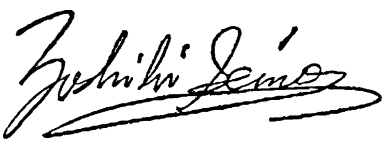
We are pleased to announce that the following article has received the first JBMM Best Paper Award.

"Imatinib mesylate inhibits osteoclastogenesis and joint destruction in rats with collagen-induced arthritis (CIA)"
by
Wataru Ando, Jun Hashimoto, Akihide Nampei, Hideki Tsuboi, Kosuke Tateishi, Takeshi Ono, Norimasa Nakamura, Takahiro Ochi, and Hideki Yoshikawa
J Bone Miner Metab (2006) 24:274–282


Dr. Wataru Ando

Abstract Macrophage colony-stimulating factor (M-CSF) is a key factor for osteoclastogenesis at the bone–pannus interface in patients with rheumatoid arthritis as well as a receptor activator of NF-κB ligand (RANKL). Imatinib mesylate inhibits the phosphorylation of c-fms, a receptor for M-CSF. The present study investigates the effect of imatinib mesylate on joint destruction in rats with collagen-induced arthritis (CIA) and on osteoclastogenesis in vitro. Imatinib mesylate (50 or 150 mg/kg), dexamethasone, or vehicle was administered daily to CIA rats for 4 weeks from the onset of arthritis. Hind-paw swelling and body weight were measured weekly. At weeks 2 and 4, the metatarsophalangeal (MTP) joints and the ankle and subtalar joints were radiographically and histologically assessed. The effect of imatinib mesylate on osteoclast formation from rat bone marrow cells with M-CSF and soluble RANKL (sRANKL) in vitro was also examined. Radiographic assessment showed that 150 mg/kg imatinib mesylate suppressed the destruction of the MTP and the ankle and subtalar joints at week 2, and MTP joint destruction at week 4 in CIA rats, although hind-paw swelling was not suppressed. The number of TRAP-positive cells at the bone–pannus interface was significantly reduced in the group administered with 150 mg/kg imatinib mesylate when compared to the group given vehicle at week 4. Imatinib mesylate dose-dependently inhibited the proliferation of M-CSF-dependent osteoclast precursor cells in vitro as well as osteoclast formation induced by M-CSF and sRANKL. These findings suggest that imatinib mesylate could prevent joint destruction in patients with rheumatoid arthritis.

We offer our sincere congratulations on behalf of the *Journal of Bone and Mineral Metabolism,* with best wishes for further development of the authors' research.



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