Overall, biochemical findings suggest a suppressed bone turnover and the increased BMD indicates that bone formation and resorption are affected unequally. We suggest that imatinib uncouples bone formation from bone resorption in favor of the former, disturbing bone homeostasis and leading to a net increase in bone mineral density.

This is further supported by a recent study by Fitteri *et al.*,[11] who reported an increased trabecular bone volume in imatinib treated patients. In contrast to our study, they did not analyze the bone mineralization, but measured the trabecular bone volume on decalcified iliac crest biopsies obtained before and during imatinib treatment. Therefore, bone mineralization, the most important measure of osteopenia, was not addressed.

The present report is the first demonstrating altered calcium and phosphate metabolism in imatinib-treated CML patients, concomitant with an increased cortical bone mineralization. Our study alleviates previous concerns about an accelerated osteomalacia, and measures to prevent this hypothesized long-term side effect seem unnecessary and might even be harmful.[2,12]

If imatinib is shown to suppress bone resorption without decreasing the bone quality, tyrosine kinase inhibitors could be novel antiosteolytic agents in skeletal disorders.

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Key words: imatinib, calcium, phosphate, chronic myelogenous leukemia, bone mineral density.

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Heterogeneous promoter activity of the telomerase reverse transcriptase gene in individual acute myeloid leukemia cells defined by lentiviral reporter assay

Acute myeloid leukemia (AML) generally constitutes a hierarch… prolif-
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ARTICLE RETRACTED

The article entitled "Heterogeneous promoter activity of the telomerase reverse transcriptase gene in individual acute myeloid leukemia cells defined by lentiviral reporter assay" by Seiichiro Kobayashi, Yasushi Soda, Yuansong Bai, and Arinobu Tojo, published ahead-of-print on May 27, 2008 as doi: 10.3324/haematol.12123, and on July 1, 2008 as Haematologica 2008; 93:1103-5, (1) has been retracted on June 27, 2008, by the corresponding author, Dr. Arinobu Tojo. In his email to the editorial office, Dr. Tojo stated that an investigation of the Institutional Review Board (IRB) records showed that the above study had not been approved by the IRB.

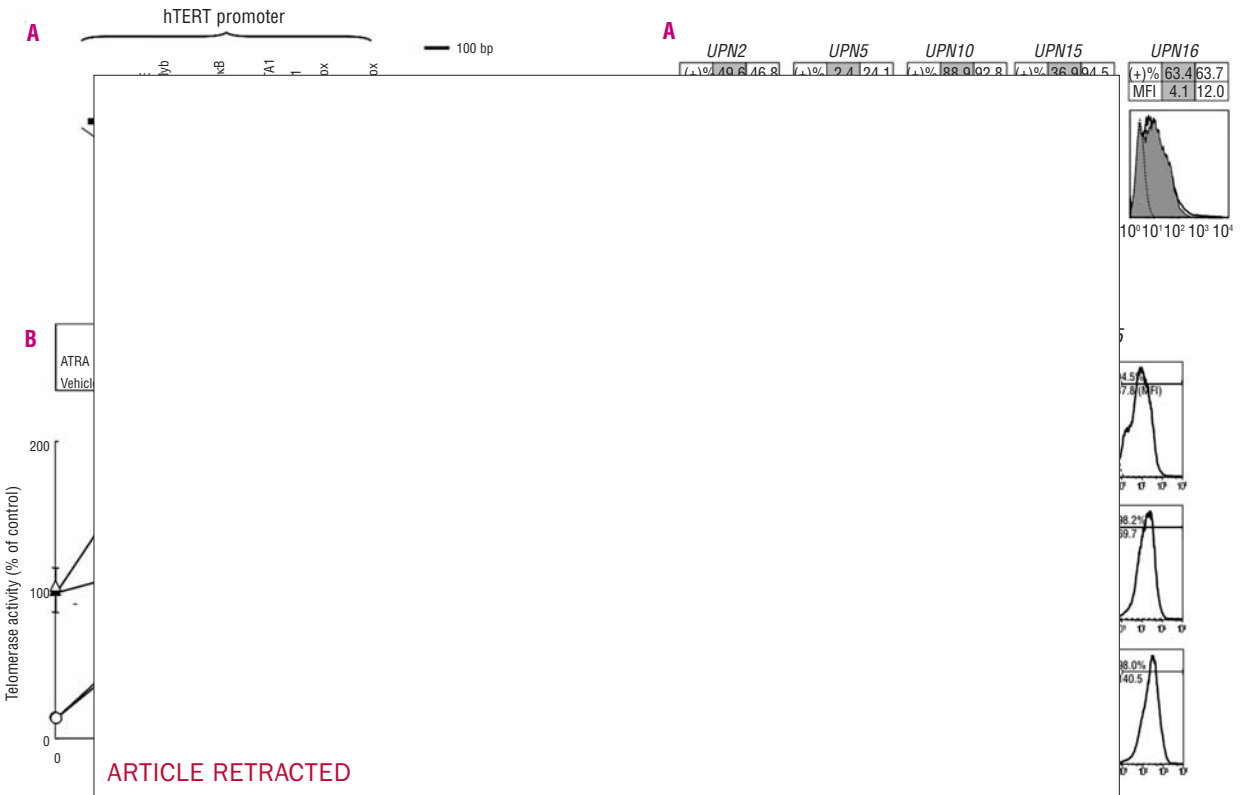

Figure 1. **(A)** The lent hTERT gen nal; SD, sp ment; R, re ed U3; cP sequence; regulatory moter. **(B)** the indicat ity (△) we results witl as the me with either out 1 μM A were deter are withou the mean ±

cute myeloid ams indicate respectively. ercentage of en box, CSF⁺; g/mL IL3, 10 s-transduced ed with CSFs, 2, 6, and 10. etermined by , the percent-

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Key words: AML, telomerase, hTERT promoter, lentiviral vector.

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Expression of the nuclear oncogene Ski in patients with acute myeloid leukemia treated with all-trans retinoic acid

For acute myeloid leukemia (AML), a two-hit model has been proposed for leukemogenesis:[1,2] the first mutation involving tyrosine kinase signaling such as a RAS mutation or activation of this pathway via Flt-3-mutation, the second aberration via truncated or fusion transcripts caused by chromosomal translocations, deletions or inversions.[3]

We have recently found that the nuclear oncogene Ski is overexpressed in AML, especially in AML with deletion of chromosome 7.[4] Ski represses all-trans retinoic acid (ATRA) signaling and myeloid differentiation in AML cells *in vitro*. Expression of a Ski mutant unable to interact with N-CoR (nuclear receptor corepressor) and thus unable to co-operate with histone deacetylases (HDAC)[5] did not inhibit ATRA signaling. *In vitro* treatment of cells with the HDAC inhibitor valproic acid abrogated the inhibitory effect of wild type Ski.[4]

ATRA has proved to be a highly efficient agent in the therapy of patients with acute promyelocytic leukemia (APL; FAB classification M3) characterized by the fusion protein PML-RARα, which results in impaired retinoic acid signaling.[6] Discrepant results have been published regarding the use of ATRA in AML other than APL. A study from the British Medical Research Council (MRC) did not find a significant difference in survival of AML patients except AML-M3 up to the age of 55 years treated with chemotherapy versus chemotherapy plus ATRA.[7] By contrast, a study of the German-Austrian AMLSG study group including 242 AML patients >60 years, excluding APL patients, revealed a significant