tested for IgH rearrangement, followed by t(4;14) and t(11;14), and for deletion 13q, 16q, 17p and hyperdiploidy using probes described in Chiecchio et al (2006). We carried out a univariate analysis by Fisher exact tests and corrected for multiple testing using label swapping permutation using the program PLINK. Genotyping was available on 754 patients from the Myeloma IX trial with FISH status for hyperdiploidy (355 cases), IgHsplit (306 cases); 13qdel (306 cases); p53del (61 cases); del16 (123 cases); t(4;14) (77 cases); t(11;14) (90 cases). In addition, we had 1456 UK control datasets from the WTCCC (Wellcome Trust Case Control Consortium Study) available to us across a subset of the BOAC panel (1100 SNPs), to examine etiological risk of karyotype subtypes. We found a number of significant associations with SNPs in metabolizing genes between karyotype subgroups, which might indicate an association of exposure to particular toxins with risk of developing a given karyotype. We will also report on SNP associations with innate immunity and DNA repair genes with karyotype subtypes.

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Protective Role of CYP1A1*2A in the Development of Multiple Myeloma

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Introduction and Aims: We had previously reported the association of the N QO1*2/*2 polymorphism with a decreased risk for multiple myeloma (MM) in Koreans (odds ratio [OR], 0.24; 95% CI, 0.01-0.68). **Materials and Methods:** The associations of polymorphisms of other metabolizing enzymes (CYP1A1 , GSTM1, and GSTT1) with the MM risk were investigated in 116 Korean MM patients and 176 Korean controls using TaqMan Allelic Discrimination and multiplex polymerase chain reaction. **Results:** The ORs for CYP1A1*1/ *2A and CYP1A1*1/ *2B genotypes were 0.43 (95% CI, 0.19-0.98) and 0.51 (95% CI, 0.26-0.98), respectively, which was significantly associated with a decreased MM risk. With regard to CYP1A1 alleles, the OR for the CYP1A1*2A allele was 0.57 (95% CI, 0.326-0.995), which was also significantly associated with a decreased MM risk. However, null types of GSTM1 and GSTT1 polymorphisms were not associated with the MM risk. These results were different from those of a previous report on white patients that suggested the association of the GSTT1 polymorphism with an increased MM risk and no association of CYP1A1 with the MM risk. **Conclusion:** The associations of polymorphisms of metabolizing enzymes with the risk for MM differed between Koreans and white patients, suggesting an ethnic variation in the susceptibility to MM.

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Defective Stem Cell Mobilization in OCIF-Knockout Mice

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Introduction and Aim: Osteoclast inhibitory factor (OCIF), that is identical to osteoprotegerin (OPG), has an important role on the suppression of osteoclast (OC) activation. The OCIF knockout (OCIF-KO) mice show enhanced OC activation in bone, which results in diffuse osteolytic or osteopenic changes resembling bone changes in myeloma. Therefore, OCIF-KO mice might become a bone disease model with persistent stimulation of OC. We analyzed whether lenograstim could induce sufficient blood stem cells in OCIF-KO mice, in order to elucidate the role of OC activation on blood stem cell mobilization. **Materials and Methods:** OCIF-KO C57BL/6 mice were purchased by Snow Brand Milk Company. Wild-type C57BL/6 mice were used as control. X-ray photographs of hind limb of mice were taken using Sofron SRO-iM50. Histologic findings of murine tibia were analyzed by the standard method after decalcification. Bone mineral density (BMD; mg/sq cm) was measured by dual-energy X-ray absorptiometry (DCS-600, Aloca). G-CSF (lenograstim 100 microgram/kg sc, 4days) was used as a mobilizer. Blood stem cells were measured using monoclonal antibodies reactive against murine CD34, lineage-specific antigens, and c-Kit. Cell numbers of blood, marrow, and spleen were counted using cell counter (Sysmex F-820). Stem cells after 4-days lenograstim were analyzed by flow cytometry (FACS Vintage, Becton-Dickinson). **Results:** 1) Radiographs of hind limbs of mice. OCIF-KO mice showed decreased calcified area. 2) Histology of mice. OCIF-KO mice showed marked decrease of trabecular bone. 3) BMD. OCIF-KO mice showed significantly decreased BMD. 4) Blood leukocyte count. No significant difference between OCIF-KO mice and wild type. 5) Blood stem cell (Lin-, c-Kit$^+$, CD34$^+$) count: In wild type, stem cell count (SC-C) showed significant increase after lenograstim (0.016% of WBC vs. 0.081%). In OCIF-KO mice, SC-C did not show significant increase after lenograstim (0.021% vs. 0.032%). **Conclusion:** Lenograstim-induced blood stem cell mobilization was defective in OCIF-KO mice. Further study must be done to clarify the mechanism of OC activation or osteopenia on the efficiency of lenograstim-induced blood stem cell mobilization.

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Genes MAGE-C1 and MAGE-A3 Are Central to the Survival and Chemotherapy-Resistance of Myeloma Cells

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Introduction: Expression of cancer-testis (CT) antigens is