P53 GENE MUTATIONS ARE RARE IN BRAZILIAN PATIENTS WITH MULTIPLE MYELOMA. Ortega MM\textsuperscript{1}, Duarte ASS\textsuperscript{1}, Melo MB\textsuperscript{2}, Lorand-Metze I\textsuperscript{1}, Costa FF\textsuperscript{1}, Lima CSP\textsuperscript{2} Department of Internal Medicine\textsuperscript{1}, Haematology and Haemotherapy Center \textsuperscript{2}, State University of Campinas, São Paulo, Brazil. manoela@obelix.unicamp.br

Multiple myeloma (MM) is a malignant clonal disorder characterized by infiltration of plasma cells in the bone marrow accompanied by osteolytic lesions, impaired hemopoiesis, hypogammaglobulinemia and renal disease. Molecular mechanisms underlying the development and evolution of the clonal expansion of plasma cells in MM are still unknown. A probable genetic alteration involves the p53 gene, a tumour suppressor gene situated in short arm of chromosome 17(17p13), which is a nuclear phosphoprotein implicated in the control of apoptosis and regulation of the cell cycle, by blocking transitions from G0/G1 phase to S phase in response to DNA damage. The role of the p53 gene mutations in the pathogenesis of MM and its potential use as a prognostic indicator remains uncertain. Therefore, we have used the technique of polymerase chain reaction single strand conformation polymorphism (PCR-SSCP) and sequencing to detect p53 mutations in exons 4-10. Genomic DNA was collected from bone marrow aspirates of 58 consecutive patients with MM (six were of stage IA, nine of stage IIA, three of stage IIB, twenty-one of stage IIIA, fourteen of stage IIIB and in five patients the staging was not performed) and one patient with plasma cell leukemia (31 male, 28 female; mean age\(\pm\)SD:57.8\(\pm\)11.6 years) from March 1999 to December 2000. Seven patients (11.86\%) presented a polymorphism in homozigosity in exon 4, at position 72 changing a guanine for a cytosine codifying an arginine (CGC) instead of a cysteine (CCC). A polymorphism in exon 6 at position 213 (CGA\(\rightarrow\)CGG) was identified in heterozigosity in one patient (1.69\%) and in homozigosity in other patient (1.69\%). One patient (1.69\%) showed a heterozigous mutation in intron 6 located 31 bp from the 3’ end of exon 6 of the p53 gene, changing an adenine for a guanine. We conclude that p53 mutation is a rare event in MM, not associated with clinical stage, and would seem to be of limited value as a prognostic indicator. Supported by FAPESP