product was expressed in the tumor at high levels in all three cases. The other 5
deletions generated a frameshift, which was predicted to result in the production
of a truncated protein product. In the case of the deletions, a 2-5 base pair repeat
was present close to the detected deletion, while the insertion duplicated the
sequence immediately upstream of the insertion site. Overall our findings indicate
that small intragenic p53 deletions/insertions are not rare events in ovarian
cancer, and that p53 exon 6 is the target in the vast majority (86%) of the cases.

#1815 The exon 5 of the p53 tumor suppressor gene is a target for
deletions in ovarian cancer. Angelopoulou, K., Katsaros, D., and Diamandis,
E.P. Mount Sinai Hospital, Toronto, Ontario, Canada M5G 1X5; University of
Toronto, Toronto, Ontario, Canada M5G 1L5; University of Turin, Turin, Italy 10131
Missense point mutations, leading to inactivation of p53 protein, are the most
frequent alterations in human cancer. Little however, is known about small
deletions or insertions occurring in this gene. We have analyzed 56 primary ovarian
tumors for the presence of such abnormalities. The analysis was based on
multiplex PCR amplification of exons 1 through 11, and fragment analysis of the
generated PCR products. Mutations were detected in 14% (8/56) of the tumors.
Deletions were much more prevalent than insertions (7 vs 1). Six of the deletions
and the insertion affected exon 5, while the other deletion was in exon 7. Two
Deletions and the insertion did not disrupt the reading frame and the protein