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 5 is the target in the vast majority (88%) 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 occuring 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