Background: Human Papillomavirus (HPV) natural history studies have revealed that human cancer is a rare consequence of an infec-
tion by some mucosa tropic high risk-HPV of this common sexually transmitted infection. HPV integration and persistent infection are critical events in progression to cervical carcinogenesis.

**Objectives:** Study of viral load and integration of HPV-16 in samples with different clinical/pathological status.

**Materials and Methods:** A total of 73 cervical samples infected with HPV-16 were retrospectively evaluated: 8 negative cytology, 4 ASC-H, 16 CIN1, 23 CIN2/3, 13 carcinoma and 9 treated carcinoma. DNA samples were extracted by a commercial kit (Qiagen). After spectrophotometrical quantification, the amplification was performed by in house PCR with set of primers: MY09/MY11 or PGMY09/PGMY11. β-globin was used as internal control. HPV types were determinated by RFLP, with Rsal and Ddel or by Microarrays (Papil-locheck). Viral load was performed by real time PCR, with primers from E6 and E2 region. Caski cells were used as a positive control and albumin was used as an internal control. HPV-16 integration was determined by the quantitative ratio between E2 and E6 gene.

**Results:** Episomal, mixed and integrated form of HPV-16 was detected in 25%, 22% and 53% of all samples, respectively. The highest value of HPV-16 viral load was observed in carcinomas (both mixed and integrated form), CIN1 and CIN2/3 (episomal form). In treated carcinoma, the highest viral load was observed predominantly in the integrated form.

**Conclusions:** The carcinomas have a higher viral load than CIN1 and CIN2/3, in accordance with some authors. These data show that integration is a very early stage of the neoplastic progression to carcinoma. This methodology may not be the best approach to the determination of the integration status (Kalantari et al, 2001).