tion was achieved less often in women than in men (p=0.004). In men, the expression of EGFR, VEGF, p53, P21, CerbB2 of NSCLC was 47.1%, 36.0%, 36.8%, 49.3%, and 30.1% respectively. In women, the expression of EGFR, VEGF, p53, P21, CerbB2 of NSCLC was 42.4%, 29.6%, 21.6%, 26.4%, and 48.8% respectively. Female gender significantly increased the risk of high expression of CerbB2, RR: 2.208, 95% CI: 1.330-3.667, p=0.002, and decreased the risk of high expression of P21, RR: 0.372, 95% CI: 0.159-0.870, p=0.023. The high expression of EGFR and VEGF may indicated the poor prognosis. The expression of p53 was significantly related to smoking.

Conclusions: Women with lung cancer were more peripheral type and smoked less intensively but had more passive smoking. Over-representation of adenocarcinoma and smaller lesions was observed in the women. Women with lung cancer had a better lung function and expression of CerbB2 in NSCLC related to women but expression of P21 has been verified associated with smoking correlated with men. Our study suggested the interaction between environmental and genetic factors is important.

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Frequent DNA copy number gains in 2p in lung cancer
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Methods: 105 lung cancers (43 AC, 35 SCC, 9 LCLC, 12 SCLC, and 6 others) were studied for chromosome 2 count using CEP-2 (alpha satellite) DNA probe for fluorescence in situ hybridization (FISH). 82 of these lung tumours were studied using FISH for DNA copy number changes in 2p16 and/or 2p21 with one to four BAC probes. Additionally, 28 of the lung tumours were microdissected and analysed by fragment analysis with 15 microsatellite markers in the 2p16-p21 region. Allelic imbalances were determined and compared with the copy number results.

Results: The average of the chromosome 2 count (CEP-2 signal) among the 105 lung tumours was 2.7 (range 1.7 to 5.0). CEP-2 mean signal count varied among the lung tumour types as follows: AC: 2.6; SCC: 2.8; LCLC: 3.0; SCLC: 2.4. The average DNA copy number among the 82 tumours studied was 2.7 also in 2p16 region, studied using three different BAC probes for FISH (n= from 28 to 69 for different probes). In contrast, in 2p21 the DNA sequences seemed to be gained with respect to the CEP-2 probe in lung tumours (n=39). The average copy number among all lung tumours was 4.0. The mean signal count at 2p21 between the lung tumour types did not differ significantly. The ratio between the 2p21 locus signals and CEP2 varied from 1.4 to 1.8, showing a low copy number gain in 2p21 in all lung tumour types. The frequency of allelic imbalance among informative cases was 57% in 2p21 and varied from 46% to 68% amongst different markers in 2p16. All lung cancer types showed some allelic imbalances in 2p.

Conclusions: Majority of lung tumours had three copies of chromosome 2 in tumour cells, possibly having triploid or near triploid genome. In 2p16 the DNA copy number was gained with respect to the centromere copy number. Our results showed that the region 2p21 may harbour DNA sequences important to the development of lung cancer. The results with fragment analysis and FISH were mainly similar but also displayed some differences. Uniparental disomy (UPD) may cause some of these discrepancies. Our study also showed the importance of using several different techniques in quantitative molecular genomic studies since recognition and interpretation of low copy number gains are difficult tasks in a complex genome such as in lung cancer.