3006
**Different morphological changes in non-small cell lung cancers and their corresponding lymph node metastases after neoadjuvant therapy?**
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**Aims:** Lymph node metastases are one of the most decisive adverse prognostic factors for patients with lung cancer. The assessment of therapy-induced tumor regression in pulmonary and mediastinal lymph nodes is therefore of special interest.
**Methods:** Morphological changes following neoadjuvant therapy are comparatively analyzed in 69 patients (19 female, 50 male; median age 51.8 years) with locally advanced NSCLCs (32 squamous cell carcinomas, 31 adenocarcinomas, 6 large cell carcinomas) using a standardized regression grading system.
**Results:** Concerning the quality of therapy-induced tumor regression the same morphological changes were established in the area of the primary tumor and in the corresponding lymph node metastases, with foci of necrotic tumor tissue, foam cell reaction, cholesterol clefts, giant cell formation, and scarry fibrosis. In contrast to this, markedly higher percentages were demonstrated for most of the different characteristics of therapy-induced tumor regression in the primary tumors as compared to their lymph node metastases, whereas comparable frequencies of "scarry fibrosis" were found in both.
**Conclusions:** These findings can be explained by a faster and therefore at the time of resection more advanced resorption of the therapy-induced tumor necroses in the lymph node metastases. This is due to the smaller tumor volume in metastatic lymph nodes with subsequent formation of smaller therapy-induced tumor necroses during neoadjuvant therapy.

3007
**Prognostic significance of S-100 immmunostaining in non-small cell lung cancer (NSCLC)**
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Altered S-100 protein expression was demonstrated in a variety of malignancies. The aim of this study was to assess prognostic value of this feature in NSCLC. Study group included 86 NSCLC patients who underwent pulmonary resection. S-100 protein expression was assessed immunohistochemically (DAKO ZO311) in paraffin embedded tumor tissue samples obtained during surgery. Any tumor cell staining was considered positive. S-100 expression was found in 32 cases (37%). There was no correlation between positive staining and clinical characteristics including age, sex, pathology type of tumor, staging and cigarette smoking. However, there was a tendency to simultaneous occurrence of S-100 expression and *P53* expression (p=0.06; Fisher exact test). At the time of analysis (September 2003), 13 patients were alive. Follow-up for the entire group was 8.7 years (range: 7.9-9.0). Median survival was 2.3 years, and 3 and 5 year survival probabilities were 47% and 31%, respectively. In a multivariate analysis (Cox model) S-100 protein expression in tumor cells was, apart from T and N features, the negative predictor of survival (p=0.016). In conclusion: S-100 protein expression in tumor cells negatively impacts prognosis in NSCLC patients.

3008
**Impact of chemotherapy on survival in 587 NSCLCs: a retrospective study in a Belgian regional hospital**
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In the last 5yrs. chemotherapy became common in treatment of non-small cell lung cancer (NSCLC). This study evaluates whether this change in treatment improves the survival rate.
798 (f86, m712) from 12826 patients who visited the pulmonary specialist in a regional hospital (1989-2003), were diagnosed with LC. After excluding SCLC (119), complex or multiple (24), inoperable LCs (24) or LCs indefinable by TNM (44), 587 (f61, m526) patients were studied for survival depending on therapy and stadia of LC (Survival Kaplan Meier, Cox Regression, SPSS v11.5).
Non-treated stadia IIIA,B+IV patients (NT) decreased from 130 of 226 before Oct97 to 82 of 229 afterwards. Non-chemotherapy (NC, mainly radio (R)) decreased from 76 (R=69) to 52 (R=39) after Oct97. Chemotherapy alone (CA) or combined with other therapies (C+, mainly R) increased from 8 and 12 (+R=11) to 44 and 51 (+R=46) respectively. IIIB+IV patients surviving for at least 1yr increased from 11.5% before Oct97 to 26.0% afterwards. Increasing median survival days (95%CI, Kaplan Meier) were observed over NT, CA, NC to C+ in IIIA from 93 (67,117), 108 (85,131), 274 (208,340) to 568 (372,764) and in IIIB+IV from 68 (51,85), 187 (143,231), 259 (181,337) to 327 (230,424). In an explorative purpose (multivariate Cox Regression), therapy was found to significantly influence survival time while age at diagnoses and sex didn't.
We conclude that after 1997, chemotherapy was used more frequently and in combination with other therapies improves significantly survival time. Radiotherapy remains very important, and, after 1997, is mainly combined with chemotherapy.

## 304. Pneumoconiosis: from asbestos to popcorn

3009
**The pneumoconioses: changing patterns in the United States, 1968-2000**
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**Background:** The National Institute for Occupational Safety and Health (NIOSH) maintains surveillance systems for assessing pneumoconiosis mortality and morbidity.
**Objective:** To describe trends in coal workers' pneumoconiosis (CWP) mortality and morbidity, and silicosis and asbestosis mortality, from 1968 to 2000.
**Methods:** Mortality data were drawn from national vital statistics records as either underlying or contributing cause of death using International Classification of Diseases (ICD 8, 9 and 10) codes for CWP, silicosis, and asbestosis. Rates were age-adjusted using the Year 2000 U.S. Standard Population. CWP morbidity data were derived from a national health monitoring program for underground coal miners, tabulated by tenure.
**Results:** For CWP, age-adjusted mortality rates decreased from 21.2 deaths/$10^6$ (2,870 deaths) in 1972 to 4.4 (950 deaths) in 2000. Concomitantly, the CWP prevalence in examined miners has also shown a steep decline over the same period. Silicosis mortality has also decreased, from 8.9 deaths/$10^6$ (1157 deaths) in 1968 to 0.7 (33 deaths) in 2000. In contrast, asbestosis mortality has increased from 0.5 deaths/$10^6$ (77 deaths) in 1968 to 6.9 (1,493 deaths) in 2000.
**Conclusions:** The overall burden of CWP and silicosis in the U.S. has decreased over the last 30 years, reflecting both fewer workers exposed and the efficacy of control measures. In contrast, asbestosis mortality has increased, largely a legacy of increased use of asbestos from 1940 through the 1970s.

3010
**Rate of decline of lung function in asbestosis**
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There are few studies of the rate at which lung function declines in subjects with asbestosis. In order to examine this question data was collected from all subjects attending the Dust Disease Board of NSW who had died in the previous 5 years and for whom lung function test results were available for more than 10 years prior to death. There were 89 subjects. All were males, had pleural plaques, a history of asbestos exposure and high resolution CT scan changes of asbestosis. In 39 subjects the death certificate stated that asbestosis had been the cause of death while in the other 50 subjects the cause of death was non-respiratory disease. Expressed as % of predicted normal, the mean (± 1 SD) test results for the 89 subjects at 10 years and at < 2 years prior to death were 82±15% and 78±24% for vital capacity, 75±20% and 71±14% for FEV1, 74±23% and 71±14% for DLco, plus 85±26% and 57±20% for Kco. In 53 subjects (59%) there was a fall in one or more of the test results between 10 and < 2 years prior to death. In those 50 subjects the mean DLco (as % of predicted normal) was 73±23% and 59±26% at 10 and < 2 years to death respectively, and the mean Kco was 84±26% and 56±19% ($p<0.05$). The changes in mean vital capacity (80±16% and 76±22%) and in FEV1 (73±18% and 68±14%) were small and non-significant ($p>0.05$). In the subjects in whom deterioration was detected the mean fall in DLco (compared to the initial value) was 27±7% and for Kco the fall was 36±17%. In these subjects with asbestosis, followed for more than 8 years prior to death, a deterioration in lung function occurred in 59% with the largest changes being those in DLco and Kco.

3011
**Diagnostic accuracy of BAL asbestos body concentration in assessing asbestos occupational exposure: a meta-analysis**
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The diagnosis of asbestosis relies on evidence of interstitial lung disease in patients with prolonged occupational exposure to asbestos. When occupational history is difficult to ascertain, mineralogical analysis may help to estimate exposure. A

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