肺癌预防、诊断和治疗最新进展汇编

上海市医学科学技术情报研究所

说　　明

1．《肺癌预防、诊断和治疗最新进展汇编》由上海市胸科医院主任医师、上海第二医科大学廖美琳教授主编，所收录的资料选自1991年以来世界各国著名3200种期刊，共计809篇，全部含原作者文摘。

2．收录的文献分五部分：

(1) EPIDEMIOLOGY

(2) ETIOLOGY

(3) IMMUNOLOGY, METABOLISM, PHYSIOPATHOLOGY

(4) DIAGNOSIS

(5) THERAPY

凡文献涉及两个类目者，为减少篇幅，都归属于一个类目，其他类目中不再重复出现，请读者在查阅时互相参照。

3．著录项目：

篇名（TI=Title）；　作者（AU=Author）；

出处（SO=Source）；　文摘（AB=Abstract）；

收藏单位（LC=Library Code）

LC（Library Code）为华东地区收藏单位的代号，读者如要该篇文献的全文，请查阅“收藏单位代号表”直接向收藏单位联系借阅或申请复印。“收藏单位代号表”附后。

此项工作是我们为了方便读者的一次尝试，难免会有差错，希望读者在使用过程中发现问题，及时指出，以利改进工作。

《肺癌预防、诊断和治疗最新进展汇编》编辑组

1992．9．

收藏单位代号表

SH(上海市)： (67)

0005	上海图书馆	0005E	上海科学技术情报研究所
0006	中科院上海图书馆	0082	复旦大学
0083	上海交通大学	0084	同济大学
0085	华东师范大学	0086	上海科学技术大学
1401	上海师范大学	1408	中科院东海研究站
1409	中科院上海药物研究所	1411	中科院上海有机所
1416	中科院上海生物工程实验基地	1417	中科院上海细胞生物所
1419	中科院上海生理所	1421	中科院上海生化所
1423	中科院上海植物生理所	1427	中科院上海昆虫所
1435	中科院上海原子核所	1449	中国预防医科院寄生虫病研究所
1450	中科院上海脑研究所	1540	上海医疗器械专科学校
1541A	上海医疗器械行业技术开发中心	1831	上海医科大学
1831A	上海医科大学中山医院	1832	上海医科大学儿科医院
1833	上海第二医科大学	1836	上海针灸经络研究所
1837	上海中医学院	1838	上海铁道医学院
1838B	上海医科大学妇产科医院	1839	中华医学会上海分会图书馆
1840	上海实验动物研究中心	1841	上海医药工业研究院
1842	上海市遵义医院	1843	上海生物制品研究所
1844	上海长征医院	1846	华东医院
1850	上海静安区中心医院	1855	上海市第一人民医院
1863	上海市胸科医院	1868	上海市精神卫生中心
1870	上海市血液中心	1871	上海市医学科学技术情报研究所
1875	上海市放射医学研究所	1876	上海市医药局情报所
1881	上海市卫生防疫站	1882	上海市药品检验所
1884	上海市中医文献馆	1885	国家医药管理局中成药情报中心站
1887	上海市精神卫生中心分部	1889	上海市儿童医院
1890	上海市劳动卫生职业病防治所	1890A	上海市劳动保护科研所
1891	上海市计划生育科学研究所	1892	上海医疗器械研究所
1893	上海医科大学肿瘤医院	1894	上海市化工职业病研究所
1898	上海市寄生虫病防治所	1899	中国福利会国际和平妇幼保健院
1900	上海动植物检疫所	1916	上海农学院
1917	上海农药研究所	1918	上海农科院畜牧兽医所
1919	上海农科所园艺所	1925	上海市农业科学院
3225	第二军医大学		

JS(江苏省)： (28)

0031	南京图书馆	0903	中国医科院皮研所
7960	江苏省医学情报研究所	7961	南京医学院
7967	中国药科大学	7968	江苏省肿瘤防治研究所
7968A	江苏省寄生虫病研究所	7968B	江苏省血吸虫病防治研究所
7970	镇江神经精神病院	7971	南京铁道医学院
7972	南京中医学院	7973	中华医学会苏州市分会
7973A	南京市脑科医院	7973B	无锡市临床医学实验研究所
7975	南京神经精神病院	7976	苏州市广济医院
7976A	苏州市第一人民医院	7977	扬州医学院
7978	镇江医学院	7979	中华医学会南京医学会

7983 南京药物研究所
7985B 徐州医学院附院
7987B 常州市第二人民医院
7988 江苏省原子医学研究所
7985 徐州医学院
7987A 常州市第一人民医院
7987C 江苏省医院管理学会
7989 淮阴市第三人民医院

JX(江西省): (7)
0022 江西省图书馆
5700 江西省科技情报研究所
5818 江西省寄生虫病研究所
5825A 江西省药物研究所
0925 江西省肿瘤医院
5815 江西医学院
5824 江西医学院第二附属医院

ZJ(浙江省): (11)
0032 浙江省图书馆
8202 浙江省环境保护研究所
8218A 温州医学院眼科研究室
8221 浙江省医学情报研究所
10342A浙江医科大学杭州分校
10344 浙江中医学院
0160 浙江省科技情报研究所
8217 浙江省精神病防治研究所
8219 浙江省医学科学院
10342 浙江医科大学
10343 温州医学院

AH(安徽省): (3)
0033 安徽省图书馆
8395 安徽省科技情报研究所
8409 安徽医科大学
8428 安徽省医学科学研究所
0073 中国科学技术大学
8408 蚌埠医学院
8424 皖南医学院
8427 安徽省医学情报研究所

SD(山东省): (21)
0034 山东省图书馆
8652 山东省科技情报所
8673 山东医科大学
8673E 山东医学科学院药物所
8675E 青岛医学院附属医院
8676A 山东省肿瘤防治研究所
8678 山东省寄生虫病防治研究所
8680B 青岛市立医院
8682 山东省医药工业研究所
8686 山东省中医学院附属医院
8689 山东计划生育科学技术研究所
8554 山东省医疗器械研究所
8671 山东省科技保护研究所
8673D 山东医科大学附属医院
8674 山东省中医药研究所
8676 山东省精神病医院
8677A 济宁市精神病防治院
8679 山东省医学情报研究所
8681B 济南市儿童医院
8685 山东省皮肤病防治研究所
8688 山东省省立医院

FJ(福建省): (13)
0035 福建省图书馆
8809 福建省微生物研究所
8873 福建省医学院
8886 南京军区福州总医院
8891 福建省劳动卫生职业病研究所
8895 福建省立医院
10392 福建省医学院
0155 福建省科技情报所
8871 福建省医学院附属协和医院
8877 福建省医学情报所
8887A 福建省中医学院
8893 福建省中医药研究所
8900 福建省皮肤病防治研究所

9999 各大医学图书馆基本都有

CONTENTS

1. EPIDEMIOLOGY

TI: Absence of evidence for a significant background incidence of diffuse malignant mesothelioma apart from asbestos exposure.
AU: Mark-EJ; Yokoi-T
SO: Ann-N-Y-Acad-Sci. 1991 Dec 31; 643: 196-204
AB: The incidence of diffuse malignant mesothelioma is rising. Physicians can diagnose a disease only when they know that it exists, so one explanation for the rise in incidence is more widespread appreciation of the clinical and pathological features of the disease. Modern pathology has ascribed specific requirements for the diagnosis of diffuse malignant mesothelioma. A priori one cannot assume that these requirements have increased the likelihood of the diagnosis because the requirements also can serve to exclude the diagnosis depending on the findings. Most cases of diffuse malignant mesothelioma are suspected by macroscopic and routine microscopic techniques that have been available since the last part of the nineteenth century. Although single reported instances of some pulmonary diseases have survived from the nineteenth century, pathologists did not identify enough cases to convincingly establish the existence of diffuse malignant mesothelioma of the pleura as an entity until the 1930s or 1940s. One must conclude that the background level of diffuse malignant mesothelioma in Europe and in the United States prior to 1930 was extremely low. No case was detected at the Massachusetts General Hospital until 1946.

TI: [Causes of mortality among workers exposed to inorganic dust containing crystalline silica]
AU: Starzynski-Z; Marek-K
SO: Med-Pr. 1991; 42(6): 467-75
AB: The authors briefly comment on the characteristics and results of recent epidemiological studies devoted to the reasons of mortality in persons exposed to dusts containing crystalline silica. The most important data concerning these studies are presented in the table accompanied by an author commentary which discusses results obtained by various authors and justification behind this subject in Poland.

TI: Pulmonary metastatic malignant tumours in Ibadan, Nigeria: 10 years autopsy review.
AU: Awotedu-AA; Odunfa-AO; Aghadiuno-PU; Ogunlesi-AO; Igbokwe-EO; Akinduro-OM
SO: Cent-Afr-J-Med. 1991 Sep; 37(9): 285-9
AB: This study reports the findings of a ten year review of autopsy records at the University College Hospital, Ibadan, Nigeria of patients who died of malignant tumour metastases to the lungs and pleurae. During the study period 1977 to 1986, a total of 3,549 autopsies were performed out of which 339 cases 10.5 pc died of malignant tumours. One hundred and thirteen of these tumours (33.3 pc) metastasised to the lungs and pleurae among other sites. Further analysis of these 113 patients showed that 49 were male and 64 females giving a male:female ratio of 1:1,3. In addition, the ages of the patients ranged between 9 months and 90 years with a mean of 38.3 years. The uterus was the commonest organ from which pulmonary metastases occurred (28.3 pc), with choriocarcinoma being the predominant historical type of uterine tumour. The liver was the next most common organ 26.5 pc with male to female ration of 3:3,1. Twenty other organs were also identified, the breast, kidney and oesophagus 7 pc each; pancreas ovary and thyroid 3 pc each being the most important. Other organs are adrenals, foot, neck, cervix and rectum--2 pc each.

TI: New potential chemopreventive agents for lung carcinogenesis of tobacco-specific nitrosamine.
AU: Chung-FL; Morse-MA; Eklind-KI
SO: Cancer-Res. 1992 May 1; 52(9 Suppl): 2719s-2722s
AB: Cigarette smoking is the major cause of lung cancer in humans. The continuous increase in the prevalence of cigarette smoking worldwide demands a practical means to circumvent this serious health problem. Our research has focused on the development of new chemopreventive agents against lung carcinogenicity of the tobacco-specific nitrosamine 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone. Several aromatic isothiocyanates have been identified as effective inhibitors of 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone-induced lung tumorigenesis.

Phenethyl isothiocyanate, a natural constituent of cruciferous vegetables, protects F344 rats and A/J mice from 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone-induced lung tumorigenesis. The alkyl chain length in the aromatic isothiocyanates is an important structural feature for the inhibitory potency. The inhibitory efficacy increases as the alkyl chain elongates up to 6 carbon atoms. Thus, 6-phenylhexyl isothiocyanate is approximately 50 to 100 times more potent than phenethyl isothiocyanate. The remarkable efficiency of 6-phenylhexyl isothiocyanate suggests its potential as a chemopreventive agent in intervention trials. The tissue distribution and excretion of phenethyl isothiocyanate were studied in mice. Two major urinary metabolites were identified as the mercaptopyruvic acid and the N-acetylcysteine conjugates. A urinary marker was developed to quantitate the uptake of phenethyl isothiocyanate in humans after consumption of watercress, a cruciferous vegetable rich in gluconasturtiin, the glucosinolate precursor of phenethyl isothiocyanate. Considering the anticarcinogenic activity of phenethyl isothiocyanate, this marker may eventually be useful in assessing the role of dietary phenethyl isothiocyanate uptake in lung cancer risk.

TI: Determination of biomarkers for intermediate end points in chemoprevention trials.
AU: Lee-JS; Lippman-SM; Hong-WK; Ro-JY; Kim-SY; Lotan-R; Hittelman-WN
SO: Cancer-Res. 1992 May 1; 52(9 Suppl): 2707s-2710s
AB: Renewed interest is being directed toward chemoprevention as a means of reducing cancer mortality. To overcome the inherent problems associated with using cancer development as a study end point, there has recently been a great surge of interest in defining the biomarkers associated with specific stages of the carcinogenic process as intermediate end points. We have detailed the evidence supporting the concept of field cancerization, a concept of general importance that is probably applicable to carcinogenesis and chemoprevention at many organ sites in humans, and presented results of tests of the potentially useful biomarkers proliferating cell nuclear antigen and blood group antigen. Because microassay techniques are more readily applicable to small biopsy samples, further expansion of these studies and exploration of panels of additional biomarkers are expected to generate exciting results in the field of chemoprevention.

TI: Lung cancer detection and prevention: evidence for an interaction between smoking and genetic predisposition.
AU: Sellers-TA; Potter-JD; Bailey-Wilson-JE; Rich-SS; Rothschild-H; Elston-RC
SO: Cancer-Res. 1992 May 1; 52(9 Suppl): 2694s-2697s
AB: The initiation and promotion of cancer is thought to result from a series of genetic mutations, some of which may be inherited. Our analysis of 337 lung cancer families suggested that, after allowing for an individual's pack-years of tobacco use, the pattern of disease was best explained by Mendelian codominant inheritance of an allele that produced earlier age of onset. Since lung cancer rarely occurs in the absence of exposure to tobacco, differences in the prevalence of smoking across generations could have a profound influence on the fit of genetic models. In the present study, families were partitioned into two groups, based on the birth cohort of the proband, i.e., born before World War I (age at death, greater than or equal to 60 years) or born after World War I (age at death, less than 60 years). This partition was chosen because the year 1915 signaled the start of the dramatic rise in tobacco use in the United States. In younger proband families, in which parents were more likely to smoke, Mendelian codominant inheritance provided the best fit to the data. In older proband families, for whom smoking among parents was less prevalent, the "no major gene" and "environmental" hypotheses were rejected; however, no Mendelian models could be distinguished. If the results on the families with the most homogeneous exposure to tobacco across generations (born after World War I) reflect the true underlying biology, then the influence of genetic factors in the pathogenesis of lung has been underestimated; the cumulative probability of lung cancer at age 80 for a noncarrier of the gene, at the average level of tobacco consumption, is close to zero, implying that virtually all lung cancer occurs among gene carriers. Identification of this putative genetic factor has profound implications for the detection and prevention of lung cancer.

TI: Application of molecular genetics to the early diagnosis and screening of lung cancer.
AU: Birrer-MJ; Brown-PH
SO: Cancer-Res. 1992 May 1; 52(9 Suppl): 2658s-2664s

AB: Recent studies of the molecular biology of lung cancer have identified multiple abnormalities. Despite this vast cataloging of genetic lesions, the chronology of these events and those which occur early remains essentially unknown. This review summarizes the genetic abnormalities in lung cancer cells, including mutation, amplification, and overexpression of dominant protooncogenes as well as deletion and mutation of recessive oncogenes. In addition, possible candidate genes exist which may participate in the early activation events of lung cancer, and evidence for their role in the early development of cancer is discussed. These lesions may be helpful in developing strategies to screen for lung cancer.

TI: Methods and strategies in lung cancer control.
AU: Castonguay-A
SO: Cancer-Res. 1992 May 1; 52(9 Suppl): 2641s-2651s
AB: By the Year 2000, deaths related to lung cancer will increase worldwide to 2 million, mainly due to an increase in cigarette smoking by young adults. The control of lung cancer is a multiphased research and application strategy. The identification of cancer risks and causes and the reduction of carcinogen exposure are the first two phases of this strategy. This paper deals with four interventions with a significant potential impact on the consumption of tobacco. (a) A total ban on the sale of tobacco to minors would be one of the most effective measures to reduce the prevalence of smoking among teenagers and would result, in the long run, in a decrease in lung cancer. (b) In developing countries, the production of tobacco is expected to continue to increase until the Year 2000 and to result in a rapid increase in the incidence of lung cancer. (c) The taxation of tobacco is a powerful tool to reduce smoking, especially among teenagers. (d) In order to reduce the incidence of lung cancer, it is crucial that health professionals inform patients about the health consequences of smoking. Cancer chemoprevention, which could reduce the risk of lung cancer among smokers and exsmokers, is an alternative to primary prevention. The very first step in the chemoprevention strategy is to characterize the chemopreventive agents. The investigator can test its inhibitory capacity in vitro or proceed directly to a tumor inhibition study in laboratory animals. Tumor inhibition studies are carried out at the maximum tolerated dose. Compounds which have demonstrated inhibitory activity are then entered in the next phase of the strategy, the study of chemopreventive efficacies. Chemopreventive agents are be classified according to their mechanism in inhibiting the process of carcinogenesis. One of the most promising approaches to cancer chemoprevention is the use of a combination of chemopreventive agents. The National Cancer Institute funded the development of 14 compounds or combinations of compounds as lung cancer chemopreventive agents. Biomarkers of early lesions are generally used to assess the efficacies of the chemopreventive agents in clinical trials. The biomarker should be able to measure the early phase of the carcinogenic process, be relatively inexpensive, and be the least invasive possible. The biomarkers which have been developed for lung cancer are: index of metaplasia of bronchial tissues and exfoliated atypical bronchial cells.(ABSTRACT TRUNCATED AT 400 WORDS)

TI: The natural history of lung cancer estimated from the results of a randomized trial of screening.
AU: Walter-SD; Kubik-A; Parkin-DM; Reissigova-J; Adamec-M; Khlat-M
SO: Cancer-Causes-Control. 1992 Mar; 3(2): 115-23
AB: The results from a randomized controlled trial of screening for lung cancer in Czechoslovakia have been used to estimate parameters of the natural history, using a model to simulate the disease process and the effects of screening. The results suggest that the period before clinical presentation during which lesions can be detected by screening is very short (seven to eight months). This implies that to detect three-quarters of all lung cancers by screening, two examinations per year are necessary, and that such a program would advance diagnosis by six months if there were complete participation. The results of the trial itself suggest that the benefit, in terms of a reduction in mortality from lung cancer, is likely to be very small.

TI: [Preliminary specification of X-ray CT for lung cancer screening (LSCT) and its evaluation on risk-cost-effectiveness]
AU: Iinuma-T; Tateno-Y; Matsumoto-T; Yamamoto-S; Matsumoto-M
SO: Nippon-Igaku-Hoshasen-Gakkai-Zasshi. 1992 Feb 25; 52(2): 182-90
AB: In Japan mass screening for lung cancer is widely performed by chest X-ray film of 10 x 10 cm, with sputum cytology for high-risk groups. However, the effectiveness of the present mass screening program for lung cancer has been

less than expected, although some date have shown improvement of the survival rate as a result of mass screening. We propose a new X-ray CT method of screening for lung cancer called Lung Cancer Screening CT (LSCT). The preliminary specifications for LSCT are as follows: scan speed is within 10 sec for a 40 cm length with a 1 cm slice thickness, and the absorption dose for lung is less than 1 cGy. A computer-assisted diagnostic system is necessary for image reading. The risk-benefit analysis of LSCT indicates that Japanese men and women over 45 years old can be screened in 1992 and thereafter. The cost per person.year of LSCT screening is estimated to be 1.4 x 10(6) yen for a population incidence rate of 50 x 10(-5) person/year and average life expectancy of 30 years. In 1992 the above condition will be satisfied by men aged 55 years old and above, but not by women of any age. We believe that LSCT should be developed further and tested in the high-risk group of men with an incidence rate of 100 x 10(-5) persons/year or more.

TI: [Flow cytometric analysis in non-small-cell bronchial carcinoma and its prognostic significance]
AU: Liewald-F; Sunder-Plassmann-L; Valet-G; Wulf-G; Weiss-M; Schildberg-FW
SO: Chirurg. 1992 Mar; 63(3): 205-10
AB: Tumor and lymph node infiltration, and the DNA-ploidy status of a tumor contain prognostic information in addition to the information obtained by histological examination of surgical samples. Specimens from 112 patients with non-small-cell lung carcinoma obtained immediately after surgery were investigated by means of flow cytometry. DNA-aneuploidy was found in 43% of the primary tumors. Independent from tumor stage, patients with DNA-euploid tumors lived significantly longer (p less than 0.01) than with DNA-aneuploid carcinomas. In 29 cases the DNA-ploidy status of the primary tumor (PTU) could be compared with that of the N2 lymph node metastases (LM). 7 samples revealed a change from DNA aneuploidy in the PTU to DNA-euploidy in the LM. Patients with DNA-euploid PTU and DNA-euploid LM lived significantly longer than patients with DNA-aneuploid PTU/DNA-euploid LM, and patients with DNA-aneuploid PTU/DNA-aneuploid LM. In case of lymph node infiltration only the simultaneous measurement of DNA ploidy of PTU and LM offers an accurate prognostic evaluation. Local tumor recurrence exhibited stability of DNA ploidy, showing DNA euploidy in 12 out of 13 PTU and their corresponding recurrent tumor. Thus, the DNA-ploidy status offers additional prognostic informations which is useful for an extended tumor classification.

TI: Lung cancer in filling station attendants.
AU: Grandjean-P; Andersen-O
SO: Am-J-Ind-Med. 1991; 20(6): 763-8
LC: SH 1831
AB: At the Danish census on 9 November 1970, 4,055 men and 1,195 women aged 20-64 years indicated an employment that was coded as retail sale of oil and gasoline; almost all individuals probably worked as filling station attendants. Record linkage at Danmarks Statistik showed that 529 of the men had died during the following 17 years. Respiratory cancer (75 deaths) was the only cause of death that showed a significant excess (standardized mortality ration, 1.58; 95% confidence interval, 1.25-2.00) when compared to all men gainfully employed at the time of the census. An increased mortality due to cardiovascular disease could not be related to any particular diagnostic subgroup; the mortality in women did not differ from expected rates. These results are in accordance with data from other countries on occupational groups exposed to high levels of exhaust fumes.

TI: Trends in lung cancer mortality in Scotland and their relation to cigarette smoking and social class.
AU: Williams-FL; Lloyd-O
SO: Scott-Med-J. 1991 Dec; 36(6): 175-8
LC: ZJ 10342
AB: This paper describes the trends in lung cancer rates in Scottish men and women during 1959-85, the relationship between lung cancer and cigarette consumption, and between lung cancer and social class, and the urban-rural gradient of lung cancer. Lung cancer rates in Scottish men have declined in all age groups under the age of 74 for at least the past two decades; the most notable decrease was in men aged 40-44 years, whose rates halved between 1970 and 1980. In women, who began smoking in large numbers only after World War II, lung cancer mortality declined slightly in those between 40-54 years and rose in those over 54 years. Trends in cigarette consumption did not fully explain the decline in lung cancer. Marked urban-rural gradients in the SMRs for lung cancer were evident in all periods, and these strengthened over time.

Correlations between lung cancer and social class differed markedly from those found in previous studies, except for those with social classes II and V.

TI: A case-control study on occupational lung cancer risks in an industrialized city of Japan.
AU: Yamaguchi-N; Kido-M; Hoshuyama-T; Manabe-H; Kikuchi-Y; Nishio-T; Ohshima-LH; Watanabe-S
SO: Jpn-J-Cancer-Res. 1992 Feb; 83(2): 134-40
AB: A hospital-based case-control study was conducted to evaluate occupational risks of lung cancer in an industrialized city of Japan. The lung cancer cases were obtained from 3 major hospitals in the city. The control group consisted of patients with a variety of diseases hospitalized in the same wards of the same hospitals as the cases. After matching on sex, 5-year age category and hospital, 144 cases and 676 controls comprised the study group. A self-administered questionnaire was used to obtain lifetime job histories and smoking status. The conditional logistic regression model was used to estimate relative risks after controlling for smoking and employment in other jobs. The workers in shipbuilding, ironworks and other plants (mostly chemical plants) showed statistically significant increases in lung cancer risk with relative risks of 6.18, 2.02, and 2.66, respectively. An increase in risk with the duration of employment was also observed in the "other plants" category. Building and road construction workers also showed increased relative risks, 1.95 and 1.79, but they were not significant. When the risk was evaluated on the exposure chemicals, the workers exposed to inorganic acids and bases had significantly increased risk. The workers exposed to asbestos, dust or organic chemicals also showed increased risk but the effects were not significant. The combined effect of smoking and employment in ironworks showed a good fit to an additive model, while that in the "other plants" category was closer to a multiplicative model.

TI: The effect of surgical treatment on survival from early lung cancer. Implications for screening.
AU: Flehinger-BJ; Kimmel-M; Melamed-MR
SO: Chest. 1992 Apr; 101(4): 1013-8
AB: We assessed the effect of surgery on survival from stage I non-small-cell lung cancer based on data collected in these screening programs. The majority of patients diagnosed in each program were treated by surgical resection, but 5 percent of the Sloan-Kettering group, 21 percent of the Hopkins group and 11 percent of the Mayo group failed to receive surgical treatment. Approximately 70 percent of the stage I patients in each program who were treated surgically survived more than five years, but there were only two five-year survivors among those who did not have surgery. We conclude that patients with lung cancers detected in stage I by chest x-ray film and treated surgically have a good chance of remaining free of disease for many years. Those stage I lung cancers which are not resected progress and lead to death within five years. Therefore, every effort should be made to detect and treat lung cancer early in high-risk populations.

TI: Gender and histologic type variations in smoking-related risk of lung cancer.
AU: Brownson-RC; Chang-JC; Davis-JR
SO: Epidemiology. 1992 Jan; 3(1): 61-4
AB: We conducted a registry-based case-control study to examine the relation between smoking and lung cancer by gender and histologic type. Our analyses were based on 14,596 cases and 36,438 age-matched controls. Relative risk associated with ever-smoking, and level of smoking was consistently higher in females than males for all lung cancers combined (ever-smoking odds ratios: 12.7 for females and 9.1 for males) and for each histologic type except adenocarcinoma. Female-male differences in relative risk were larger in younger age groups. The largest estimates of the attributable fraction due to smoking were observed for small cell carcinoma (97% in females and 91% in males); conversely, the smallest value was noted for adenocarcinoma (86% in females). Although our study was unable to measure absolute risk, our findings, other recent studies, and contemporary female smoking patterns raise concerns that female smokers may assume a proportionally greater burden of lung cancer morbidity and mortality in the future.

TI: Synergism between occupational arsenic exposure and smoking in the induction of lung cancer.
AU: Hertz-Picciotto-I; Smith-AH; Holtzman-D; Lipsett-M; *Alexeeff-G*

SO: Epidemiology. 1992 Jan; 3(1): 23-31
AB: We assembled data from numerous studies to examine whether active smoking and occupational exposure to arsenic act synergistically (more than additively) to increase the risk of lung cancer. Although several smaller studies lacked the power to reject simple additive relations, the joint effect from both exposures consistently exceeded the sum of the separate effects by about 70 to 130%. The only study not showing a greater than additive effect appeared to have inadequate data to address this question. We calculated the excess fractions for the synergism; these showed that a minimum of between 30% and 54% of lung cancer cases among those with both exposures could not be attributed to either one or the other exposure alone. Previous authors addressing the synergism between arsenic exposure and smoking have evaluated deviations from a multiplicative model, which is inappropriate for this purpose. Reports of no interaction or "negative" interaction have therefore been misleading. Taken as a whole, the evidence is compelling that arsenic and smoking act in a synergistic manner to produce lung cancer. Substantial reductions in the lung cancer burden of smokers occupationally exposed to arsenic could be achieved by reductions in either exposure. The mechanism for the synergism is unclear.

TI: Bias in the attribution of lung cancer as cause of death and its possible consequences for calculating smoking-related risks.
AU: Sterling-TD; Rosenbaum-WL; Weinkam-JJ
SO: Epidemiology. 1992 Jan; 3(1): 11-6
AB: Most published calculations of mortality risk, especially those for lung cancer associated with smoking, are based almost exclusively on the underlying cause as recorded on death certificates. Such risk calculations implicitly assume that the conditional probability of recording lung cancer as the underlying cause of death, given that it really is the underlying cause, is the same for all exposure groups. If these probabilities are not equal for all exposure groups, we call the resulting bias a cause of death attribution bias. We analyzed the 1986 National Mortality Followback Survey, a sample of 18,733 U.S. death certificates, and the 1954-1962 Dorn study, a follow-up study of approximately 250,000 holders of U.S. Veterans Life Insurance. Both data sets include information on the smoking habits of decedents and on the underlying and contributing causes of their deaths. We found that lung cancer as an underlying cause is recorded with a much smaller relative frequency if the decedent is known to be a never-smoker and with a much larger relative frequency when the decedent is known to be a smoker. On the other hand, lung cancer as a contributing cause is recorded with a much larger frequency if the decedent is known to be a never-smoker and with a much smaller frequency when the decedent is known to be a smoker. The reverse is true for cancers other than of the lung. There is no similar pattern related to smoking for other causes of death (specifically for myocardial infarction, other chronic ischemic heart disease, diabetes, or cerebrovascular disease).(ABSTRACT TRUNCATED AT 250 WORDS)

TI: A nested case-control study of lung cancer among silica exposed workers in China.
AU: McLaughlin-JK; Chen-JQ; Dosemeci-M; Chen-RA; Rexing-SH; Wu-Z; Hearl-FJ; McCawley-MA; Blot-WJ
SO: Br-J-Ind-Med. 1992 Mar; 49(3): 167-71
AB: In an attempt to assess whether silica induces lung cancer, a nested case-control study of 316 male lung cancer cases and 1352 controls was carried out among pottery workers and tungsten, copper-iron, and tin miners from five provinces in south central China. Exposure to dust and silica for each study subject was evaluated quantitatively by cumulative exposure measures based on historical industrial hygiene records. Measurements on confounders such as inorganic arsenic, polycyclic aromatic hydrocarbons (PAHs), and radon were also collected from the worksites. Information on cigarette smoking was obtained by interviews of the subjects or their next of kin. A significant trend of increasing risk of lung cancer with exposure to silica was found for tin miners, but not for miners working in tungsten or copper-iron mines. Concomitant and highly correlated exposures to arsenic and PAHs among tin miners were also found. Risk of lung cancer among pottery workers was related to exposure to silica, although the dose-response gradient was not significant. Risks of lung cancer were significantly increased among silicotic subjects in iron-copper and tin mines, but not in pottery factories or tungsten mines. The results of this study provide only limited support for an aetiological association between silica and lung cancer.

TI: Inhibition of N-nitrosodiethylamine- and 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone-induced tumorigenesis in A/J mice by green tea and black tea.
AU: Wang-ZY; Hong-JY; Huang-MT; Reuhl-KR; Conney-AH; Yang-CS
SO: Cancer-Res. 1992 Apr 1; 52(7): 1943-7
AB: The effect of p.o. administration of tea on nitrosamine-induced carcinogenesis was investigated. Female A/J mice were given N-nitrosodiethylamine (NDEA) (10 mg/kg) p.o. once a week for 8 weeks and were killed 16 weeks after the last dose. More than 90% of the mice had forestomach and lung tumors. The animals had an average of 8.3 forestomach and 2.5 lung tumors/mouse. With 0.63 or 1.25% green tea infusion (12.5 g green tea leaves brewed with 1 liter of boiling water) as the sole source of drinking water for the entire experimental period, the pulmonary tumor incidence was decreased by 18 or 44%, and the tumor multiplicity was reduced by 36 or 60%, respectively. The treatments also decreased the forestomach tumor incidence by 18 or 26% and tumor multiplicity by 59 or 63%, respectively. Administration of 0.63 or 1.25% green tea infusion, either during the NDEA treatment period only or starting 1 week after the completion of NDEA treatment, also decreased the pulmonary tumor incidence and multiplicity and the forestomach tumor multiplicity. The inhibitory effects of green tea infusion were also observed in a similar experiment using a higher dosage of NDEA (20 mg/kg). Treatment of female A/J mice with a single dose (103 mg/kg) of 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone (NNK) resulted in the formation of pulmonary adenomas in almost all of the animals with an average of 9.3 tumors/mouse after 16 weeks. When 0.6% decaffeinated green tea or black tea extract was given during the NNK-treatment period, tumor multiplicity was reduced by 67 or 65%, respectively. When the tea extract was given after the NNK-treatment period until the end of the experiment, 0.6% green tea extract decreased the tumor incidence and multiplicity by 30 and 85%, respectively. In this protocol, 0.6% black tea extract reduced tumor multiplicity by about 63% but did not significantly affect the tumor incidence. The results clearly demonstrated an inhibitory action of green tea and black tea on nitrosamine-induced tumorigenesis.

TI: Fitting cancer mortality data with cumulative damage models.
AU: Becker-N; Rittgen-W
SO: Math-Biosci. 1992 Feb; 108(1): 57-73
AB: Cumulative damage models conceive the epidemiologically observed aspects of carcinogenesis as some kind of total balance over a complex biological process and suggest that this total balance might behave as a wear-and-tear process. The essential concepts of this mechanistic model are exposure to a carcinogenically damaging environment and resistance of a host system against those damages. Intensity of exposure and magnitude of host resistance are the parameters to be assessed. The paper describes (1) the statistical methods for fitting this model to birth cohort data; (2) for which cancer sites the model provides acceptable fits and for which it does not; and (3) how model extensions provide improvements in the goodness of fit. It is shown that from a theoretical viewpoint the consideration of extra-Poisson variation is needed for descriptive epidemiological applications. The practical examples indicate that the present version of the model provides acceptable fits for only a few cancer sites and that refinements are needed in the majority of sites. However, plausible model extensions suggest considerable improvements of goodness of fit.

TI: Prognostic value of flow cytometrically determined DNA-ploidy, intracellular pH and esterase activity of non-small cell lung carcinomas.
AU: Liewald-F; Sunder-Plassmann-L; Dienemann-H; Kahle-H; Wulf-G; Valet-G
SO: Anal-Cell-Pathol. 1992 Mar; 4(2): 103-14
AB: 30 surgical specimens of patients with non-small cell lung carcinomas (NSCLC) were investigated. Significant increases of intracellular pH values in epithelial and inflammatory cells, in the percentage of dead epithelial and inflammatory cells and in the cell volume of vital inflammatory cells in cancerous lung tissue were encountered. Furthermore, decreases of the esterase activity of vital epithelial cells and of the percentage of free cell nuclei were observed. The DNA aneuploidy in 36.6% of the tumours was frequently associated with non-squamous cell carcinomas and stage II, III, IV tumours. Patients with DNA aneuploid tumours had a significantly shorter survival rate than those with DNA euploid tumours. Within the different tumour stages a similar tendency was observed which was, however, only significant in stage III tumour patients. Stage III tumours constitute therefore a heterogeneous entity with a worse prognosis for DNA aneuploid tumour patients. The intracellular pH values and esterase activity as well as the cell volume, the

percentage of free cell nuclei and dead inflammatory or epithelial cells contained no significant prognostic information.

TI: Incidence of cancers of the larynx and lung near incinerators of waste solvents and oils in Great Britain.
AU: Elliott-P; Hills-M; Beresford-J; Kleinschmidt-I; Jolley-D; Pattenden-S; Rodrigues-L; Westlake-A; Rose-G
SO: Lancet. 1992 Apr 4; 339(8797): 854-8
AB: The Small Area Health Statistics Unit (SAHSU) is a new independent facility for the investigation of disease near industrial installations in the UK. SAHSU analysed the incidence of cancers of the larynx and lung near the incinerator of waste solvents and oils at Charnock Richard, Coppull, Lancashire (which operated between 1972 and 1980) and nine other similar incinerators in Great Britain, after reports of a cluster of cases of cancer of the larynx near the Charnock Richard site. Postcoded cancer registration data were available for 1974-84 in England and Wales and 1975-87 in Scotland. Lag periods of 5 and 10 years were used between start-up (or first registration) of the incinerators and cancer incidence. Standardised observed/expected (O/E) ratios were assessed within 3 km and 3-10 km of each site and then aggregated over all sites. Expected values were based on national rates (regionally adjusted) with and without stratification by Carstairs' index, a measure of the socioeconomic profile of areas that uses census data for enumeration districts. Data were also assessed over a range of circles up to 10 km to test for trend in O/E ratios with distance. For Charnock Richard, none of the O/E ratios within 3 km or from 3-10 km differed significantly from unity, for either cancer or lag period. In the analysis of all sites with stratification by Carstairs' index, none of these O/E ratios differed significantly from unity for the two cancers. There was no evidence of decreasing risk with distance from the sites of either cancer. We conclude that the apparent cluster of cases of cancer of the larynx reported near Charnock Richard was unlikely to be due to its former incinerator.

TI: The value of sputum cytology.
AU: MacDougall-B; Weinerman-B
SO: J-Gen-Intern-Med. 1992 Jan-Feb; 7(1): 11-3
AB: OBJECTIVE: To assess the value of cytologic examination of expectorated sputum in the diagnosis and management of patients with suspected lung cancer. DESIGN: Retrospective chart review. SETTING: Inpatient wards, tertiary care university hospital. MEASUREMENTS AND MAIN RESULTS: The charts of 357 patients were reviewed. Two hundred eighty-eight of the 357 patients had had initial sputum cytologic examination prior to other diagnostic procedures, of which 41 (15%) had positive cytologic results. Thirty-six of the 41 were confirmed histologically or shown to have metastatic spread by noninvasive tests. Of the 222 patients with negative or unsatisfactory sputum tests, 97 went on to bronchoscopy and 35 had needle-aspiration biopsies. In the population of patients whose chest x-rays were highly suggestive of primary or metastatic lung cancer, the positive rate for cytologic examination was 38/94 (40%). There was no false-positive test in this study. Of the 50 patients with positive cytologic results, five (10%) had diseases that were of a different cell type; two of these five (40%) had diseases that involved small-cell cancer. There was an unsatisfactory delay in obtaining these samples for analysis. CONCLUSIONS: Sputum cytology was found to be too insensitive and insufficiently accurate to be included in the routine workup of a patient suspected of having lung cancer. The results of the test did not influence further diagnostic procedures. This test should, therefore, be reserved for patients considered on initial assessment to be too sick for further investigations and treatment.

TI: Passive smoking and canine lung cancer risk.
AU: Reif-JS; Dunn-K; Ogilvie-GK; Harris-CK
SO: Am-J-Epidemiol. 1992 Feb 1; 135(3): 234-9
AB: A case-control study was conducted to determine whether household exposure to environmental tobacco smoke is associated with an increased risk for lung cancer in pet dogs. Lung cancer cases and controls with other forms of cancer were obtained from two veterinary teaching hospitals during 1985-1987. Exposures assessed included the number of smokers in the household, the amount smoked, and the proportion of time spent indoors by the pet. A weak relation was found for exposure to a smoker in the home (odds ratio = 1.6, 95% confidence interval 0.7-3.7), after controlling for confounding in stratified analyses. Strong evidence for a further increase in risk associated with more than one smoker in the home was not found, nor was a significant trend

observed for increasing number of packs of cigarettes smoked per day or an exposure index based on number of smokers in each household, packs smoked per day, and the proportion of time the dog spent within the home. However, skull shape appeared to exert effect modification; the risk was restricted to breeds with short and medium length noses (odds ratio = 2.4, 95% confidence interval 0.7-7.8). Despite the inconclusive findings of the current study, epidemiologic studies in pet animals may add to our understanding of environmental tobacco smoke effects in human populations.

TI: Cancer mortality survey in a spa area (Misasa, Japan) with a high radon background.
AU: Mifune-M; Sobue-T; Arimoto-H; Komoto-Y; Kondo-S; Tanooka-H
SO: Jpn-J-Cancer-Res. 1992 Jan; 83(1): 1-5
AB: The 1952-88 cancer mortality records for inhabitants of the Misasa spa area, Japan, which has a high radon background, and a neighboring control area without any radon spa were analyzed (average outdoor Rn concentration: 26 mBq.liter-1 in Misasa vs. 11 mBq.liter-1 in the control area). Standardized mortality ratios (SMRs) for cancers of all sites were significantly lower among the inhabitants of both Misasa (male 0.538; female 0.463) and the control area (male 0.850; female 0.770), than in the whole Japanese population. Poisson regression analysis showed that the relative risks among the inhabitants of Misasa were significantly lower than in the control area for deaths from cancers of all sites (0.67) and stomach cancer (0.59). The relative risk of lung cancer death was also lower (0.55 times) in Misasa than in the control area, although the difference was not statistically significant. These results suggest that the linear no-threshold hypothesis for radiation risk may not be valid for exposure to low doses of radon.

TI: Does beta-carotene explain why reduced cancer risk is associated with vegetable and fruit intake?
AU: Ziegler-RG; Subar-AF; Craft-NE; Ursin-G; Patterson-BH; Graubard-BI
SO: Cancer-Res. 1992 Apr 1; 52(7 Suppl): 2060s-2066s
AB: Increased intake of vegetables, fruits, and carotenoids and elevated blood levels of beta-carotene are consistently associated with reduced risk of lung cancer in epidemiologic studies. Epidemiologic research also suggests that carotenoids may reduce the risk of other cancers, although the evidence is less extensive and consistent. The simplest explanation is that beta-carotene is protective. However, the possible roles of other carotenoids, other constituents of vegetables and fruits, and associated dietary patterns have not been adequately explored. To evaluate these alternative hypotheses, we are undertaking three lines of research. (a) With dietary data from the 1987 National Health Interview Survey and the 1982-1984 Epidemiologic Follow-up of the first National Health and Nutrition Examination Study, we have determined which food groups and nutrients are highly correlated with vegetable and fruit intake. (b) We have developed and characterized a liquid chromatography method for optimal recovery and resolution of the common carotenoids in blood, specifically lutein, zeaxanthin. beta-cryptoxanthin, lycopene, alpha-carotene, and beta-carotene. (c) In a population-based case-control study of lung cancer in white men in New Jersey, we are assessing whether estimates of the intake of the individual carotenoids might produce stronger inverse associations than estimates of provitamin A carotenoids based on current food composition tables.

TI: [X-ray diagnosis of peripheral chondromatous hamartomas of the lungs]
AU: Sokolov-VA; Kartashov-VM; Glushko-LS; Rudakova-NA
SO: Vestn-Rentgenol-Radiol. 1991 Nov-Dec(6): 9-12
AB: A roentgenomorphographic picture was compared with gross specimens of the lungs in 104 operated on patients with peripheral harmatochondromas. Eighty three patients were followed-up roentgenologically for 2-18 years. The resultant three variants of an x-ray picture of hamartochondromas were revealed. CT investigation showed a benign process in a majority of patients, hamartochondrosarcoma was diagnosed in 36.5 per cent. Shadow size enlargement, calcifications and their increase are not signs of malignancy.

TI: [Preventive operations of the lung and bronchial system]
AU: Krumhaar-D; Mollinedo-J; Gau-A; Sibold-M
SO: Langenbecks-Arch-Chir-Suppl-Kongressbd. 1991: 211-8
AB: There are four major prophylactic indications in thoracic surgery: (1) Intra-pulmonary coin lesions with malignancy up to 50% and other potentially malignant tumors (carcinoid tumor, cylindroma, mucoepidermoid tumor,

papilloma); (2) mediastinal tumors with potentially malignant growth (teratoma); (3) chronic lung infections including tuberculosis (bronchiectasis, abscess, chronic pneumonia, persistent tuberculoma, tuberculous cavity, destroyed lobe/lung); (4) cystic pulmonary disease followed by frequent complications (infection, bleeding, pneumothorax).

TI: Adenocarcinoma of the lung: a contribution on prognosis after potentially curative resection.
AU: Huwer-H; Donie-HW; Volkmer-I; Eich-F
SO: Thorac-Cardiovasc-Surg. 1991 Dec; 39(6): 376-8
LC: SH 1833 1839 9999
AB: From 1976 to 1989, 166 patients were operated on for primary adenocarcinoma of the lung. For better comparison, all the tumors were categorized retrospectively according to the TNM system of the UICC 4th edition of 1987. One hundred and thirty-eight patients could be potentially curatively operated. The average survival time following incomplete resection was 5 months and after potentially curative resection it was 65 months for stage I, 22 months for stage II and 6.5 months for stage IIIa. The 5-year survival rate was 53.7% for stage I, 18.5% for II and 0% for IIIa. The differences between the tumor stages are statistically significant. At the time of writing a large number of the patients have already died due to either recurrence of the tumor or, as in most cases, secondary metastases distant from the primary growth CI: 32%, II: 66%, IIIa: 79%). Comparison of the results of potentially curative operations in patients with adenocarcinoma and those with squamous cell carcinoma show a better prognosis in the equivalent stages for cases of squamous cell carcinoma. In seven cases there was the situation of ipsilateral pulmonary metastasis which could be subjected to potentially curative resection together with the primary tumor (5 bronchioloalveolar, 2 other adenocarcinomas). The prognosis of these patients was just as good, following resection, as for cases of T2N0 tumors without such metastases.

TI: [Morbidity and surgical treatment of lung cancer in middle-aged and aged patients in Moscow]
AU: Aitakov-ZN
SO: Klin-Med-Mosk. 1991 Dec; 69(12): 73-6
AB: Analysis of lung cancer incidence in Moscow residents shows that of late the rise in the incidence has occurred only in absolute number. Intensive rates remain stable. The number of cases in the age groups 1-59, 60-69 and 70 years and older is approximately the same. Of all the patients who need treatment annually, it is provided to one-third of them and surgical treatment to 16-18%. The percentage of operable cancer patients drops with age. Basing on his own 10-year surgical experience, the author thinks it proper to rise surgical activity in the elderly patients.

TI: Competing events determining relapse-free survival in limited small-cell lung carcinoma. The French Cancer Centers' Lung Group.
AU: Arriagada-R; Kramar-A; Le-Chevalier-T; De-Cremoux-H
SO: J-Clin-Oncol. 1992 Mar; 10(3): 447-51
AB: PURPOSE: We report results in terms of relapse-free survival (RFS), obtained in patients with limited small-cell lung carcinoma (SCLC) treated by four consecutive alternating protocols, using a competing risk approach with local recurrences, distant metastases, and death unrelated to cancer as competing events. PATIENTS AND METHODS: Two hundred two patients with limited SCLC were included in four consecutive protocols alternating radiotherapy and chemotherapy (CT). The alternating schedule consisted of six cycles of CT (doxorubicin, etoposide [VP16213], and cyclophosphamide [CAVP16], plus methotrexate in the first protocol; cisplatin replaced methotrexate in the other three protocols) and three courses of thoracic radiotherapy at a total dose of 45, 55, 65, and 61 Gy in the four consecutive protocols, respectively (accelerated hyperfractionation was used in the first course of the fourth protocol). A 1-week rest followed each CT cycle and each course of radiotherapy. Seventy-six percent of patients were in complete remission at the end of the induction treatment. RFS variables were determined according to a model assuming competing risks to define the first cause of failure (local disease, distant metastasis, or intercurrent death). RESULTS: No significant differences were observed between the four treatment groups. Overall results showed a 2-year cumulative incidence rate of failure of 75%. When analyzed, the first cause of failure was local recurrence only, 33%; distant only, 25%; distant and local simultaneously, 9%; and intercurrent death, 8%. CONCLUSIONS: The methodology of competing risks allowed an unequivocal description of first

events in limited SCLC. The extent of the local problem has been relatively overshadowed by the use of conventional descriptive methods.

TI: Ki-67 immunostaining and survival in operable lung cancer.
AU: Tungekar-MF; Gatter-KC; Dunnill-MS; Mason-DY
SO: Histopathology. 1991 Dec; 19(6): 545-50
LC: SH 0006 1831 1833 1838 3225 JS 7971 7985 7997 7973A
AB: One hundred and eighty-seven operable lung tumours were immunostained with the monoclonal antibody Ki-67 and divided into groups of high, moderate or low proliferation. Patients have been followed clinically for up to 7 years to ascertain whether this immunocytochemical measurement reflected tumour behaviour in terms of survival. The majority of the tumours were squamous cell carcinomas (104 cases) and adenocarcinomas (60 cases). These were divided into three groups of low, intermediate and high growth fraction, in which survival was better for tumours of lower proliferative rate up to approximately 2 years after operation. By 5 years these differences had largely disappeared and all tumours of a particular type showed a similar survival curve. Small cell carcinomas (13 cases) had high Ki-67 labelling indices, with more than 60% of patients deal in the first year, whereas carcinoid tumours (10 cases) had low labelling rates and all but one are still alive. We conclude that measurement of lung tumour growth rate with the monoclonal antibody Ki-67 shows promise as a possible indicator of short-term survival and perhaps as a means of choosing a group of patients with adenocarcinomas and squamous cell carcinomas for post-operative chemotherapy.

TI: Search for anti-metastatic therapy: effects of phenytoin on B16 melanoma metastasis.
AU: Dyce-M; Sharif-SF; Whalen-GF
SO: J-Surg-Oncol. 1992 Feb; 49(2): 107-12
AB: The ability to metastasize requires that tumor cells be able to degrade matrix. Nontoxic compounds that inhibit matrix digestion might be useful as anti-metastatic agents. We have investigated whether phenytoin, a drug commonly used in clinical practice that inhibits the production of collagenase by some cells, inhibits metastases in a standard animal model of metastasis: In vitro, phenytoin inhibited the proliferative response of B16 F10 melanoma cells to serum-containing media (75% inhibition at 25 micrograms/ml) but had no effect on their ability to degrade a type I collagen gel (1-100 micrograms/ml). Treatment of these cells with phenytoin prior to inoculation in vivo did not inhibit tumor growth, implantation in a surgical wound, or incidence of spontaneous metastases from a primary tumor growing in the foot. Pretreatment of mice with phenytoin (15, 40, and 75 mg/kg/day) diminished pulmonary metastases following tail vein injection in a minimal but dose dependent fashion; mean number of pulmonary colonies 4.6 +/- 3.1 (75/mg/kg/day) vs. 10.2 +/- 9.9 (control). However, tumor growth, implantation, and spontaneous metastases were not inhibited by pretreating the mice with the same doses of phenytoin. It is concluded that phenytoin has an insignificant inhibitory effect on tumor growth and metastasis.

TI: Prospective mortality study among iron miners.
AU: Pham-QT; Chau-N; Patris-A; Trombert-B; Henquel-JC; Geny-M; Teculescu-D
SO: Cancer-Detect-Prev. 1991; 15(6): 449-54
LC: ZJ 10342
AB: A prospective mortality study was conducted over a period of 5 years on a group of 13,801 iron miners, who were alive on January 1, 1982. During this 5-year period, 1813 deaths were registered. For 1222 (67.4%), the cause of death and work history are known. For 135 (7.4%), the cause of death is known, but not the work history. For 455 (25.1%), the cause of death is unknown. Proportional mortality ratio (PMR) is significantly higher than 1 for lung cancer (PMR = 2.51, p less than 0.001) and for stomach cancer (PMR = 2.31, p less than 0.001). The results are discussed in regard to occupational risks that result in these two kinds of increased mortality rates, and the hypothesis of redox activity on the surface of dust particles is advanced as a common denominator.

TI: The synthetic hepatic peptides pyroglutamylglutamylglycylserylasparagine and pyroglutamylglutamylglycylserylaspartic acid inhibit growth of MH1C1 rat hepatoma cells transplanted into Buffalo rats or athymic mice.
AU: Paulsen-JE; Hall-KS; Rugstad-HE; Reichelt-KL; Elgjo-K
SO: Cancer-Res. 1992 Mar 1; 52(5): 1218-21
AB: Repeated i.p. injections of the synthetic peptides

pyroglutamylglutamylglycylserylasparagine and pyroglutamylglutamylglycylserylaspartic acid inhibited the long-term growth of MH1C1 rat hepatoma cells by 50-70% in three in vivo models: metastatic colony growth in the lungs of young Buffalo rats; s.c. tumor growth in young Buffalo rats; and s.c. tumor growth in athymic mice. The amide free peptide pyroglutamylglutamylglycylserylaspartic acid which inhibited the tumor growth in all the models showed a curvilinear dose-response relationship with a maximal effect at 1000 pmol/animal in mice and at 100 pmol/animal in rats. The amidated peptide pyroglutamylglutamylglycylserylasparagine, which was only tested in the lung model, showed growth inhibition with 2, 20, or 200 pmol/animal, but 200 pmol/animal was most effective. We have recently reported that these peptides show cochromatography with hepatic growth inhibitory peptides, isolated from mouse liver.

TI: Inhibition of experimental metastasis and cell adhesion of B16F1 melanoma cells by inhibitors of protein kinase C.
AU: Dumont-JA; Jones-WD Jr; Bitonti-AJ
SO: Cancer-Res. 1992 Mar 1; 52(5): 1195-200
AB: Phorbol esters which activate protein kinase C (PKC) have been shown to enhance experimental lung metastasis. Therefore, it was reasoned that inhibitors of PKC might also modulate metastasis. We have investigated this possibility using a PKC inhibitor, MDL 27,032 [4-propyl-5(4-pyridinyl)-2(3H)-oxazolone], as well as staurosporine and H-7. Treatment of B16F1 murine melanoma cells with MDL 27,032 for 24 h in culture and subsequent i.v. injection of the cells into mice resulted in greater than 90% inhibition of lung metastasis. Inhibition of metastasis was time dependent, with 90% of maximum inhibition occurring by 8 h of incubation. The 50% inhibitory concentration (IC50) for inhibition of metastasis with MDL 27,032 was 7 microM, a value similar to that for the inhibition of B16F1 membrane-associated PKC (IC50 = 13 microM) but not cytosolic PKC (IC50 = 54 microM). B16F1 cells treated with MDL 27,032 for 24 h were less adherent than untreated cells to extracellular matrix/basement membrane proteins. Adhesion to fibrinogen and collagen IV was inhibited (IC50 = 6 microM and 48 microM, respectively) by MDL 27,032, whereas adherence to laminin and fibronectin was not affected, indicating that the drug affects specific adhesion molecules. MDL 27,032-treated cells were also found to be less adherent than untreated cells to human umbilical vein endothelial cells. The phosphorylation of an 80-kDa B16F1 cell plasma membrane protein was stimulated under conditions known to stimulate PKC activity, and MDL 27,032 inhibited this phosphorylation in a dose-dependent manner. MDL 27,032 was more potent than H-7 for the inhibition of metastasis but was significantly less potent than staurosporine. These results support the hypothesis that there is a critical role for PKC-mediated phosphorylation of cell surface adhesion receptors in metastasis.

TI: Indoor radon and lung cancer. Estimating the risks [see comments]
AU: Samet-JM
SO: West-J-Med. 1992 Jan; 156(1): 25-9
AB: Radon is ubiquitous in indoor environments. Epidemiologic studies of underground miners with exposure to radon and experimental evidence have established that radon causes lung cancer. The finding that this naturally occurring carcinogen is present in the air of homes and other buildings has raised concern about the lung cancer risk to the general population from radon. I review current approaches for assessing the risk of indoor radon, emphasizing the extrapolation of the risks for miners to the general population. Although uncertainties are inherent in this risk assessment, the present evidence warrants identifying homes that have unacceptably high concentrations.

TI: [Detection of bronchial cancer. Realities and perspectives]
AU: Tredaniel-J; Stucker-I; Migaud-I; Hirsch-A
SO: Rev-Mal-Respir. 1991; 8(6): 543-50
LC: SH 1833
AB: Primary bronchial cancer is responsible for at least 20,000 deaths per year in France. The treatment of cancer in the clinical phase remains disappointing. Numerous trials, which are reviewed here chronologically, have looked for a way to improve the prognosis by an earlier detection of this tumour. Unfortunately numerous methodological approaches have not been able to avoid the fact that the real value of such an early diagnosis is not always known. The only clear conclusion of these studies is that cytological examination of the expectorate does not lead to an improved survival in patients when compared to that which is obtained with the single radiographic