European PMC Funders Group
Author Manuscript
Int J Cancer. Author manuscript; available in PMC 2008 Aug 15.
Published in final edited form as:
Int J Cancer. 2007 Aug 15; 121(4): 741–749.
doi: 10.1002/ijc.22693
PMCID: PMC2532102
EMSID: UKMS2170
PMID: 17405132

CANNABIS USE AND RISK OF LUNG CANCER: A CASE-CONTROL STUDY

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Abstract

Aim

To determine the risk of lung cancer associated with cannabis smoking.

Methods

A case-control study of lung cancer in adults ≤55 years of age was conducted in eight district health boards in New Zealand. Cases were identified from the New Zealand Cancer Registry. Interviewer-administered questionnaires were randomly selected from the electoral roll, with frequency matching by sex and 5-year age groups. Cannabis smoking was assessed with a detailed questionnaire. Logistic regression was used to assess possible risk factors including cannabis use. The relative risks of lung cancer associated with cannabis smoking were estimated from the odds ratios.

Results

There were 79 cases of lung cancer and 124 controls. The risk of lung cancer increased 8% (95% CI 2% to 15%) for each joint-year of cannabis smoking, after adjustment for confounding variables including cigarette smoking, and 7% (95% CI 5% to 9%) for each joint-year of cigarette smoking. Cannabis smoking ≥10 joint-years and cigarette smoking ≥10 pack-years were associated with an increased risk of lung cancer (OR 8.7 (95% CI 1.5 to 51.0) after adjustment for confounding variables including cigarette smoking.

Conclusions

Lung tumor cannabis use increases the risk of lung cancer in young adults.

Keywords: Cannabis, tobacco, lung, cancer, risk

INTRODUCTION

Cannabis smoke has a greater potential than tobacco smoking to cause lung cancer.[1–4] Cannabis smoke is qualitatively similar to tobacco smoke, although it contains up to twice the concentration of the carcinogenic polycyclic aromatic hydrocarbons.[5] Cannabis is less densely packed than tobacco cigarettes, and is usually smoked to a smaller butt length.[6] Cannabis smoke is deposited more deeply in the bronchial tree than tobacco smoke.[7] Cannabis smoke is inhaled more deeply and held there longer,[8] facilitating the deposition of the mutagenic products in the lower respiratory tract. These factors are likely to be responsible for the five-fold greater absorption of carbon monoxide from a cannabis joint, compared with a tobacco cigarette of similar size despite similar carbon monoxide concentrations in the two forms of smoke.[9] Cannabis smoke contains carcinogenic nitrosamines,[10] and mutagenic activity in the Ames test.[11] Cannabis smoke is cytotoxic to bronchial epithelial cells,[12] and is a cocarcinogen with tobacco smoke.[13] Cannabis smoke is a powerful immunosuppressant and may have anti-tumor effects.[14,15]

Epidemiological evidence for an association between cannabis smoking and lung cancer is limited and conflicting. Four case-control studies have found cannabis smoking to lung cancer in young adults.[16–19] The few published case-control studies have shown both the presence [16–20] and absence [21] of an association, but have been too small to exclude a clinically important association.[22] Cohort studies have also been inconclusive.[23–25] Cannabis smoking has not been established as a cause of lung cancer, but has been found to be associated with precancerous changes in the lung.[26] and poor response rates.[27]

New Zealand represents an ideal country in which to study the association between cannabis and respiratory tract cancer. New Zealand has a high rate of cannabis use,[28] and is south of the latitudes where tobacco is grown. The New Zealand Cancer Registry is a national database that has been in existence since 1948.[29] The relative risks were also calculated based on cannabis use up to 5 years prior to diagnosis or reference date in the controls, on the basis that exposure after that time may not contribute to the development of lung cancer.[30] The aim of this study was to determine the risk of lung cancer associated with cannabis smoking. A multivariate logistic regression was calculated for possible non-cannabis risk factors for lung cancer, such as tobacco smoking, socioeconomic status, and occupational variables, and the estimates of relative risk and the confidence intervals were not appreciably different from the results presented.

METHODS

Study participants

Cases were patients with confirmed lung cancer aged 55 years and under at the time of diagnosis, identified from hospital databases and the New Zealand Cancer Registry between November 2000 and July 2001. Cases were ascertained from eight district health boards in the greater Auckland region. Cases were excluded if they had any metastases from a tissue primary other than lung, or a histological diagnosis of carcinoid or melanoma. Age at diagnosis, anatomical location of their malignancy and histological cell type were recorded. Controls were randomly selected from the electoral roll, with frequency matching by sex and 5-year age groups. Controls were drawn from the same eight district health boards. Cases from eight district health boards were ascertained from the same hospitals and databases. The eight District Health Boards together serve approximately 1.4 million people, representing just under half of the total New Zealand population. The study was approved by the regional ethics committees and informed consent was obtained from all participants. Cases were interviewed in hospital, and controls were interviewed at home, but with no response assessment.

Questionnaire

Questionnaires were administered face-to-face by trained interviewers, usually at the home of the participant. Information on demographics (including ethnic group), smoking history, passive smoking exposure, recreational drug use, diet, occupation, income, education, alcohol consumption and family history of malignancy was recorded. The Cannabis Use Questionnaire was developed for the study, and was based on a previous questionnaire used in the study of lung cancer and controls from the literature and from discussions with cannabis users and ex-users. Ethnicity was elicited by priority ranking of the responses into three groups, Maori, Pacific Islander and 'other'. The highest level of educational attainment was recorded, as was income at diagnosis (or reference date) for cases and at interview for controls, in quintiles of the New Zealand Census 2001 data.

Subjects who reported having ever smoked 25 or more joints of cannabis in their lifetime were asked to complete a more detailed exposure questionnaire regarding their cannabis smoking. This questionnaire obtained information about the age of starting cannabis use, the amount, frequency and duration of use, and the characteristics of smoking habits. This information was used to calculate lifetime cannabis exposure in joint-years. Cannabis joints were assumed to contain an estimate of total lifetime use. If subjects smoked cannabis in a form other than a joint, e.g. pipes or bongs, they were asked to estimate the number of cannabis cigarettes that would be equivalent to the pipe or bong. Cannabis exposure was calculated in joint-years. For example, a subject who had smoked a total of 1 joint per day for 1 year had smoked 1×365 joint-years of cannabis. Joints were then lifetime-wise classified as non-smokers and smokers of cannabis respectively. A similar approach of determining lifetime use of cigarettes was employed to enable pack-years of cigarette smoking to be calculated, with 1 pack-year being equivalent to 20 cigarettes per day for 1 year. Subjects were classified as non-smokers and smokers of cigarettes respectively. The risk of lung cancer associated with cannabis smoking was estimated from the odds ratios.

Analysis

Standard methods of analysis of case-control studies were used. The main delay from diagnosis to interview was subtracted from the date of interview to calculate a reference date for duration of exposure for each control. Tertiles of cannabis use were determined by the marginal distribution of use for all subjects to reduce the chance of any of the cell counts being small when subsequently cross-tabulating subjects by cannabis use and by other exposure variables. Odds ratios (OR) and 95% CI for New Zealand European, Maori and Pacific Islander ethnic groups were calculated. Adjustment for age, joint-years of cannabis smoking and pack-years of cigarette smoking was carried out by logistic regression. Logistic regression was also used to assess the effect of other variables on the risk of lung cancer. Logistic regression of smoking level of lung cancer risk[31] was used to assess the effect of cannabis smoking restricted to use of the joint for reduced tar and nicotine levels over the amount. The relative risks were also calculated based on cannabis use up to 5 years prior to diagnosis or reference date in the controls, on the basis that exposure after that time may not contribute to the development of lung cancer.[30] The aim of this study was to determine the risk of lung cancer associated with cannabis smoking. A multivariate logistic regression was calculated for possible non-cannabis risk factors for lung cancer, such as tobacco smoking, socioeconomic status, and occupational variables, and the estimates of relative risk and the confidence intervals were not appreciably different from the results presented.

Power calculation

Assuming that 15% of the population smokes cannabis, a sample size of 75 cases and 300 controls had 80% and 90% power at a type 1 error rate of 5% to detect an odds ratio of lung cancer of 2.4 and 2.7, respectively.

RESULTS

A total of 203 eligible cases were contacted and invited for interview of whom 79 (77%) agreed to participate. There were 893 controls contacted and invited for interview of whom 124 (68%) agreed to participate.

The 79 cases of lung cancer included 51 (65%) small-cell and 41 (60%) non-small-cell cancers. This is consistent with the established distribution of histological cell type seen in the general population.[32] There were 39 (49%) cases of lung cancer, similar to the female proportion (50%) of all lung cancer cases aged <55 years in New Zealand.[33] The 124 controls had a similar age and sex distribution to the cases (Table 1). Maori and Pacific Islander ethnic groups were oversampled in the cases, in proportion to the incidence of the rates of lung cancer in these ethnic groups.[34] Maori and Pacific Islanders were oversampled in the controls in proportion to the cases (RR 0.5, 95% CI 1.4 to 4.4). There was no significant association between lung cancer risk and passive smoking, diet, occupation, income, educational level, and alcohol use after adjustment for age, cigarette smoking and cannabis smoking. There was a significant association between lung cancer risk and family history of lung cancer, after adjustment for age, cigarette smoking and cannabis smoking. The risk of lung cancer increased 8% (95% CI 2% to 15%) for each joint-year of cannabis smoking, after adjustment for age and cigarette smoking (OR 1.07 (95% CI 1.02 to 1.09), after adjustment for non-cannabis risk factors for lung cancer (Table 2).

TABLE 1

Tobacco use, cannabis use and alcohol consumption and risk of lung cancer

Characteristic	Number of cases	Number of controls	RR of 95% Confidence lung cancer	interval
Cigarette smoking				
Never	36	102	1.0	
Ever	70	102	4.7	(1.6 to 14.0)
Pack-years				
Non-smoker	9	172	1.0	
1st quartile	7	40	2.0	(0.6 to 7.7)
2nd quartile	5	41	1.3	(0.4 to 5.0)
3rd quartile	15	40	4.2	(1.4 to 12.8)
4th quartile	43	31	12.1	(4.3 to 33.7)
4th quartile	22	17	23.9	(7.9 to 65.5)
Cannabis use				
Non-smoker	36	295	1.0	
Smoker	43	29	11.2	(6.5 to 19.3)
Joint-years				
Non-smoker	36	295	1.0	
1st tertile	9	11	6.9	(2.4 to 19.5)
2nd tertile	9	15	4.9	(1.8 to 13.0)
3rd tertile	25	4	57.3	(16.5 to 198.8)
Joint-years up to reference date[a]				
Non-smoker	70	295	1.0	
1st tertile	6	14	3.0	(1.0 to 8.8)
2nd tertile	6	14	0.9	(0.3 to 2.9)
3rd tertile	8	4	5.2	(1.2 to 20.8)
Alcohol				

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[a]Adjusted for age, sex, ethnicity, family history of lung cancer, and joint-years of cannabis use.

[b]Adjusted for age, sex, ethnicity, family history of lung cancer, and pack-years of cigarette smoking.

[c]Adjusted for age, sex, ethnicity, family history of lung cancer, pack-years of cigarette smoking and joint-years of cannabis use. Alcohol consumption was measured using a semi-quantitative questionnaire.

[d]Cannabis use joint-years tertile score: 13.29, 1.99–88.5, >88.5.

When joint-years of use were fitted as a continuous variable, this provided strong positive statistical power that the assessment by tertile of use, a significant increasing risk, 8%, with each joint-year of use was found (RR 1.08, 95% CI 1.02 to 1.15) (Table 2). The strength of the association was maximized when cannabis use in the form of joints was compared with tobacco use in the form of cigarettes (RR 1.07, 95% CI 1.03 to 1.11). The risk of lung cancer increased 7% (95% CI 5% to 9%) for each joint-year of cigarette smoking, after adjustment for age and cannabis smoking (OR 1.07 (95% CI 1.05 to 1.09), after adjustment for non-cannabis risk factors for lung cancer (Table 2). There was a significant association between lung cancer risk and family history of lung cancer, after adjustment for age, cigarette smoking and cannabis smoking (Table 2).

TABLE 2

Cannabis use, tobacco use and age of onset of cannabis use as continuous variables

Characteristic	OR of lung cancer	95% CI
Pack-years of cigarette smoking[a]	1.07	(1.05 to 1.09)
Joint-years of cannabis smoking[a]	1.08	(1.02 to 1.15)
Joint-years of cigarette smoking[a]	1.07	(1.03 to 1.11)

[a]Adjusted for age, sex, ethnicity, family history of lung cancer, and joint-years and age of onset of cannabis use

[b]Adjusted for age, sex, ethnicity, family history of lung cancer, and pack-years and age of onset of cigarette smoking

[c]Adjusted for age, sex, ethnicity, family history of lung cancer, pack-years and age of onset of cannabis use

Among users of cannabis no significant correlation between joint-years of use and age of onset of use was found (Pearson correlation coefficient 0.077; P=0.36). Compared with subjects who started smoking cannabis aged 21 or older, those that started under the age of 16 had a relative risk of lung cancer of 10.5 (95% CI 0.6 to 172) after adjustment for years of cannabis use, pack-years of cigarette smoking, age, sex, ethnicity, and a family history of lung cancer.

DISCUSSION

This New Zealand case-control study provides evidence of a relationship between smoking cannabis and lung cancer in young adults. For each joint-year of cannabis smoked, the risk of lung cancer increased by 8%, after adjustment for confounding variables.

There are a number of methodological considerations relevant to the interpretation of these findings. We studied subjects aged 55 years or younger due to published case series,[17,18] of cannabis smoking and lung cancer in young adults. Cannabis use and lung cancer in young adults. Additionally, the lifetime exposure of older individuals would be expected to be much lower as they would have done the typical age for onset of use when the "epidemic" of cannabis use began in the 1960s and 1970s.[35] Cannabis use was relatively rare before the 1960s.[36] The New Zealand Cancer Registry has been in existence since 1948, and therefore substantial numbers of lung cancer diagnoses occurred before the current era of widespread use of cannabis. She may have been influenced by previous case-control studies.[16–19] Cases were identified from both the National Cancer Registry and from hospital outpatient and discharge databases to ensure case ascertainment was as complete as possible.

In the initial approach to assertion was made of the primary risk factor of interest to avoid recruitment bias with either the cases or the controls. To minimize recruitment bias, cases were asked about their cannabis smoking habits before they were told that the study was about lung cancer. Cannabis exposure was measured as a product of cannabis use, which combined the variables of amount and frequency and duration of use. This approach follows the current convention for quantifying life long cannabis consumption and recognizes the evidence that the risk of lung cancer with cigarette smoking is related to both duration and intensity of exposure.[37] Cannabis use was also measured in joint-years, which is a unit of exposure that has been used in previous studies.[38] Cannabis use was also measured in pack-years of cigarette smoking,[39] and age of onset and usage imbalance techniques.[40] Examination of potential confounders of cannabis and cigarette smoke may also be responsible for the differences in their lung cancer risks. In this regard, cannabis smoke has been shown to have greater concentrations of the carcinogenic polycyclic aromatic hydrocarbons benzo(a)anthracene and benzo(a)pyrene than cigarette smoke.[41]

The risk of lung cancer from cannabis use was estimated to be super-additive (joint-years of use, with a 17-fold greater risk in those with more than 10.5 joint-years of cannabis smoking) and additive (pack-years of cigarette smoking, with a 12-fold greater risk in those with more than 10 pack-years of cigarette smoking), after cigarette consumption and lung cancer risk.[42] Cannabis use and lung cancer risk[43] and cigarette and lung cancer risk[44] have been widely reported. The relative risks were also calculated based on cannabis use up to 5 years prior to diagnosis or reference date in the controls, on the basis that exposure after that time may not contribute to the development of lung cancer.[30] The aim of this study was to determine the risk of lung cancer associated with cannabis smoking. A multivariate logistic regression was calculated for possible non-cannabis risk factors for lung cancer, such as tobacco smoking, socioeconomic status, and occupational variables, and the estimates of relative risk and the confidence intervals were not appreciably different from the results presented.

TABLE 3

The frequency distribution of cases and controls for selected variables

Variable	Category	Cases (n=79)		Controls (n=124)		Total (n=203)	
		No.	%	No.	%	No.	%
Age group	35–39	4	5.0	9	7.0	13	6.3
	40–44	13	16.5	23	18.5	36	17.7
	45–49	34	43.0	34	27.5	68	34.5
	50–54	28	35.5	58	46.5	86	42.4
	55 years	0	0.0	0	0.0	0	0.0
Gender	Male	40	50.6	107	85.6	147	66.4
	Female	39	49.4	17	14.4	56	33.6
Ethnic group	Maori	19	22.8	23	17.7	42	20.7
	Pac Islander	10	12.7	9	7.3	19	9.4
	Other	49	59.5	296	74.9	336	80.3
Cigarette smoking	Never	9	11.4	139	112.1	148	72.9
	Ever	70	88.6	102	82.3	172	84.7
Pack-years	Non-smoker	9	11.4	172	138.7	181	89.2
	1st quartile	7	8.9	40	32.6	55	12.7
	2nd quartile	5	6.3	41	33.1	46	22.7
	3rd quartile	15	19.0	40	32.3	55	27.1
	4th quartile	43	54.5	31	25.0	74	36.5
	4th quartile	22	27.8	17	13.7	39	19.2
Cannabis use	Non-smoker	36	45.6	295	238.0	331	163.1
	Smoker	43	54.4	29	23.4	72	35.5
Joint-years[a]	Non-smoker	36	45.6	295	238.0	331	163.1
	1st tertile	9	11.4	11	8.9	20	9.9
	2nd tertile	9	11.4	15	12.1	24	11.8
	3rd tertile	25	31.6	4	3.2	29	14.3

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[a]Each joint = 1 joint of 10 tobacco cigarettes smoked per day for one year

[b]Pearson correlation coefficient 0.077; P=0.36

[c]Selected Cases: Estimated Secondary School Certificate Examination Age (Y.10), Year 12, 13 (age 17, 18) were previously Forms 4 and 5

ACKNOWLEDGMENTS

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Funding

Funding was provided by The New Zealand Ministry of Health, The Hawke's Bay Medical Research Foundation and GlaxoSmithKline (UK). Associate Professor Bruce Cox was funded by the Health Research Council of New Zealand as a Wellcome Trust International Clinical Research Fellow (grant number 075065). The funders had no role in the design, data analysis or drafting of the manuscript.

REFERENCES

1. Hoffmann D, Brunnemann D, Gori G, Wynder E. On the cancinogenicity of marijuana smoke. Recent Advances Phytochem. 1975;9:63–81.
2. Kalyani R, Robertson L, Gupta B. A comparison of the carbon monoxide and pH levels in smoke from marijuana and tobacco cigarettes. Can J Pub Health. 1982;73:396–99. [PubMed]
3. Taskila S, Ghodsee P, Hao Z, et al. Tar, CO and THC delivery from the 1st and 2nd halves of a marijuana cigarette. Pharmacol Biochem Behav. 1991;40:657–61. [PubMed]
4. Wu T, Tashkin D, Djahed B, Rose J. Pulmonary hazards of smoking marijuana as compared with tobacco. N Engl J Med. 1988;318:347–51. [PubMed]
5. Vigliani E, Karch M, Krueger E, Beasley S, Simmons M, Tashkin D. Tracheobronchial histopathology in habitual smokers of cocaine, marijuana, and/or tobacco. Chest. 1997;112:1520. [PubMed]
6. Gong H, Fligiel S, Tashkin D, Barbers R. Tracheobronchial changes in habitual, heavy smokers of marijuana with and without tobacco. Am Rev Respir Dis. 1987;136:142–49. [PubMed]
7. Hinds J, Sielicki K, Krieger S, Simmons M, Tashkin D. Histologic and morphometric alterations in bronchial epithelium in habitual smokers of marijuana, cocaine or tobacco. J Natl Cancer Inst. 1990;90:1198–206. [PubMed]
8. Busch F, Sacci M, Wilson D, et al. Lung tumors and oncogene mutations in cannabis smokers. Cancer Lett. 1979;8:319–24. [PubMed]
9. Ray E, Magnani Lapaire T, Hay N, Bonati M. Chronic inhalation of marijuana and tobacco in dogs: Recent Extension Chem Pathol Pharmacol. 1976;14:305–17. [PubMed]
10. Zhu L, Sharma A, Stolzen M, et al. Delta-9-tetrahydrocannabinol inhibits antitumor immunity by a CB2 receptor-mediated, cytokine dependent pathway. J Immunol. 2000;165:373–80. [PubMed]
11. Bilkiko M, Lacroix C, Pramil S, Gaziano F. Cannabinoids and cancer: pros and cons of an antitumour strategy. Br J Pharmacol. 2006;148:123–35. [PMC free article] [PubMed]
12. Guzman M. Cannabinoids: potential anticancer agents. Nat Rev Cancer. 2003;3:745–755. [PubMed]
13. Hall W, MacDonald J, Lynskey M. Cannabis use and psychosis: incidence, prevalence and predictors. Aust N Z J Psychiatry. 2005;39:448–53. [PubMed]
14. Sridhar K, Raub W, Weatherby N, et al. Possible role of marijuana smoking as a carcinogen in the development of lung cancer at a young age. J Psychoactive Drugs. 1994;26:285–95. [PubMed]
15. Peng M, Cohen L, Mahboubi M. Lung and esophageal cancer in young patients: Historical differences in risk factors? Surgery. 1999;85:149–52. [PubMed]
16. Sasco A, Merrill R, Dari I, et al. A case control study of lung cancer in Casablanca, Morocco. Cancer, Causes and Control. 2002;13:949–56. [PubMed]
17. Hazel M, Jackson N, Zissel D, et al. Etiologic factors in primary bronchial carcinoma in Tunisia. Ca Semin Windsor. 1995;71:265–9. [PubMed]
18. Amin N, Boffetta P, Bouhoum K, et al. Risk factors for lung cancer and cancer of somatic in Tunisia. J Thorac Oncol. 2007;2:1–7. [PubMed]
19. Hamilton M, Wiesenfeld H, Cu Y, et al. Marijuana use and the risk of lung and upper aerodigestive tract cancers: results of a population-based case-control study. Gynecol Oncol. 2001;82:467–73. [PubMed]
20. Aquino Jan, the synthetic health strategies related to cannabis and the new appropriate legal status. Report of the Health Committee, Forty-seventh Parliament Provo Chebrick, Chairperson), presented to the House of Representatives August, 2003.
21. Williams J, Gerling I, Sorensen P, Bakhit R. Evaluation of risk factors for chest drug trends in New Zealand. Auckland: Massey University; 2005.
22. Britton J. Summary of long-term adverse effects of smoking marijuana on lung health. Report from the Royal College of Physicians of London to the Tobacco Controller Inquiry into Drugs. 1998
23. Parkin D, Whelan S, Ferlay J, Raymond L, Young J. Cancer incidence in five continents: VII. Lyon: International Agency for Research on Cancer, Scientific Publications; 1997.
24. Spiro A, editor. Lung Cancer. The Clinical consequences of malignancy. 2003;6(7):1–329.
25. World Health Organization. International Statistical Classification of Diseases Mortality Database http://www3.who.int/whosis/menu.cfm?path=whosis,mort_tables,mort_tables2&language=english. Accessed 1 June 2007.
26. Park B. Incidence of and duration of smoking in cannabis using cancer cases. EMJ Sci Tech. 1986;78:23–33. [PubMed]
27. Khalsa JH, Gorsuch L, Francis M, Hubbard R. Clinical consequences of marijuana. J Clin Pharmacol. 2002;42:776–789. [PubMed]
28. Hall W, Solowij N, Lemon J. The health and psychological consequences of cannabis use. [PubMed]
29. Rieth M, Arora A, Beasley S, Klaassp S, Simmons M, Tashkin D. Airway inflammation in young marijuana and tobacco smokers. Am J Respir Crit Care Med. 1996;154:198–27. [PubMed]
30. Raby T, Hubbard S, Swan G, Christiansen S. Cognitive function of upstate samples from marijuana users. Diagnostic Cytopathol. 1991;7:229–34. [PubMed]
31. Starr K, Rotenstein M. A urologic evaluation of upstate in marijuana smokers. J Urol Pract. 1996;39:39–42. [PubMed]
32. Doll R, Hill AB. Mortality in relation to smoking: Ten years observations of British Doctors. BMJ. 1964;1:1399–410. [PMC free article] [PubMed]
33. Gaddes DM. The natural history of tumors of the bronchus: A follow-up study of five-hundred cases from 1929 to 1951. Cancer. 1979;7(1):17. [PubMed]
34. Wynder, HL, Thornton SW, Raboy KJ, et al. Early age or smoking initiation and tobacco carcinogen DNA damage in the lung. J Natl Cancer Inst. 1990;82:1004–8. [PubMed]
35. Haas T, Schaan MS, Anhalt TDS, et al. Tobacco smoker induced DNA damage and an early age of smoking initiation induce chromosome loss at 3p21 in human lung cancer. Int J Cancer. 2001;91:613–7. [PubMed]
36. National Survey on Drug Use & Health 2005. http://www.oas.samhsa.gov/2k5/DAWN/DAWN.htm#2k5Table5.1. Accessed 1 June 2007.
37. Parkes DM, Hray F, Ferlay J, Pisani P. Global cancer statistics, 2002. CA Cancer J Clin. 2005;55:74–108. [PubMed]
38. World Health Organization Cancer. 2006 cited Nov 29 [?]. http://www.who.int/mediacentre/factsheets/fs297/en. Accessed 1 June 2007.
39. Pathria DM, Hray F, Ferlay J, Pisani P. Global cancer statistics, 2002. CA Cancer J Clin. 2005;55:74–108. [PubMed]
40. World Health Organization Cancer. 2006 cited Nov 29 [?]. http://www.who.int/mediacentre/factsheets/fs297/en. Accessed 1 June 2007.
41. Hoffmann D, Brunnemann D, Gori G, Wynder E. On the cancinogenicity of marijuana smoke. Recent Advances Phytochem. 1975;9:63–81.
42. Taskila S, Ghodsee P, Hao Z, et al. Tar, CO and THC delivery from the 1st and 2nd halves of a marijuana cigarette. Pharmacol Biochem Behav. 1991;40:657–61. [PubMed]
43. Wu T, Tashkin D, Djahed B, Rose J. Pulmonary hazards of smoking marijuana as compared with tobacco. N Engl J Med. 1988;318:347–51. [PubMed]
44. Gong H, Fligiel S, Tashkin D, Barbers R. Tracheobronchial changes in habitual, heavy smokers of marijuana with and without tobacco. Am Rev Respir Dis. 1987;136:142–49. [PubMed]