SMOKING AND LUNG CANCER PREVALENCE: SLOVAKIAN CASE STUDY

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INTRODUCTION

In the first decade of this century lung cancer was an uncommon tumor. This is in sharp contrast to the late nineteenseventies and early eighties (Efron 1984).

- In 1977 the World Health Organization reported that in many countries death rates were either stationary or declining in both males and females, for cancers other than lung. The USA, Australia, Austria, Canada, Japan, Mexico, Sweden, Switzerland, and others were among the affected countries.
- In 1979 the American Cancer Society reported that the overall incidence of cancer had decreased slightly in the past 25 years and that there was an increased death rate in men, which was mainly the result of lung cancer (Figure 1).
- In 1982 the American Cancer Society reported, "Lung cancer rates are indeed the monster of cancer statistics, causing the overall cancer death rate to increase over 18 years from 157.0 to 169.0 per 100,000 persons".

Most industrialized countries have recorded similar increases of over 100% incidence in neoplasms of the lung between 1950 and 1964 (Liebow 1975). As a result of intensive epidemiological research carried out in this field during the last 20 years, it is now generally accepted that cancer of the lung is a disease of modern civilization and, in large part, preventable. The incidence of lung neoplasms correlates directly with population density, urbanization, industrialization, tobacco smoking, and even with the registration of automobiles (Hoffman and Gilliam 1954). All these facts suggest that we are facing a real epidemic of lung can-

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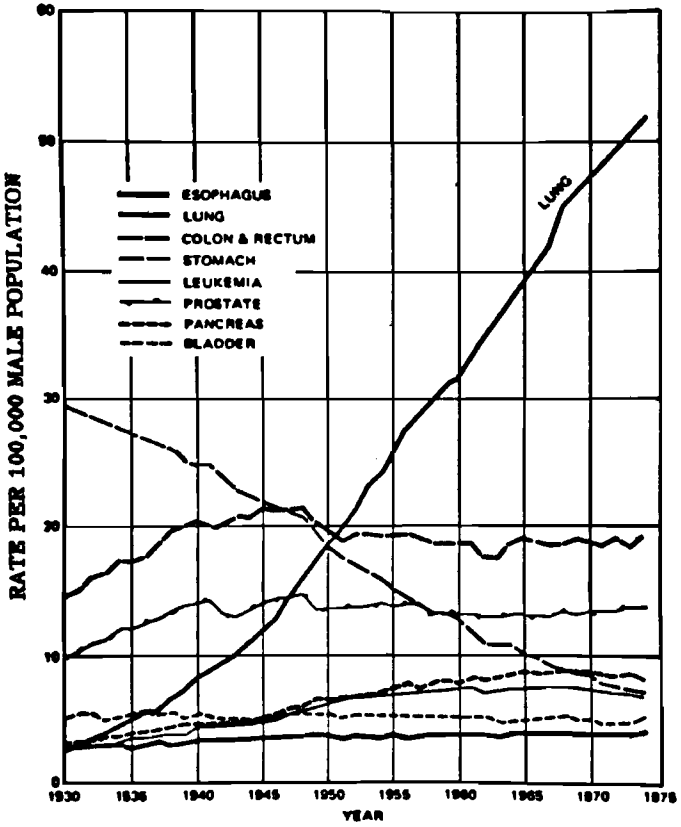
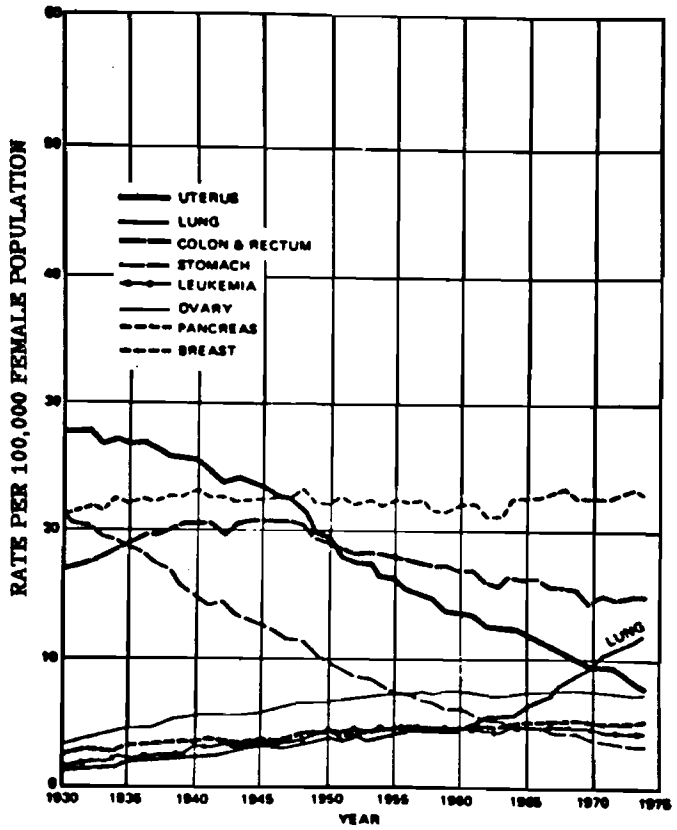
Age-adjusted cancer death rates* for selected sites, males, United States, 1930-1974.



*Standardized on the age distribution of the 1940 U.S. Census Population.

Age-adjusted cancer death rates* for selected sites, females, United States, 1930-1974.



*Standardized on the age distribution of the 1940 U.S. Census Population.

Figure 1. Cancer death rates for males and females in the USA, 1930-1974. (Sources: U.S. National Center for Health Statistics and U.S. Bureau of the Census.)

cer. The counteractions of health care systems are well known but we are interested in the future development of this process and how it could affect the population in forthcoming years. How effective could preventive campaigns be, assuming different approaches? Where to concentrate preventive efforts—in the younger or in the older part of the population? Many scientists are looking for the answers to such questions. To develop a mathematical description of processes in the population suffering from the spread of lung cancer may help answer some of these questions and forecast future development. The descriptive model, being realized on a digital computer, could be of substantial help to health care managers, specialists in epidemiology, other physicians, and even to nonphysicians with interests in this field.

MULTICAUSAL ORIGIN OF LUNG CANCER

The etiological factors in lung cancer are divided into: personal air pollutants (e.g. smoking) and nonpersonal air pollutants (e.g. atmospheric contaminants and industrial exposure). Recent new evidence suggests some other personal and nonpersonal hazards for mankind.

Tobacco smoking is encountered as the most common etiological factor in bronchogenic carcinoma. The suggestion that smoking, and in particular cigarette smoking, may be important in the production of lung cancer has been made by many writers on the subject, even though well-controlled and large-scale clinical studies are lacking. Adler (1912) was one of the first to think that tobacco might play some role in this respect. Müller (1939), from a careful but limited clinical statistical study, offered good evidence that heavy smoking is an important etiological factor. In 1941 Ochsner and De Bakey (1941) called attention to the similarity of the curve of increased sales of cigarettes to the greater prevalence of primary cancer of the lung. They emphasized the possible etiological relationship of cigarette smoking to this condition. Based on a study of 684 cases of proved lung cancer cases using special interviews (634 personal interviews, and for 33 cases the information was obtained by mailing a questionnaire), Wynder and Graham (1950) concluded that excessive smoking, and in particular cigarette smoking, over a long period is at least one important factor in the striking increase of bronchogenic carcinoma.

Recently, strong evidence for the connection between smoking and lung cancer has appeared. The lung cancer epidemiology is under extensive study all over the world. Let us have a look at data for several countries.

The 40-year incidence trends of bronchogenic carcinoma in Olmsted County, Minnesota, show that in men lung cancer has risen rapidly with each decade. During the last decade this increase was due to an increase of the rate among men over 65, rates in men under 65 appearing to have plateaued. The incidence in women increased for the first time in the decade 1965-1974. For all the cases together the five-year survivorships were 11% (Seidman et al. 1976). While in the USA both the incidence and mortality of other neoplasmas have leveled off or decreased in the last decade, the death rate for lung cancer in men has increased exponentially and is today 18 times higher than 40 years ago (Figure 1) (Seidman et al. 1976). These changes were accompanied with changes in the smoking population. Two important phenomena are discernible:

- The rate of self-reported smoking has been declining significantly.
- The predominance of males in the smoking population has been receding. This reflects that the rate of smoking among adult males has decreased persistently and significantly since 1964, while the rate of smoking among women actually rose through much of the 1960s, falling slowly in the 1970s (Warner 1983).

Mortality for lung cancer in England, 1968-1980, expressed as standardized mortality ratios, is stable in males, while in females it is rising (Figure 2) (Frey et al. 1984). There seems to be an overall decline in the number of cigarette smokers. Between 1972 and 1980 the proportion of smokers in all groups fell, but especially among professionals. The average weekly cigarette consumption in smokers in 1980 was 124 (18 per day) for men and 102 (15 per day) for women. The decrease of smoking was remarkable mainly in certain social groups, notably physicians. The decrease was accompanied with a decline in lung cancer death among medical doctors, contrasting with a significant increase in the overall population (Table 1) (Frey et al. 1984).

Also, in Japan the pattern of lung cancer has been changing rapidly. Until several years ago, the number of deaths in Japan from pulmonary tuberculosis was far higher than that from lung cancer. As recorded in 1947, the death toll due to pulmonary tuberculosis was 121,912, 159 times that for cancer cases (768). By 1972 this figure had decreased to 11,983 deaths from tuberculosis, but the number of lung cancer deaths approached 12,290 (Hirayama 1976). The data for 1977 were

Table 1. Trend in lung cancer mortality of English doctors, 1953-1965.

Mortality/Smoking habits	Trend
Lung cancer death in doctors	25% decrease
Lung cancer death in the general population	26% increase
Ex-smokers in doctors	12% increase
Filter cigarettes in England	1-6%

SOURCE: Frey et al. (1984).

8,803 for tuberculosis, compared with 17,235 for lung cancer. If that pace continues, the death rate for lung cancer is expected to equal that for stomach cancer shortly (Hirayama 1979). Per capita increase in cigarette consumption in recent years in Japan correlates with recent increases in lung cancer morbidity and mortality rates (Hirayama 1977).

Australian mortality statistics show that, in 1977, lung cancer was the most commonly reported cause of death from cancer in men. Looking at standard mortality ratios plotted over time (Figure 3) one can see an accelerating rate of mortality from lung cancer in the case of women. The situation for men is far more optimistic, with a definite slowing down in the rate at which lung cancer mortality is increasing (Rohan and Christie 1980).

Data from Czechoslovakia appear to have similar features to those from other developed countries. Based on routine cancer statistics, the incidence of lung cancer in men has generally increased, with an average yearly increase of 1.2%. A similar trend is found in male mortality, but with a smaller yearly increment. Female incidence and mortality shows lower values, with an average yearly increase of incidence of 0.7% (Plesko et al. 1985). The dependence between smoking and lung cancer incidence has also been fully proved in Czechoslovakia (Trefny 1978; Kubik 1981).

The general incidence of cancer in India is lower than in European countries or the USA. Cancers of the upper alimentary and respiratory tracts (oral cavity, pharynx, larynx, oesophagus, and lung) account for more than half of the cancers in men and about a quarter in women (Steinfeld 1985). Smoking habits are quite different in India, smoking being synergistic with tobacco chewing. Cigarette smoking is of comparatively recent origin and its effect on lung cancer prevalence is rising.

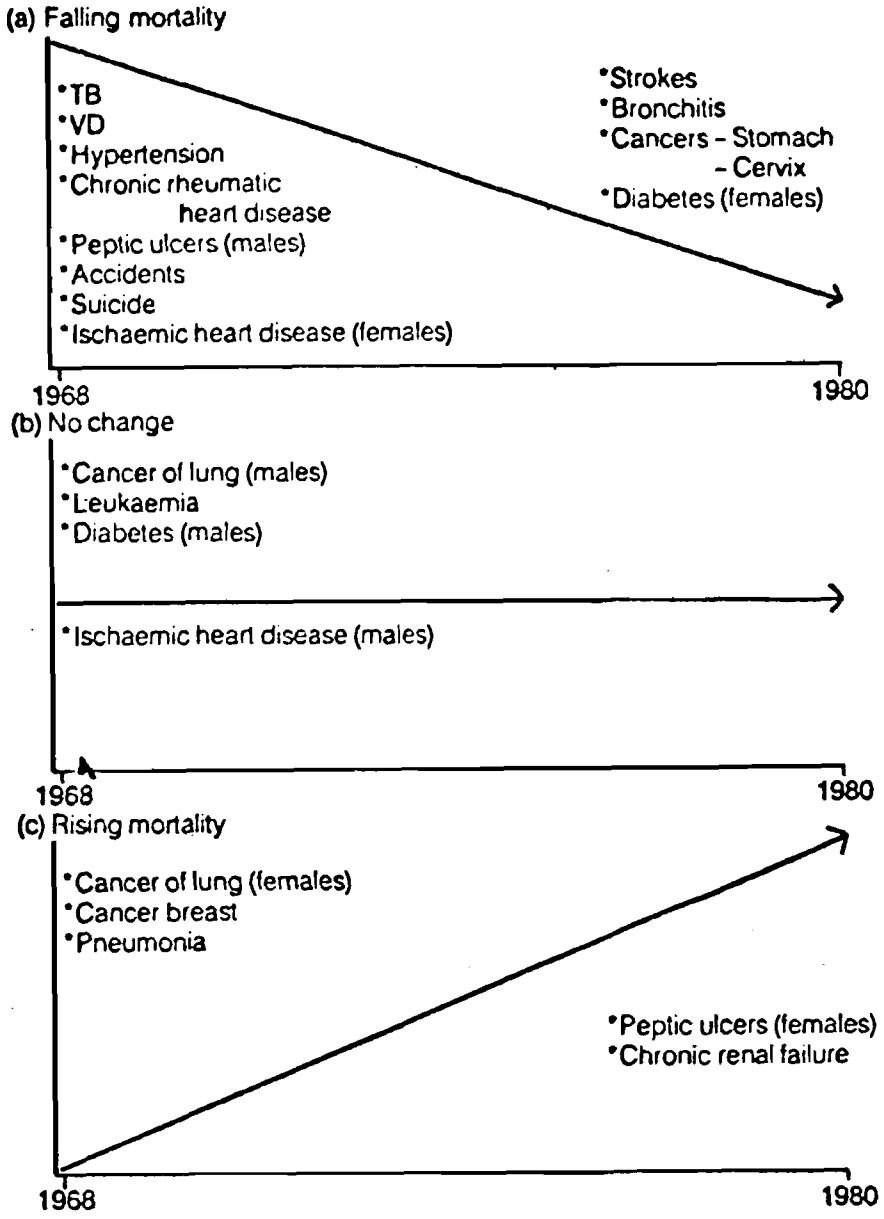


Figure 2. The mortality features of different diseases in England. [Source: Frey et al. (1984).]

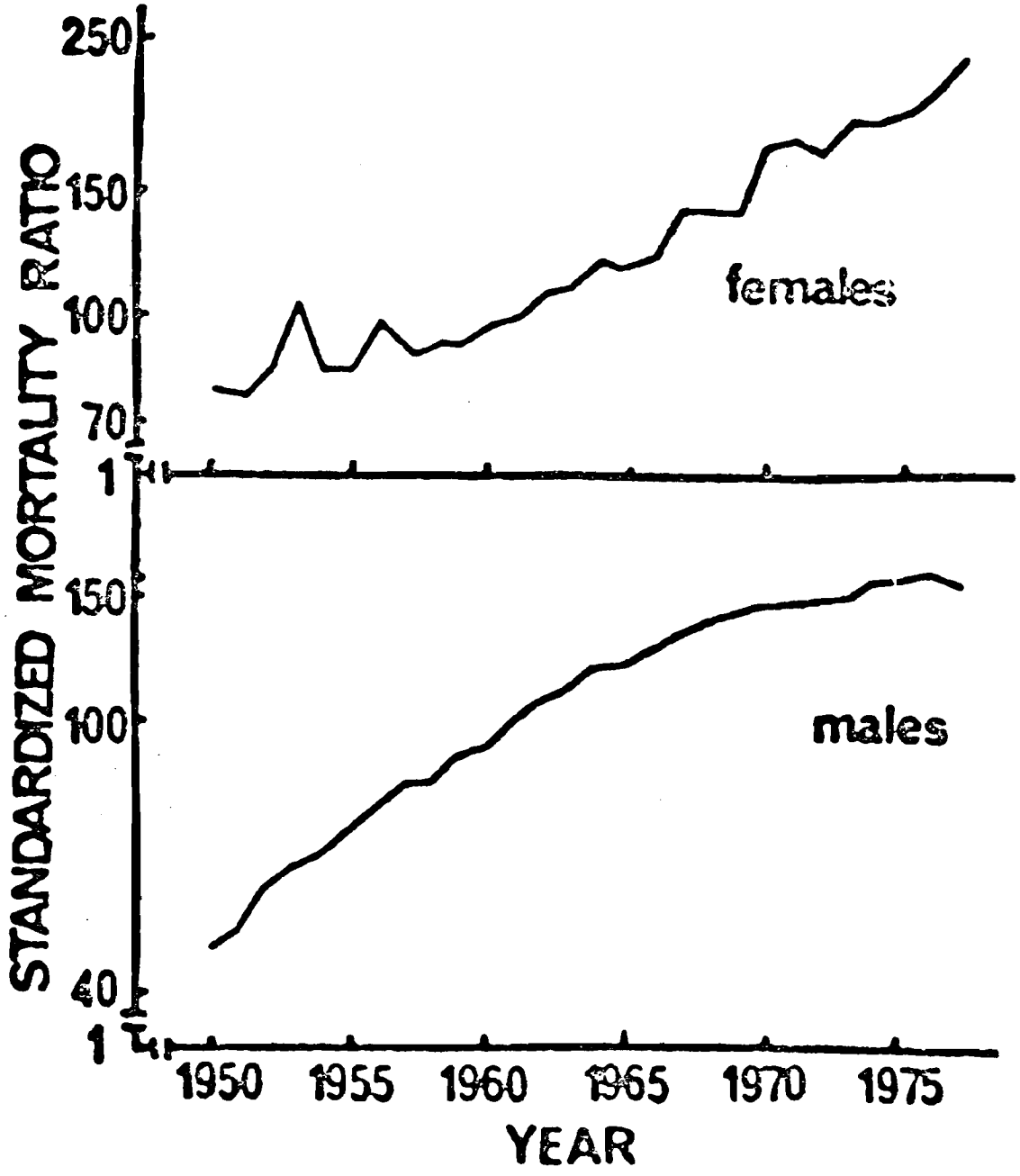


Figure 3. Standardized mortality ratios for lung cancer in Australia, 1950-1977. [Source: Rohan and Christie (1980).]

The possibility of tobacco as a major etiological factor in human cancer is now accepted by all reputable major medical and scientific organizations (Steinfeld 1985). In addition to epidemiological studies, many experimental animal studies have been undertaken, including that of Auerbach et al. (1970), who conducted long-term experiments on carcinogenesis in animals. The number of research reports on the problem is currently about 40,000 (Steinfeld 1985); it is not possible to mention all of these here! However, we have described some of them to illustrate the problem itself.

Several findings from epidemiological studies were not possible to explain as due to smoking alone. Stocks and Campbell (1955) found urban cancer mortality rates in mid-1950 England to be twice as high as rural cancer mortality rates. They attributed 50% of the lung cancer rate in Liverpool to smoking and 40% to air pollution. Similar findings were soon reported elsewhere and an exhaustive literature overview is given in Greenberg (1983). A list of chemicals with proved association to lung cancer is given in Table 2. They range from the very potent radioactive emitters to some with no activity (Beamio et al. 1975).

Table 2. Carcinogens associated with lung cancer.

X-irradiation	beuropyrem
uranium	iron oxide
cobalt	tar
chromium	coal distillates
nickel	petroleum distillates
asbestos	beryllium
molybdenum	arsenic
vanadium	bis(chloromethyl) ether

SOURCE: Ioachim (1978).

The debate on air pollution and lung cancer is still ongoing. The arguments for a relationship are as follows:

- Urban air contains substances with proven carcinogenic effects (cigarette smoke, industrial pollutants, motor vehicle exhaust gases, and construction materials).
- Urban excess of lung cancer cannot always be attributed solely to cigarette smoking and occupational exposure (Greenberg 1983).

The arguments against are that cities with the worst quality air do not necessarily have the highest lung cancer rates and that much higher lung cancer rates in men compared to women seem to be associated with their smoking habits and occupations.

There seems to be a close correlation between lung cancer risk in different occupations. Data from the Finnish Cancer Registry and Finnish National Census of December 31, 1970, reveal that the standardized incidence ratio (SIR) was highest for males in mining and quarrying (2.08). A higher than expected ratio was also observed in manufacturing (1.29). Among females the only SIR significantly different from unity was found in agriculture, forestry, and fishing (0.44) (Pukkala et al. 1983).

The fact that the prevalence of lung diseases is high in individuals with a history of alcohol abuse has led to the suggestion of a correlation between alcohol intake and lung cancer. A study of the consumption of alcohol and tobacco in relation to cancer in the USA found a positive, simple correlation between wine and spirits and lung cancer, but a negative relationship between beer consumption and lung cancer in women after controlling for cigarette consumption (Breslow and Enstrom 1974). An exhaustive discussion on the results of different studies is given in Potter and McMichael's (1984) overview. However, the situation is still not clear enough to enable quantification of the risk of lung cancer in men, due to alcohol consumption.

LUNG CANCER MODEL TARGETS

The introduction of computers to enable our understanding of cancer epidemiology has led toward the creation of National Cancer Registries in many countries all over the world. That is why reasons for the prevalence of cancer cases are much better understood than those for the prevalence of other chronic diseases. It reminds one of the history of tuberculosis between World Wars I and II and immediately after World War II. However, the future development of cancer diseases in the population is still being discussed. The association of different types of cancer with different risk factors is complex and must be solved if we are to forecast the development of cancer prevalence.

As is clear from the previous section, the main risk factor in lung cancer is undoubtedly smoking. Smoking can be understood as one of those diseases caused by the individual's own actions. Studies of the future impacts of changes in smok-

ing and smoking habits, could be of much help in establishing different anti-smoking policies. Because of the complex character of these changes and their impacts, a model could forecast what changes in lung cancer prevalence could be anticipated and how a change in prevalence will effect the health care system and society. The model might also be useful for international comparisons. The educational process could benefit in teaching postgraduate medical doctors to quantify their knowledge, as well as to interpret static epidemiological results.

DESCRIPTION OF INPUT DATA

We made use of data from the Slovak Socialist Republic, stratified according to age (18 age categories) and sex. Some coefficients were not available for Slovakia; we used data from studies in Europe and, in one case, from the USA.

Initial Lung Cancer Prevalence

Data on lung cancer prevalence for Slovakia are currently under preparation by the National Cancer Registry; we made use of incidence data to roughly estimate lung cancer prevalence.

The prevalence has been estimated according to the following formula:

$$P_{i,j}(t) = 2I_{i,j}(t-1) - \mu_{i,j}(t)P_{i,j}(t-1) \qquad (1)$$

where $P(t)$ stands for lung cancer prevalence at time t, for sex i and age category j; $I_{i,j}$ stands for lung cancer incidence for sex i and age j, and, finally, $\mu_{i,j}(t)$ is the lung cancer mortality rate for sex i and age j at time t. We did not use a more sophisticated approach because, after receiving the original prevalence data, we intend to skip the procedure of prevalence estimation. However, if there are no available data on lung cancer prevalence, the use of model DYMOD (Kitsul 1980) or a similar one is highly recommended.

We had access to lung cancer incidence data from 1971 to 1983 for the Slovak Socialist Republic according to the Slovak National Cancer Registry. The model uses only the prevalence data from year 1983 (Table 3) stratified by sex and age.

Mortality Data

The data on lung cancer mortality by sex and age come from the Slovak National Cancer Registry (Table 4, Figure 4). The data on general mortality come from the official demographic statistics yearbook of 1983.

Proportions of Smokers in the Population

The principal source of data on smokers in Czechoslovakia is Katriak (1983). Unfortunately he did not classify smokers into as many age categories as we would like to have, which is why we use his data as a basis for our expert estimation. In Table 5 we show the proportion of nonsmokers, current smokers, and quitters by sex and age. Figure 5 displays proportions smoothed by Q-spline.

The study on sociological aspects of tobaccoism (Katriak 1983) in Czechoslovakia was a source for data on transition coefficients between nonsmokers and smokers. Coefficients for transition from smokers to quitters were estimated from the data of the Hammond study of ex-smokers (Hammond and Percy 1958). The numbers roughly correlate with findings of Russell (1976) and Olejnikov et al. (1983). Table 6 and Figure 6 summarize these transition coefficients.

Risk of Lung Cancer

Because of lack of data from Czechoslovakia we use the results of a case-control interview study of lung cancer carried out in five European countries (Lubin et al. 1984). The results of these are given in the form of relative risks associated with smoking and stopping smoking compared with nonsmokers. The data of lung cancer risk for non-smokers were found in Enstrom (1979). Table 7 and Figure 7 display the risk of lung cancer in terms of the number of cases per 100,000 persons by age, sex, and smoking habits.

DERIVATION OF THE MODEL STRUCTURE

The previous two sections indicate what the possible causes of lung cancer are and what data are available. From this information the model structure was easily developed after making some preliminary assumptions.

Table 3. The lung cancer prevalence data for the Slovak Socialist Republic estimated for 1983.

	Sex	
Age	Male	Female
0-4	0	0
5-9	1	0
10-14	2	0
15-19	0	0
20-24	1	0
25-29	5	1
30-34	2	2
35-39	27	3
40-44	99	1
45-49	259	9
50-54	434	21
55-59	628	39
60-64	635	51
65-69	964	59
70-74	948	105
75-79	831	118
80-84	554	121
85+	551	197

Table 4. Lung cancer deaths in the Slovak Socialist Republic, 1983, by age and sex.

	Sex	
Age	Male	Female
0-4	0	0
5-9	0	0
10-14	0	0
15-19	0	0
20-24	2	0
25-29	0	0
30-34	2	1
35-39	14	2
40-44	36	6
45-49	77	8
50-54	165	20
55-59	288	21
60-64	255	28
65-69	212	27
70-74	279	36
75-79	189	24
80-84	72	14
85+	9	9

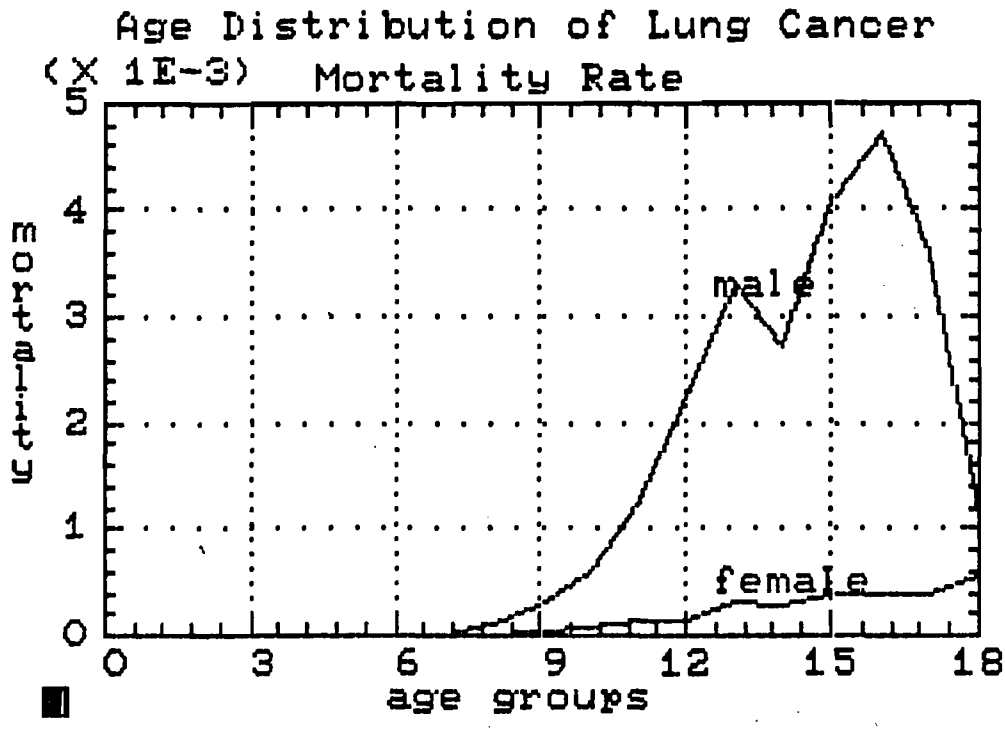


Figure 4. Lung cancer mortality rates for Slovakia, 1983.

Assumption One

Smoking cigarettes is generally recognized to be the principal cause of lung cancer, which led us to discount other, not yet definitely proved, possible etiological factors, such as air pollution and alcohol. This allows us to underline the important role anti-smoking actions have in diminishing the prevalence of this disease.

Assumption Two

According to the results of several epidemiology studies, stopping smoking leads to a decrease in the risk of developing lung cancer, a decrease that is directly related to the time elapsed since stopping. However, many people used to quit smoking several times during their adulthood, so the duration of nonsmoking is very different for each individual. It is difficult to find reliable data on this process, which is why we assume that:

- Nonsmokers never smoke during their life span.
- Smokers smoke during their whole life.
- Quitters stop and never start again.

Table 5. Population divided according to smoking habits.

	Smoking history					
	Never smoked		Current smokers		Quitters	
Age	Male	Female	Male	Female	Male	Female
0-4	1	1	0	0	0	0
5-9	1	1	0	0	0	0
10-14	1	1	0	0	0	0
15-19	0.49	0.62	0.31	0.18	0.20	0.20
20-24	0.49	0.62	0.31	0.08	0.20	0.30
25-29	0.26	0.46	0.24	0.16	0.50	0.38
30-34	0.26	0.46	0.24	0.16	0.50	0.38
35-39	0.26	0.46	0.24	0.16	0.50	0.38
40-44	0.26	0.46	0.24	0.16	0.50	0.38
45-49	0.23	0.54	0.32	0.13	0.45	0.33
50-54	0.23	0.54	0.32	0.13	0.45	0.33
55-59	0.23	0.54	0.32	0.13	0.45	0.33
60-64	0.23	0.54	0.32	0.13	0.45	0.33
65-69	0.37	0.81	0.39	0.08	0.32	0.11
70-74	0.37	0.81	0.39	0.08	0.32	0.11
75-79	0.37	0.81	0.39	0.08	0.32	0.11
80-84	0.37	0.81	0.39	0.08	0.32	0.11
85+	0.37	0.81	0.39	0.08	0.32	0.11

Assumption Three

There are plenty of differences in smoking habits: cigarette versus other types of smoking; low, medium and high tar cigarettes; depth of inhalation; etc. The assumption that there is no difference between smoking habits has been adopted in this model.

Based on these assumptions, the model structure (Figure 8) can be derived. It consists of two main blocks:

- Population forecast.
- Lung cancer prevalence forecast.

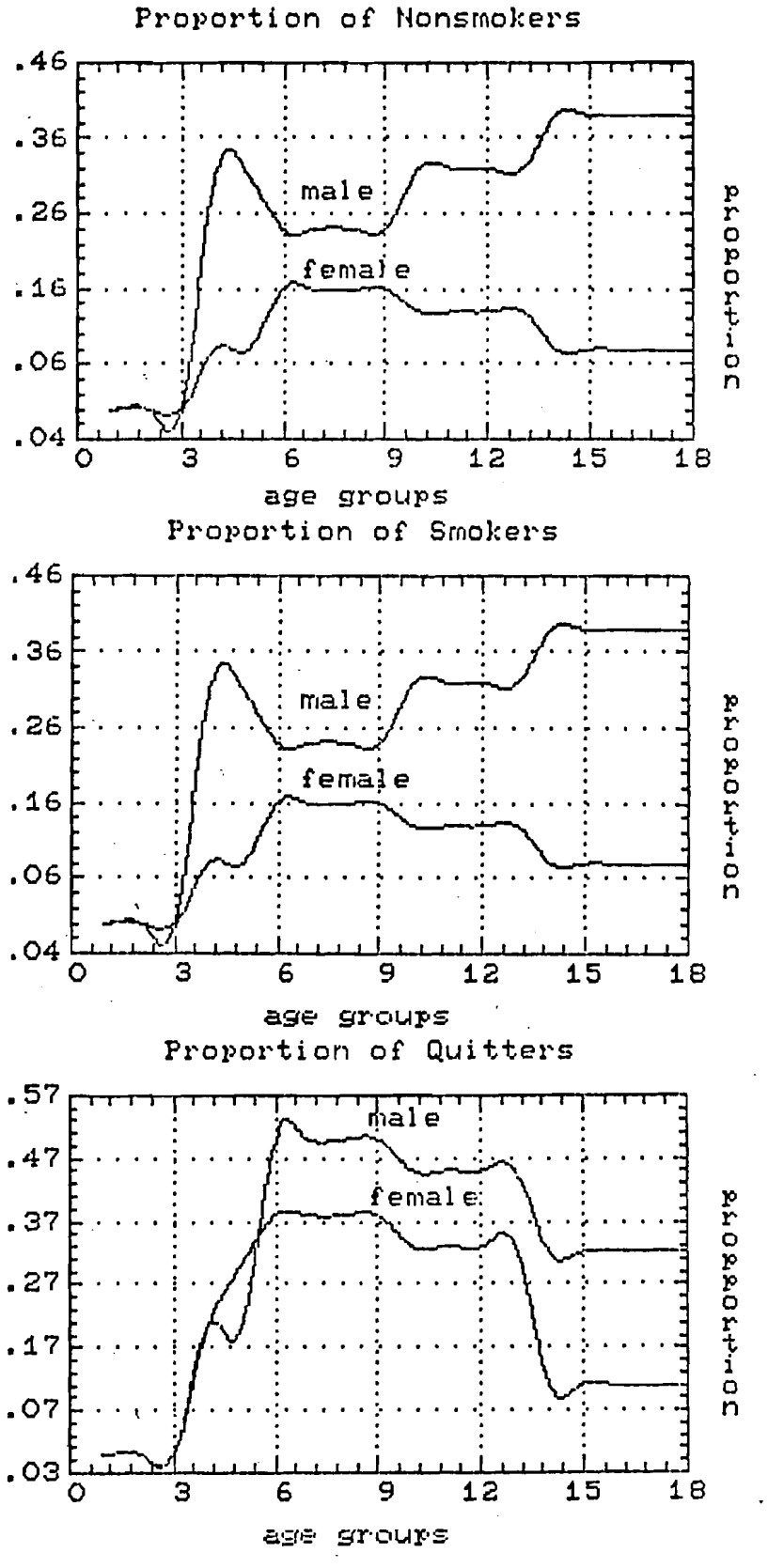
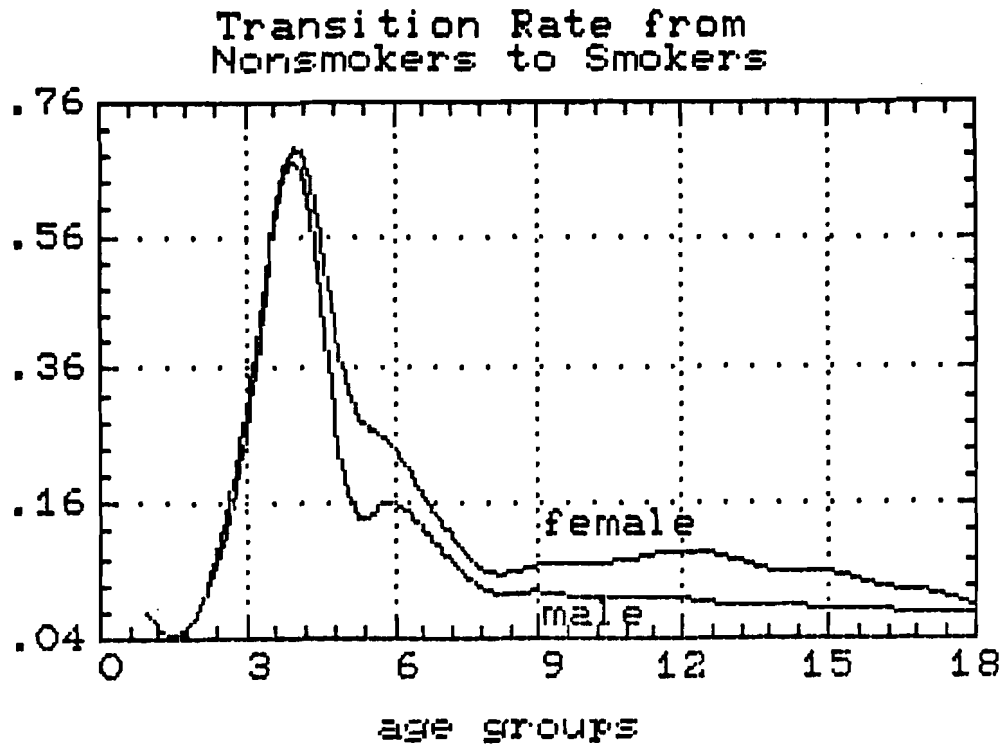


Figure 5. **Population divided according to smoking habits, curves smoothed by Q-splines.**

Table 6. Coefficient of transitions from nonsmokers to smokers and from smokers to quitters.

		Age groups								
Risk groups	Sex	0-4	5-9	10-14	15-19	20-24	25-29	30-34	35-39	40-44
Smokers	Male	0	0	0.29	0.69	0.35	0.24	0.13	0.06	0.07
	Female	0	0	0.26	0.67	0.20	0.16	0.09	0.03	0.03
Quitters	Male	0	0	0.11	0.11	0.11	0.11	0.14	0.14	0.19
	Female	0	0	0.11	0.11	20.11	0.11	0.14	0.14	0.19

		Age groups								
Risk groups	Sex	45-49	50-54	55-59	60-64	65-69	70-74	75-79	80-84	85+
Smokers	Male	0.07	0.06	0.09	0.06	0.06	0.06	0.04	0.03	0.01
	Female	0.02	0.02	0.02	0.01	0.01	0.005	0.003	0.0001	0
Quitters	Male	0.19	0.26	0.26	0.29	0.29	0.40	0.40	0.40	0.40
	Female	0.19	0.26	0.26	0.29	0.29	0.40	0.40	0.40	0.40



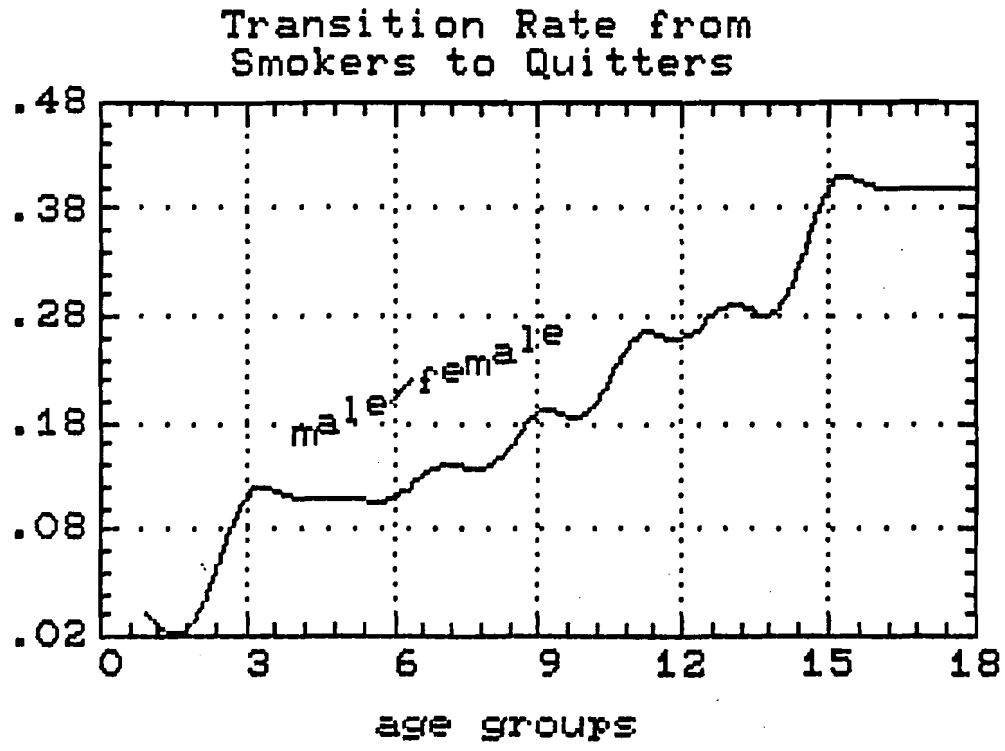


Figure 6. **Transition rates between smoking categories by sex and age.**

Table 7. Risk of lung cancer according to smoking habits (cases/100,000 persons).

		Age groups								
Smoking habit	**Sex**	**0-4**	**5-9**	**10-14**	**15-19**	**20-24**	**25-29**	**30-34**	**35-39**	**40-44**
Never smoked	**Male**	0	0	0	0	0	0	2.3	2.3	2.3
	Female	0	0	0	0	0	0	0.5	0.5	0.5
Smokers	**Male**	0	0	0	7.6	7.6	7.6	7.6	7.6	7.6
	Female	0	0	0	0.85	0.85	0.85	0.85	0.85	0.85
Quitters	**Male**	0	0	0	1.38	1.38	1.38	1.38	1.38	1.38
	Female	0	0	0	0.3	0.3	0.3	0.3	0.3	0.3
		Age groups								
Smoking habit	**Sex**	**45-49**	**50-54**	**55-59**	**60-64**	**65-69**	**70-74**	**75-79**	**80-84**	**85+**
Never smoked	**Male**	3.5	3.5	32.2	32.2	65.6	65.6	89.9	89.9	89.9
	Female	2.7	2.7	11.4	11.4	19.6	19.6	38.8	38.8	38.8
Smokers	**Male**	25.5	25.5	299.5	299.5	656	656	899	899	899
	Female	9.45	9.45	51.3	51.3	170.5	170.5	337.6	337.6	337.6
Quitters	**Male**	5.25	5.25	86.9	86.9	203.4	203.4	278.7	278.7	278.7
	Female	4.6	4.6	20.5	20.5	56.8	56.8	112.5	112.5	112.5

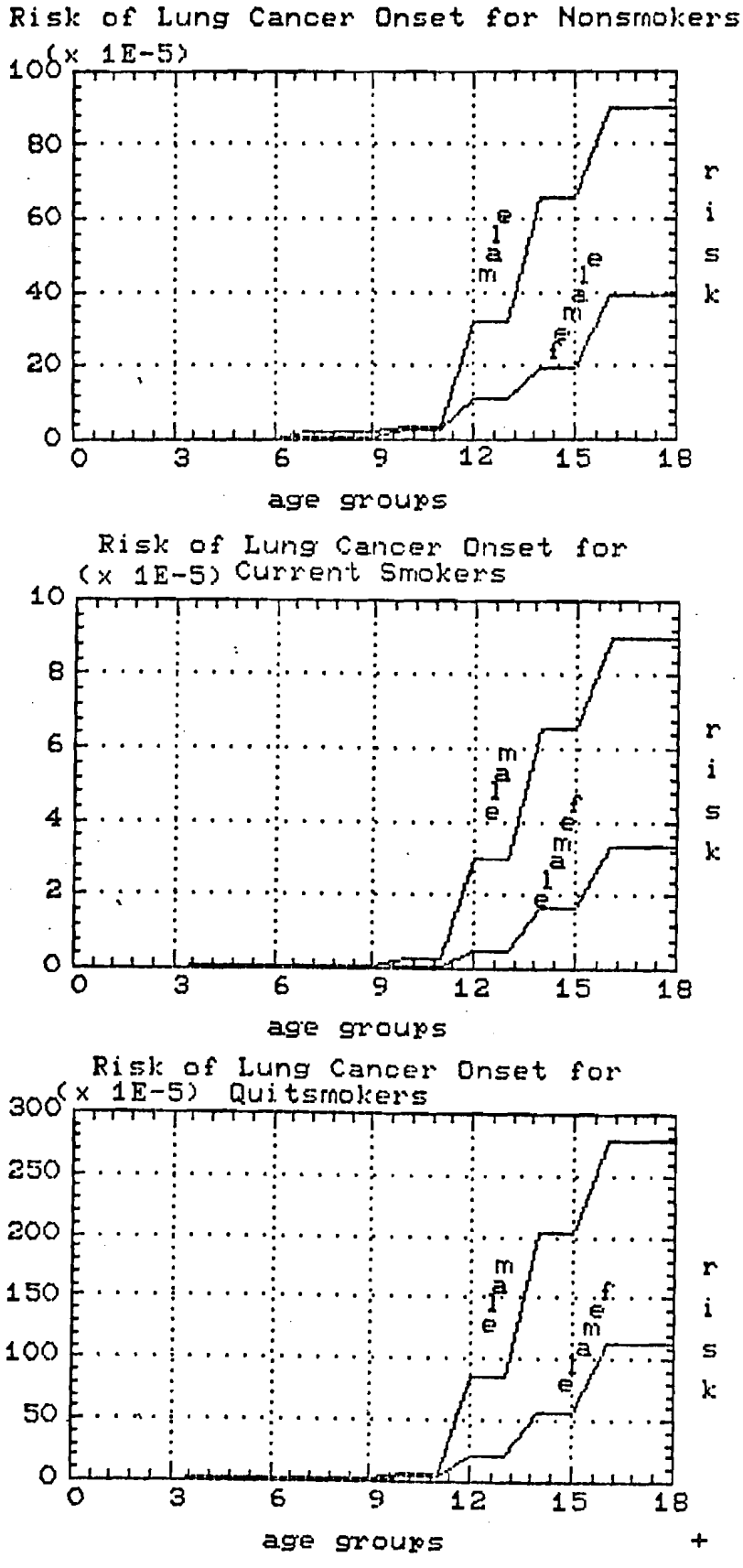


Figure 7. **Risk of lung cancer onset by smoking habits, sex, and age.**

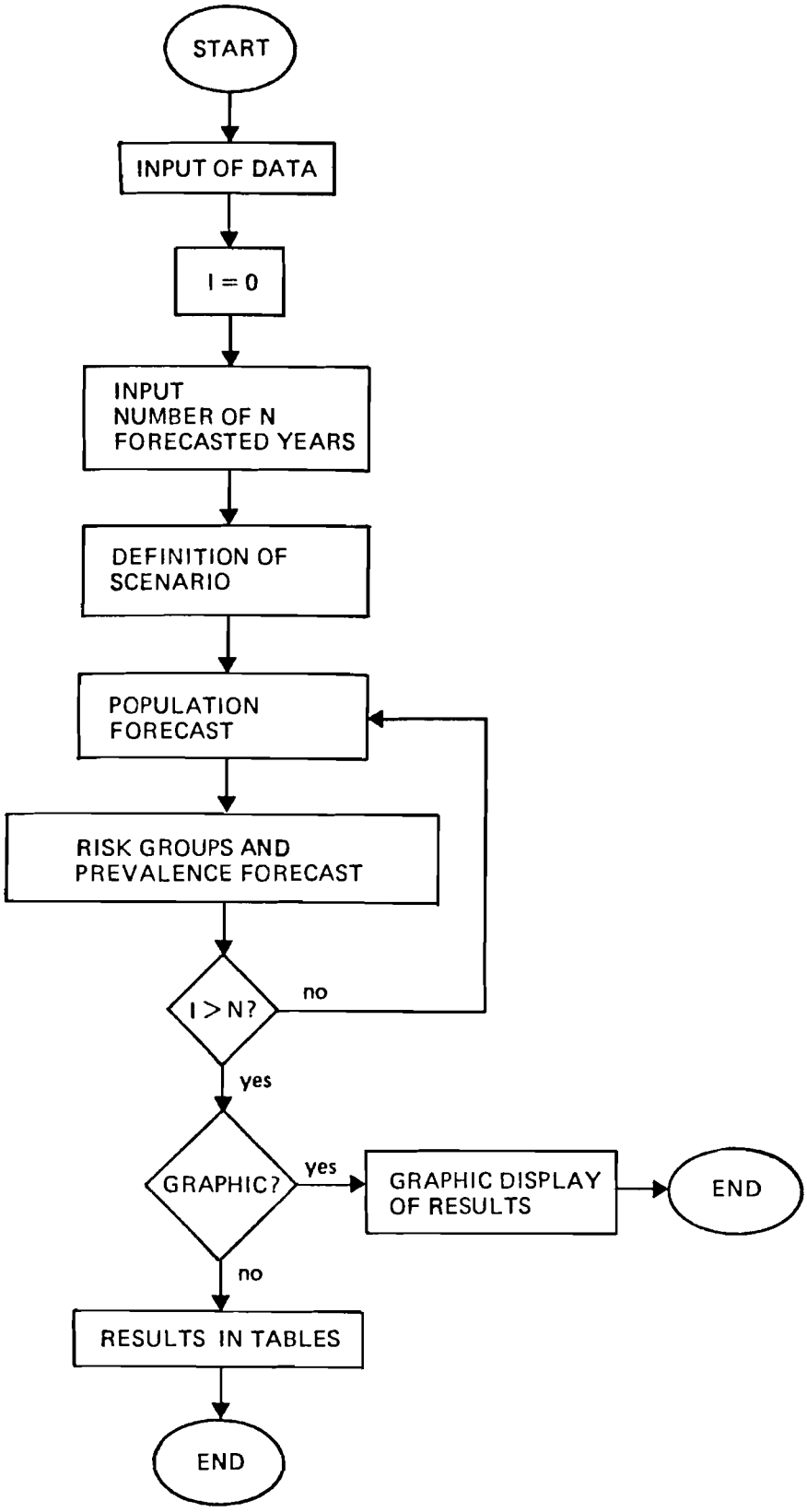


Figure 8. The lung cancer model realization in computer program.

The first block is the same as the one we used in our previous study on smoking and chronic obstructive pulmonary disease (COPD) (Rusnak et al. 1985). The second computes the amount of people in different smoking statuses, by sex and age. As far as we know how many people in each risk category are becoming ill with lung cancer, the prevalence can be estimated. By changing the model's parameters, the user can test different hypotheses about smoking distribution over population, or about the effects of anti-smoking campaigns on lung cancer prevalence.

Mathematical Model Description

The population forecast is based on a simplified idea of population dynamics. Denoting $p_{i,j}(t)$ for population at time t, sex i, and age group j, the equation used is:

$$p_{i,j}(t) = p_{i,j}(t-1) + a - 0.2p_{i,j}(t-1) - \mu_{i,j}p_{i,j}(t-1) \qquad (2)$$

$$i = 1,2 \;\; ; \;\; j = 1,\ldots,18$$

if $j = 1$, $a = b$ (b – number of births)

$j > 1$, $a = 0.2p_{i,j-1}(t-1)$

$j = 18$, $p_{i,j}(t-1) = 0$.

The death rate is the total death rate for the population, denoted μ, the mortality rate for nonlung cancer cases is $\tilde{\mu}$, and lung cancer mortality rate is $\bar{\mu}$. One can write

$$\mu = \tilde{\mu} + \bar{\mu} \;\; .$$

In order to describe the dynamics of the populations at different risk, we have to introduce some more variables:

$n_{i,j}(t)$ = number of nonsmokers with sex i, age j, at time t.

$s_{i,j}(t)$ = number of smokers with sex i, age j, at time t.

$q_{i,j}(t)$ = number of quitters with sex i, age j, at time t.

Coefficient $\rho^1_{i,j}$ describes the risk of lung cancer onset for nonsmokers with sex i, age j (per 100,000 persons). Coefficients $\rho^2_{i,j}$ and $\rho^3_{i,j}$ stand for the same type of risk, but for smokers and quitters, respectively. Transitions between groups are marked by the coefficients τ:

$\tau^1_{i,j}$ for transition from nonsmokers to smokers

$\tau^2_{i,j}$ for transition from smokers to quitters.

One can derive the following equations for the forecast of lung cancer development in nonsmokers, smokers, and quitters:

$$n_{i,j}(t) = n_{i,j}(t-1) + a - (\tau^1_{i,j} + c + \rho^1_{i,j} + \tilde{\mu}_{i,j})n_{i,j}(t-1) \qquad (3)$$

$$i = 1,2 \quad , \quad j = 1,\ldots,18$$

if $j = 1$, $a = b$ (b – number of births)

$j > 1$, $a = 0.2n_{i,j-1}(t-1)$

$j < 18$, $c = 0.2$

j , $c = 18$ $c = 0$

$$s_{i,j}(t) = s_{i,j}(t-1) + a + u^1_{i,j}n_{i,j}(t) - (\rho^2_{i,j} + \tilde{\mu}_{i,j} + c)s_{i,j}(t-1) \qquad (4)$$

$$i = 1,2 \quad , \quad j = 1,\ldots,18$$

if $j = 1$, $a = 0$

$j > 1$, $a = 0.2s_{i,j-1}(t-1)$

$j < 18$, $c = 0.2$

$j = 18$, $c = 0$

$$q_{i,j}(t) = q_{i,j}(t-1) + a + \tau^2_{i,j}s_{i,j}(t) - (\rho^3_{i,j} + \tilde{\mu}_{i,j} + c)q_{i,j}(t-1) \qquad (5)$$

$$i = 1,2 \quad , \quad j = 1,\ldots,18$$

if $j = 1$, $a = 0$

$j > 1$, $a = 0.2q_{i,j}(t-1)$

$j < 18$, $c = 0.2$

$j = 18$, $c = 0$.

The dynamics of lung cancer prevalence, $l_{i,j}(t)$, can be expressed as:

$$l_{i,j}(t) = l_{i,j}(t-1) + \rho^1_{i,j}n_{i,j}(t-1) + \rho^2_{i,j}s_{i,j}(t-1) + \rho^3_{i,j}q_{i,j}(t-1) \qquad (6)$$

$$+ a - (\tilde{\mu}_{i,j} - \bar{\mu}_{i,j} + c)l_{i,j}(t-1) \quad , \quad i = 1,2 \quad , \quad j = 1,\ldots,18$$

if $j = 1$, $a = 0$

$j > 1$, $a = 0.2 l_{t,j-1}(t-1)$

$j < 18$, $c = 0.2$

$j = 18$, $c = 0$.

Computer Realization

The model was implemented on an IBM PC micro-computer and the program was written in IBM Compiler Basic. The user can define the range of forecast; maximum is 40 years.

The model allows the user to test several hypotheses, with the quantification of each hypothesis being expressed by the coefficient change (in percent). Communication between the user and the model is done interactively, with only graphic routines invoked separately using the STATGRAPH program system on an IBM PC XT or NEWPLOT graphic system on the VAX.

The model communicates with other programs and systems. The results of model runs can be stored in database system dBase III in the database LCA. It allows the user to retrieve necessary information in the interactive way. A detailed description of the LCA database exploitation is to be found in Joestl-Segalla et al. (1986). Data for graphic routines as STATGRAPH and NEWPLOT may be retrieved from database LCA. Files used for input into the model and for communication purposes are listed in Table 8.

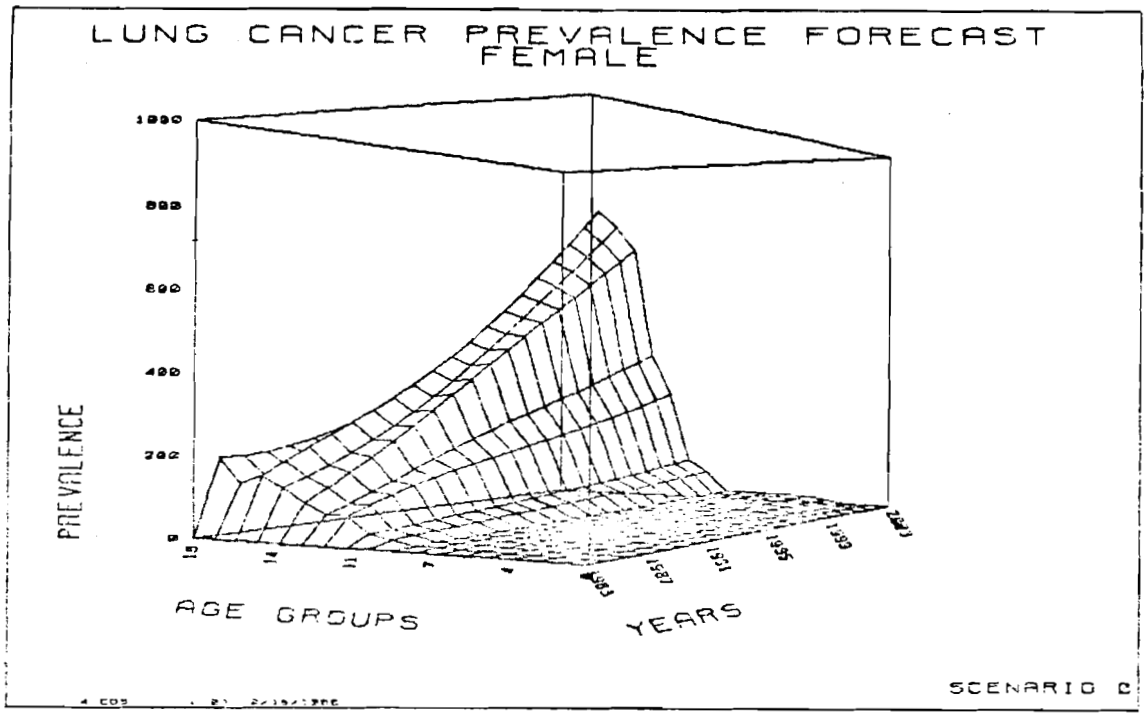
RESULTS

As was already stated, the main target of this model is to project future development in lung cancer prevalence. For this purpose the projection of risk factors was also done.

Basic projection on data from Slovakia with no scenario are shown in Figure 9. The steady increase in the number of cases is more significant in the female population, as compared to the males. The growth of the number of female cases will

Table 8. Files used by the lung cancer model.

File name	Structure	Description	File name	Structure	Description
POPFORC.SSR	SEX 1,2 AGE 1,18 TIME 1,20	population forecast for Slovakia	____.TAB	table	results of model run in a form of a table
CADEATHR.SSR	SEX 1,2 AGE 1,18	lung cancer death rates and general mortality rates	____.BSF	see dBase III database LCA	file with model's results to be entered into database LCA
SICKRISK.SSR	TYP 1,2,3 SEX 1,2 AGE 1,18	TYP 1 nonsmokers TYP 2 smokers TYP 3 quitters risk of lung cancer onset according to smoking habits	____.KOM	see Keyfitz's model	communication file for Keyfitz's model
			____.SUM	sums	sums of the model's results
CATRANS.SSR	TYP 1,2 SEX 1,2 AGE 1,18	transitions TYP 1 nonsmokers-smokers TYP 2 smokers-quitters	____.LCS		output file from database LCA
CAPROP.SSR	TYP 1,2 SEX 1,2 AGE 1,18	proportions of TYP 1 smokers TYP 2 quitters	____.NWP		file with data for NEWPLOT
LUNGCASE.SSR	SEX 1,2 AGE 1,12	number of lung cancer cases			



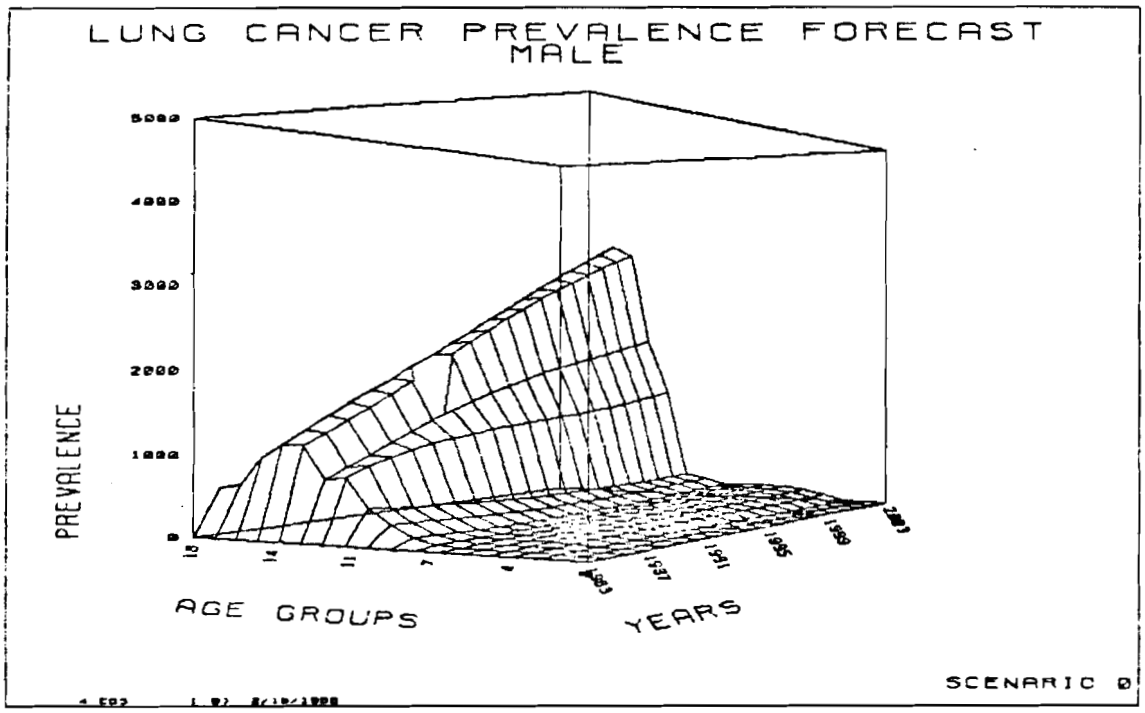
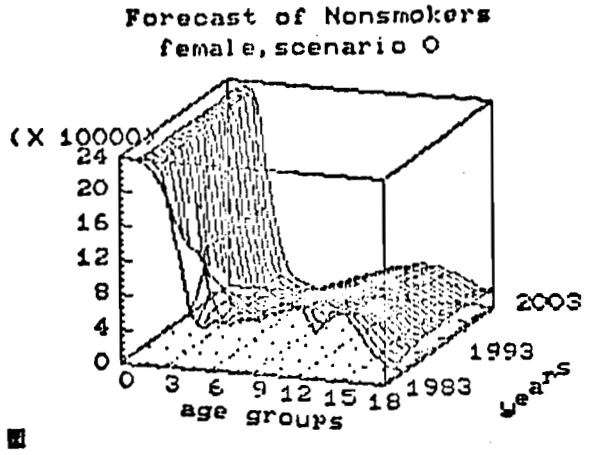


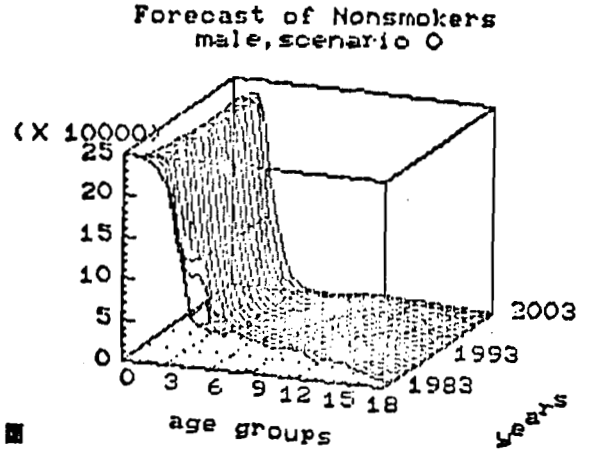
Figure 9. Lung cancer prevalence projection under basic conditions.

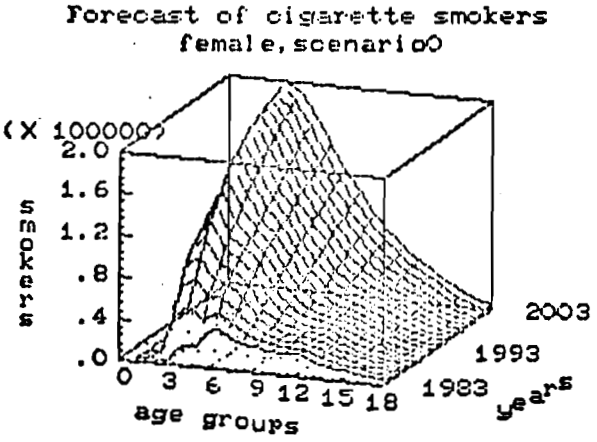
increase up to four times in some age categories, compared with two times in the same category for males. While some age categories (55-59) show only a moderate trend of increase in the male population, the same female population continues to grow at a much steeper rate. Three male age groups (50-54, 45-49, and 40-44) have the opposite tendency, while the female population keeps on growing. The reasons for these patterns are more understable if the projection of risk factors is taken into account. Projections of populations in risk are summarized in Figure 10. The steady increase in the numbers of smokers is shifted towards the older age categories, which indicates the recent trend in many countries that more and more women begin smoking and they retain this habit during a significant part of their lifespan.

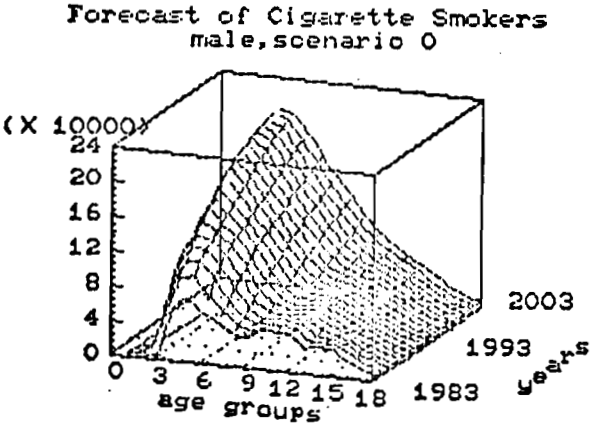
The model was run on several scenarios. We tried to highlight the impacts of preventive measures in terms of diminution of population in risk by changing transition coefficients. For example, a change in the transition between nonsmokers to smokers would imply a change in the smoking population. A similar effect is assumed after changing the transition from smokers to quitters. The other type of scenario concerns the change in risk of disease onset. It is not likely that direct influence could be done to the lung cancer onset. A majority of the screening activities are usually uneffective and too late to prevent the disease onset and as a law expensive and invasive. Nevertheless, introduction of lower tar cigarettes or more effective filters could be concerned as effective preventive measures as well as diminution of the air pollution in towns or in working places.

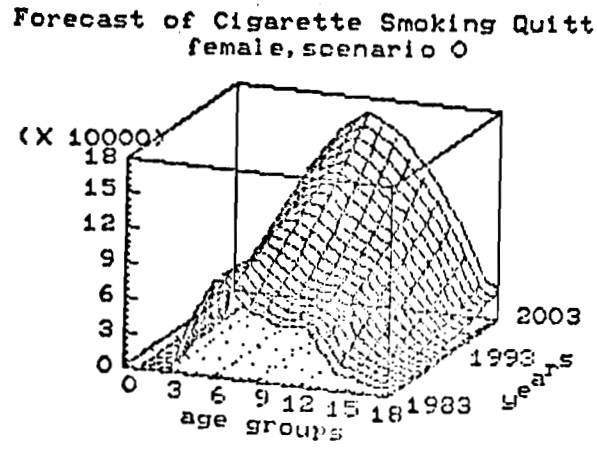
We have tested several different scenarios. The list of scenarios can be found in Table 9. The original assumption of model sensitivity to minor changes was not proven, as can be seen in Figure 11. The first two scenarios displayed (7 and 8) do not show any significant decrease in the three-dimensional graph. However, in the tabular form of results, the change is discernible. The overall tendency shows that a 20-year forecast span is too short for a more significant improvement. Can we call the prevention of up to 100 lung cancer cases insignificant? Scenario 9 shows retardation in steady increase in lung cancer prevalence, especially in the male population. Several other scenarios try to highlight the effects of more significant changes. Scenario 13 (Figure 12) assumes a reduction in the transition from nonsmokers to smokers to zero, introduced in 1986. While the general pattern from the first sight is the same compared to the basic projection, a closer view will reveal the reduction of cases in all age categories. Even this reduction was not powerful enough to completely stop the steady increase. A similar reduc-











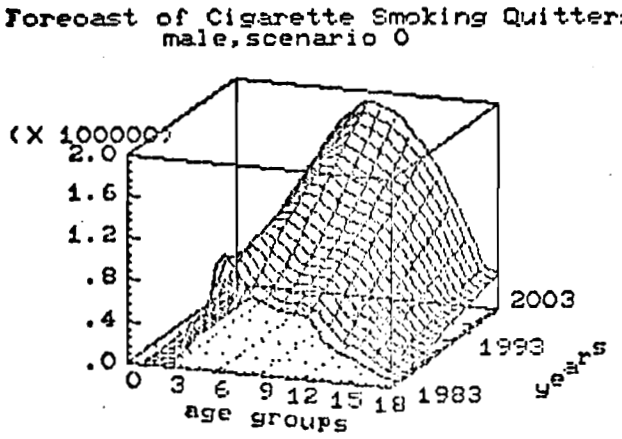
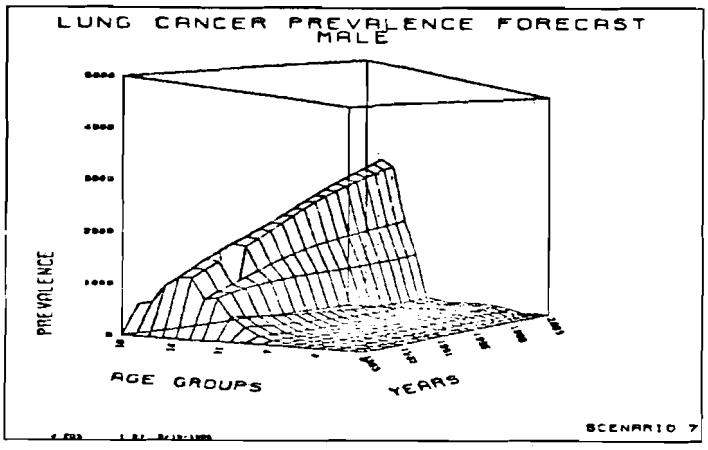
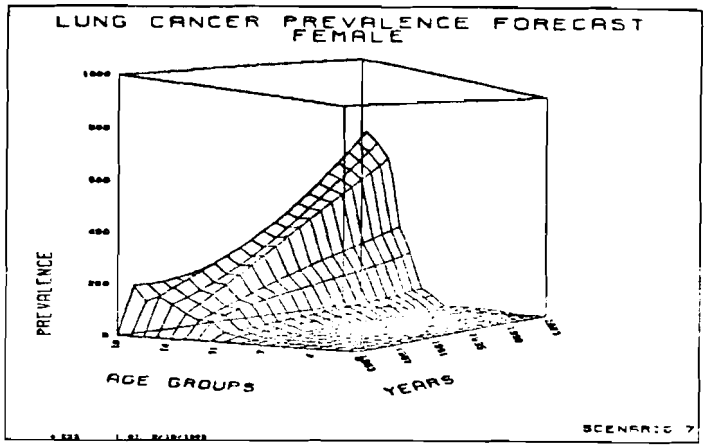


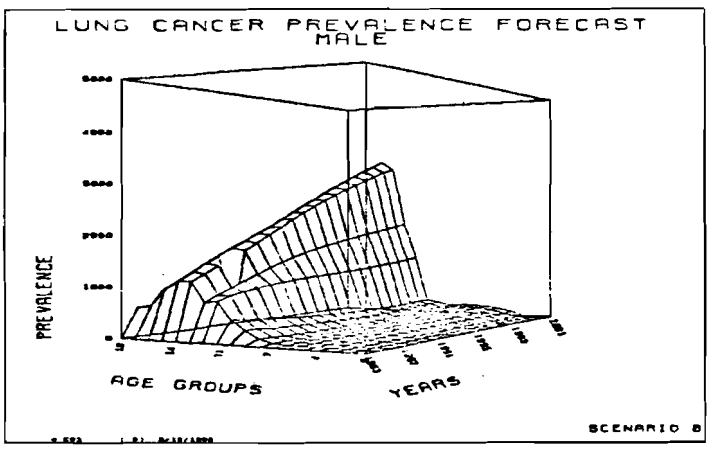
Figure 10. Projections of risk group development under basic conditions.

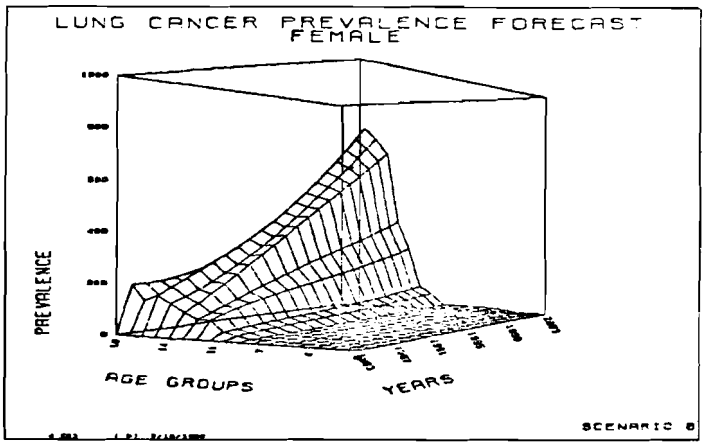
Table 9. Scenario description.

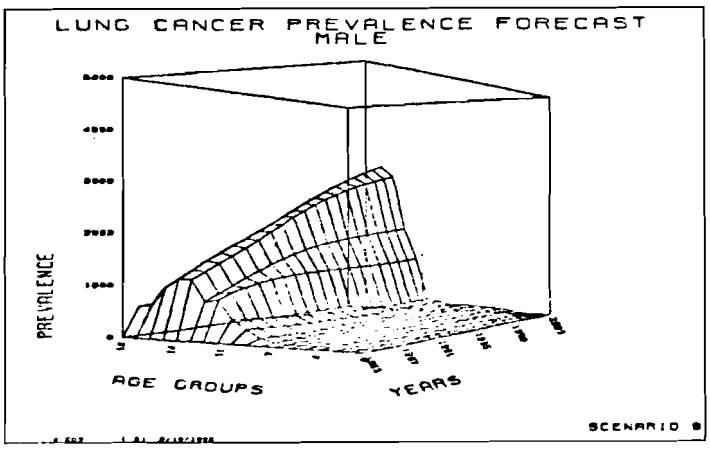
Scenario number	Changed factor	Year of change	Age category	Sex	Percentage of change	Comment
1	transition nonsmokers-smokers	1986	15-29	M/F	80	decrease in number of people who start smoking
2	transition smokers-quitters	1986	40-59	M/F	130	increase in quitters
3	transitions nonsmokers-smokers smokers-quitters	1986	15-29 40-59	M/F	80 130	combination of scenarios 1 and 2
4	risk in nonsmokers	1986	40-59	M/F	75	reduction in risk
5	risk in smokers	1986	40-59	M/F	75	risk reduction in smokers
6	risk in quitters	1986	40-59	M/F	75	risk reduction in quitters
7	transition nonsmokers-smokers risk in smokers	1986	15-29 40-59	M/F	80 130	combination of scenarios 1 and 5
8	transition smokers-quitters risk in quitters	1986	40-59 40-59	M/F	130 75	combination of scenarios 2 and 6
9	transition smokers-quitters	1986	15-59	M/F	10	decrease in smokers
10	risk in smokers	1986	20-59	M/F	50	risk reduction in smokers
11	transition smokers-quitters	1986	20-59	M/F	200	increase in quitters
12,13	transition nonsmokers-smokers	1986	All	M/F	0	decrease in smokers
14	transition nonsmokers-smokers	1986	All	M/F	50	decrease in smokers
15	transition nonsmokers-smokers	1986	All	M/F	25	decrease in smokers
16	risk in smokers and quitters	1986	All	M/F	50	risk reduction in smokers and quitters
17	risk in smokers and quitters	1986	All	M/F	25	risk reduction in smokers and quitters
18	risk in smokers and quitters	1986	All	M/F	0	risk reduction in smokers and quitters











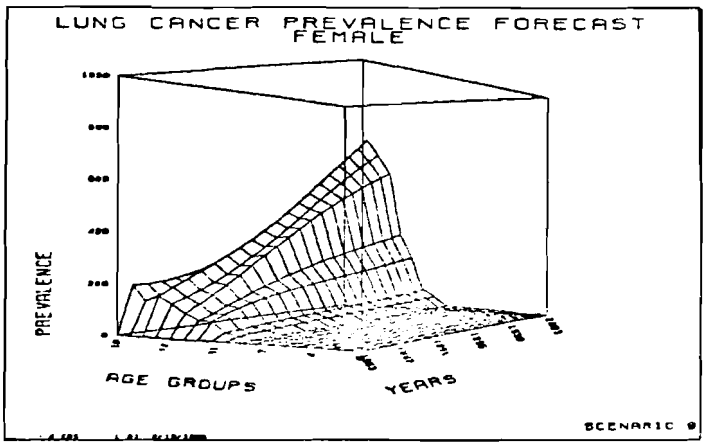
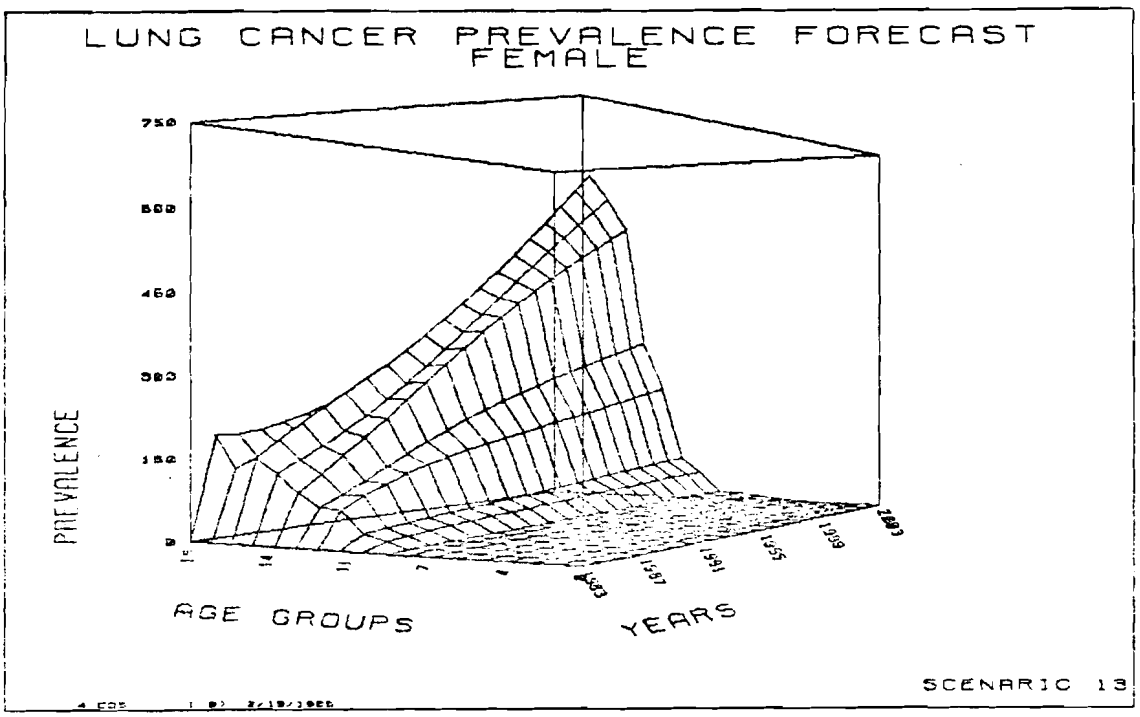


Figure 11. Comparison of several scenarios.



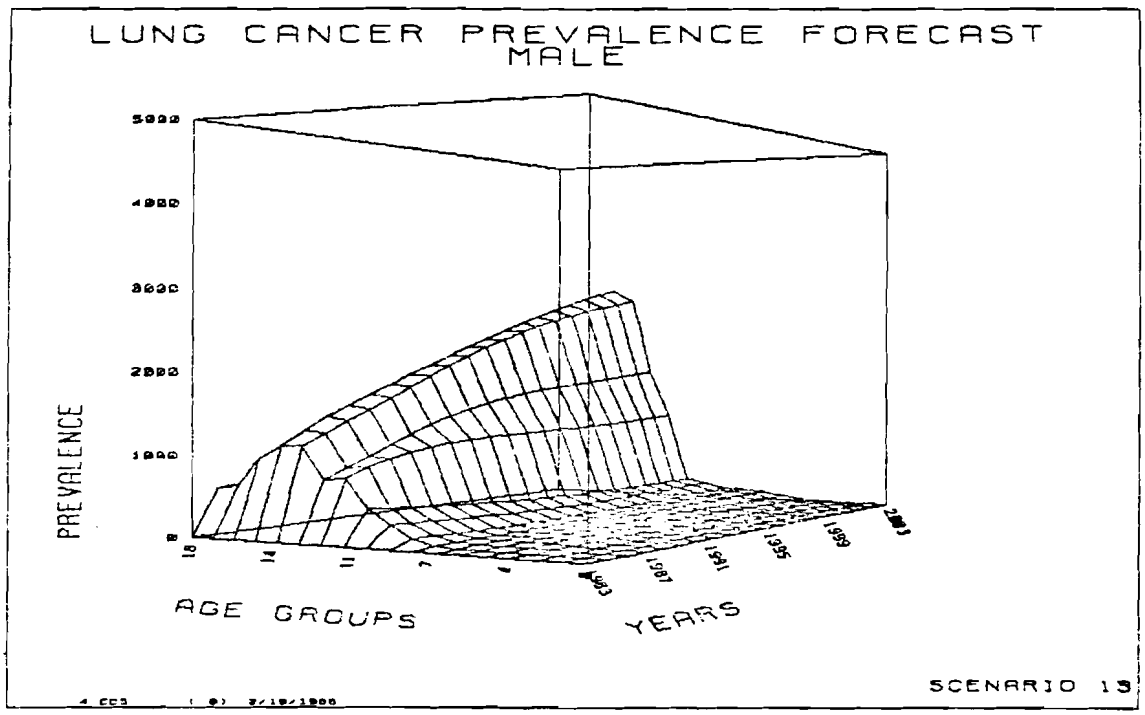
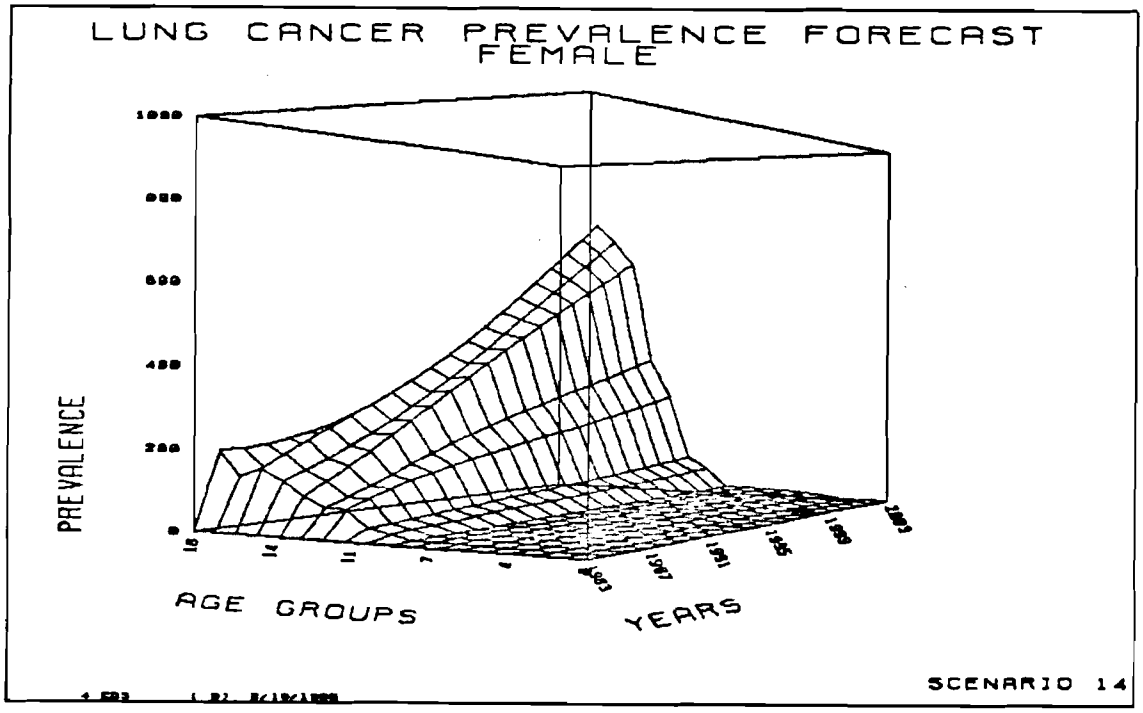


Figure 12. Results of scenario 13 – no transition from nonsmokers to smokers is assumed.

tion to 50% is shown in Figure 13 and the decrease is not as outstanding as compared to scenario 15 in Figure 14 (reduction to 25%). Scenarios 16, 17, and 18 anticipate changes in the risk of the onset of lung cancer in smokers and quitters. The introduction of a 50% change in risk will cause a dramatic change in lung cancer prevalence trends (Figure 15), as compared to scenario 14 (Figure 13) or basic projection (Figure 9). Even more optimistic projections were achieved by scenarios 17 and 18 (Figures 16 and 17).

CONCLUSION

The results of the lung cancer model confirmed the general notion on complexity of the relationship between smoking and lung cancer. The quantification of this relationship is still a task which remains to be solved. A continuous increase in lung cancer prevalence (as can be seen from the projections made upon the current situation) is similar to the one done by researchers from the Finnish Cancer Registry (Teppo et al. 1985). A similar trend can be imagined by looking at the time-series data on lung cancer all over the world. In order to change the current situation and prospective development, one might ask several questions of the type: "What will happen when ...?". The assumed change is in the form of an effective, antismoking campaign or in the more effective lung cancer prevention, even treatment. It is not rational to expect dramatic changes in smoking behavior of people in developed countries. Besides public education, several therapeutic methods are applied to cure those who wish to stop smoking. The results from both of these methods are still not encouraging enough. Lebeau in Lehrl et al. (1985) has clearly demonstrated both on the basis of personal experience and from data taken from literature on the subject, that the percentage success rate at the end of treatment, which is always of short duration, is relatively good and is approximately 60% whatever the method used, except in the case of treatment with drugs, this being the least effective method with a success rate of only 45%. The results of the first scenarios testing were not too encouraging to us. But after the time of delay between starting to smoke and the onset of disease was taken into account, the situation became clear. Now we understand what potential is hidden above the antismoking campaigns. The cessation of smoking would definitely lead toward diminution of the incidence of lung cancer, and the other chronic diseases as well. We have to be patient enough and wait for the results. Many scientists try to understand how the life expectancy of people can be increased. But the regular cigarette smoker sacrifices seven years of life for his habit and addiction. This



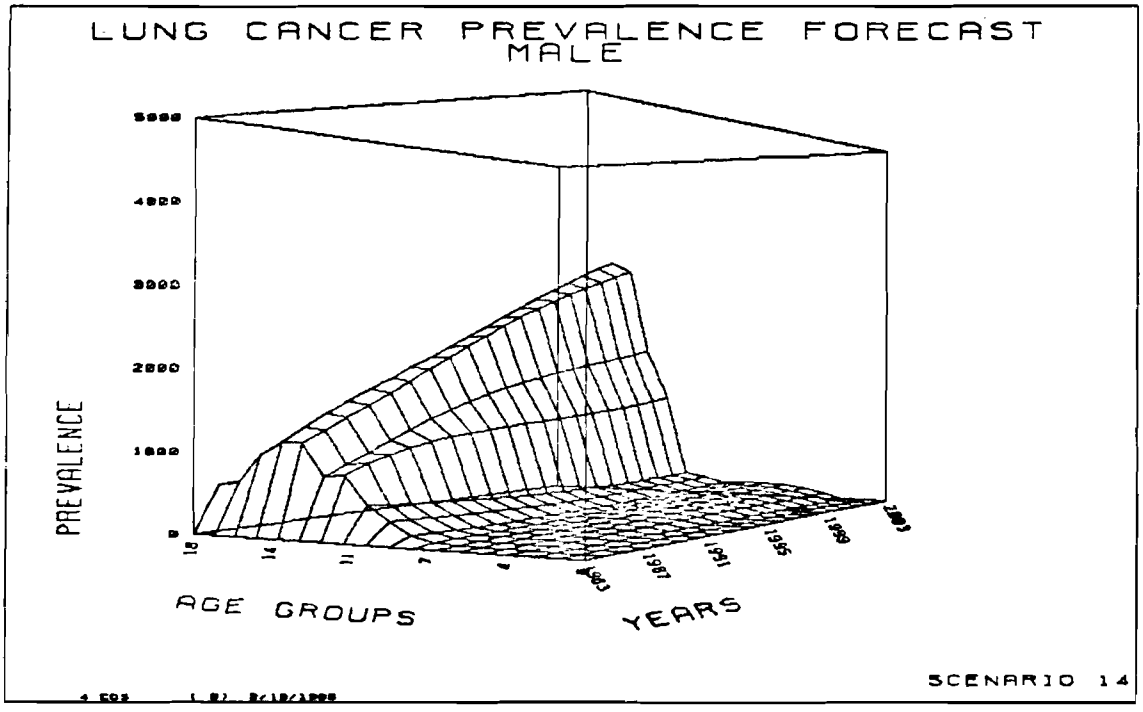
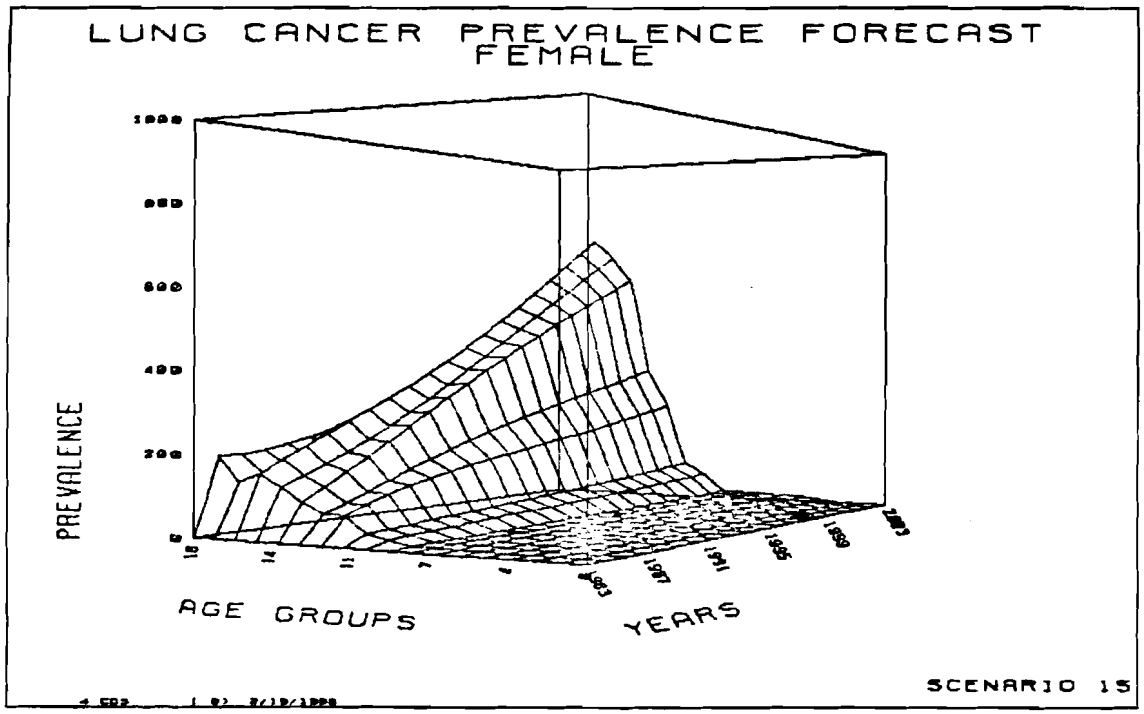


Figure 13. Results of scenario 14 — reduction in transition rates from nonsmokers to smokers to one-half of the original value for all ages.



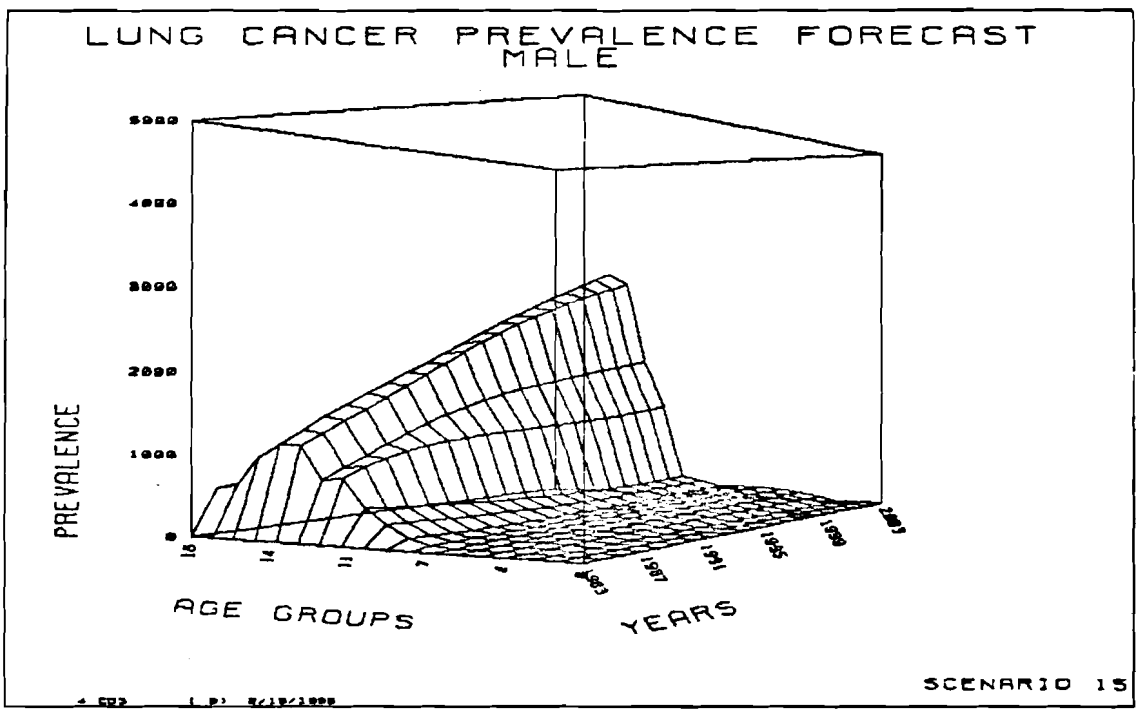
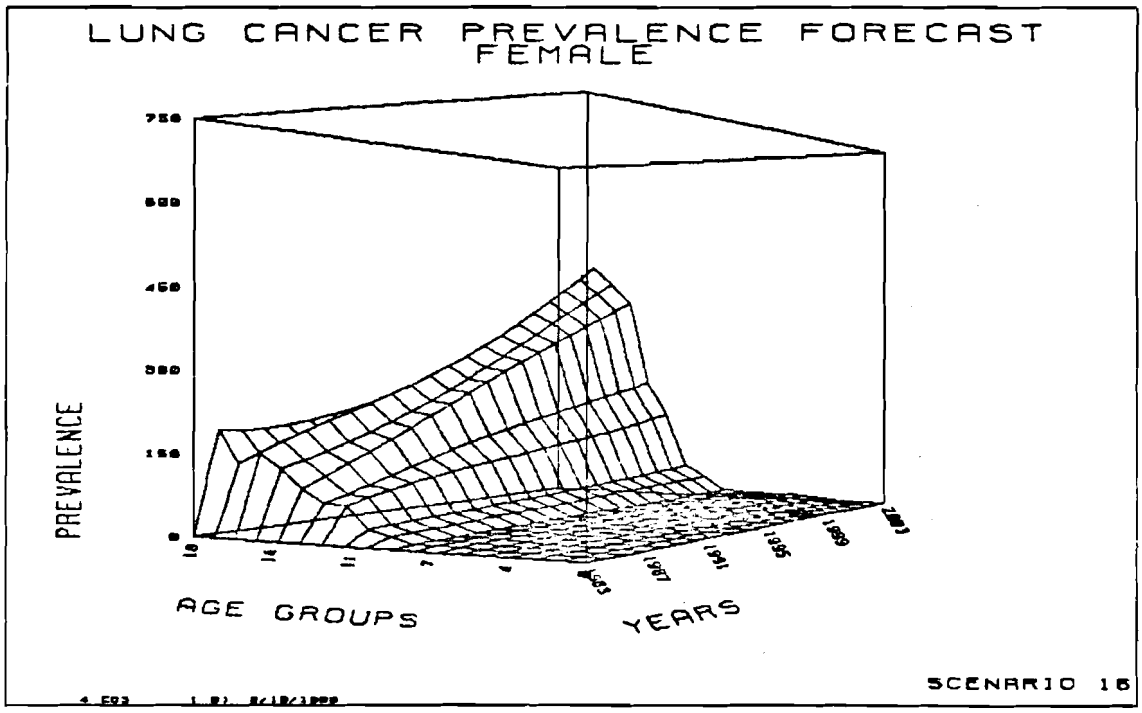


Figure 14. Results of scenario 15 — transitions from nonsmokers to smokers reduced to one-quarter of the original value for all ages.



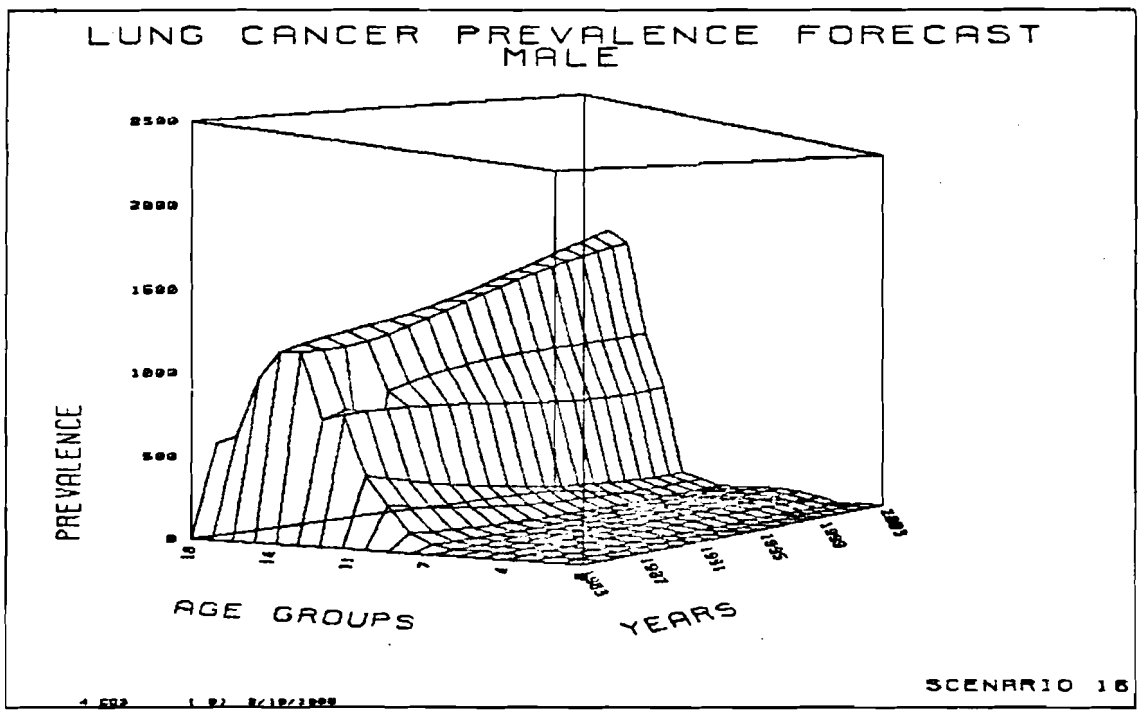
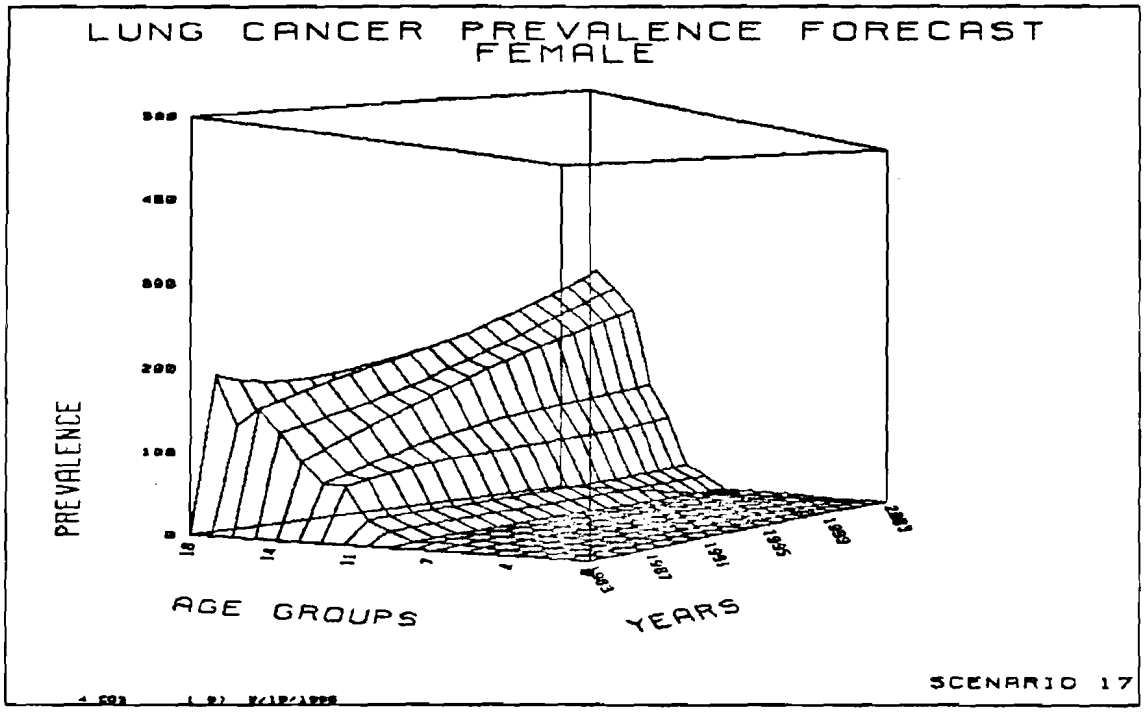


Figure 15. Results of scenario 16 — risk of lung cancer onset in smokers and quitters reduced to half of the original value for all ages



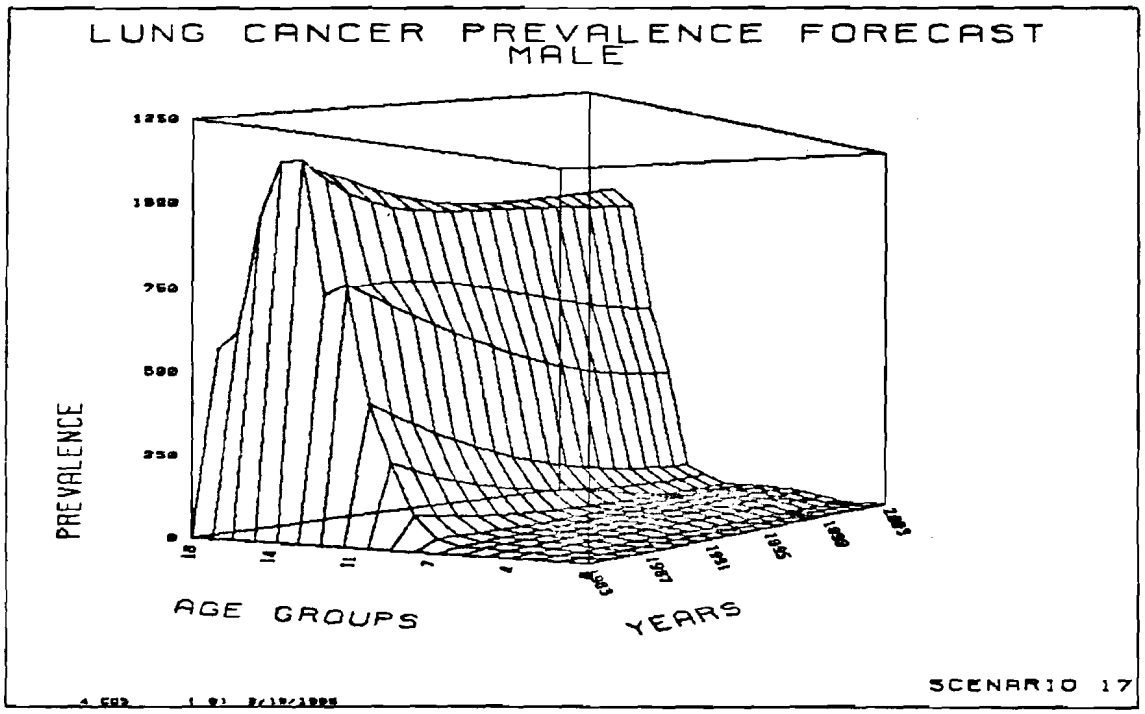
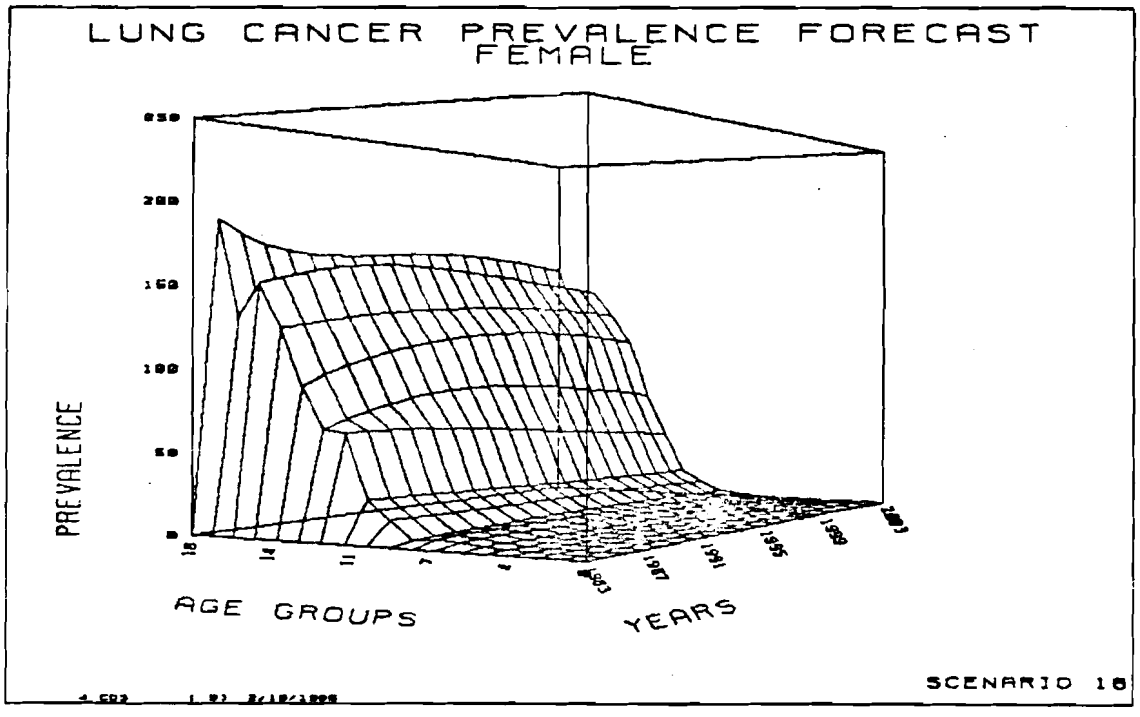


Figure 16. Results of scenario 17 — risk of lung cancer onset in smokers and quitters reduced to one quarter of the original value.



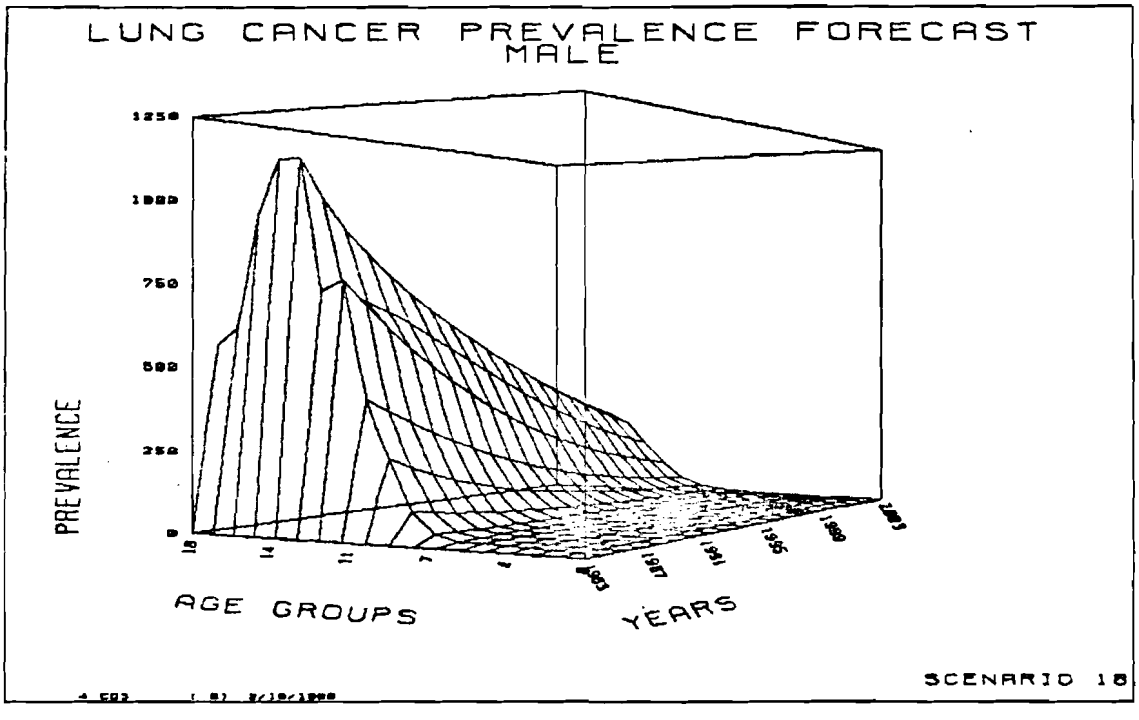


Figure 17. Results of scenario 18 – risk of lung cancer onset in smokers and quitters approaches zero in all ages.

translates into about 5.5 minutes of life for each cigarette smoked (Swann 1978).

The prevalence could easily be converted into economic terms as sick-leave days, hospital days, or other health or social-related expenditure. The increase in prevalence will be tailed by the increase in consumption of health care resources, which are usually limited. Early identification programs for lung cancer (annual chest roentgenograms and sputum cytology) effective in identifying squamous cell carcinoma at a time when early detection can improve survival (Flehinger 1984) are very expensive and mass screening is not recommended as cost-effective (Early Lung Cancer Study Group 1984). That is why smoking cessation probably seems to be the only way to stop the increase in lung cancer incidence and death. That is why Petty (1985) proclaims that every physician's office or clinic can become a smoking cessation center.

The model itself is still under development. The authors plan to use it for international comparisons within developed countries. A more detailed stratification of smoking habits is desirable as well. Such refinement will be possible only if a detailed study on smoking would be available. The possible merge of this model with the others on chronic diseases will be a worthwhile task for the future.

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