THE FUTURE OF LUNG DISEASES: COPD MODEL FOR SLOVAKIA

M. Rusnak
A. Yashin
P. Kristufek

December 1985
CP-85-49

INTERNATIONAL INSTITUTE FOR APPLIED SYSTEMS ANALYSIS
2361 Laxenburg, Austria

CONTENTS

THE FUTURE OF LUNG DISEASES: COPD MODEL FOR SLOVAKIA

M. Rusnak, A. Yashin** and P. Kristufek****

INTRODUCTION

The initial explorations of bronchial tree pathology can be traced back to the early part of the nineteenth century. Laennec was the first to demonstrate the so-called "catarrh pulmonaire" and its significance to the disease (Bajan 1983), but the attention of physicians centered upon tuberculosis and pneumonia up until the 1950s. The death of more than 4000--mainly older--people during a catastrophic four-day smog in London (1952) and the realization that chronic bronchitis and its complications were the fatal causes has proved the importance of studying this group of diseases (Protivinski 1968).

Intensive research has demonstrated the necessity for a more precise definition of the group of illnesses described under the general term chronic nonspecific lung diseases. Common efforts of specialists from all over the world have culminated in accepted definitions of chronic bronchitis, pulmonary emphysema and bronchial asthma by the World Health Organisation (WHO 1980). Recently, a common term has been used by mostly American authors for all of these diagnostic units: chronic obstructive pulmonary disease (COPD).

Numerous studies have shown an undesirable spread of COPD in the developed countries. The high and still growing prevalence of these diseases creates a burden on health-care systems, which leads to an associated growth in health care expenditures and in the number of sick-leave cases and disabled people.

It is generally understood that the causes of COPD are largely from within the society itself: life style (smoking), environmental (air) pollution, working conditions, and social and economic circumstances are believed to be responsible for

*Martin Rusnak, Research Institute for Medical Bionics, Jedlova 6, 883 08 Bratislava, Czechoslovakia
**Anatoli Yashin, IIASA, A-2361 Laxenburg, Austria
***P. Kristufek, Institute for Pneumophtisiology and Geriatrics, Podunajske Biskupice, 82556 Bratislava, Czechoslovakia

the onsets of these chronic diseases. The growth in COPD prevalence is influenced by recent demographic trends, especially population aging.

The large proportion of people with these sicknesses makes a complete registration of all the cases a practical impossibility. But COPD prevalence must be estimated in some way because of the necessity to forecaste and plan appropriate health care resources. We have developed an appropriate tool for the analysis of possible trends and describe COPD model in this paper. The authors hope that it will be of some help in answering specific questions about COPD development. The model uses data from the Slovak Socialist Republic and allows the user to test several scenarios, as described in Chapter 7.

1. COPD EPIDEMIOLOGY IN SOME COUNTRIES

Much new information on COPD and its effect on the health of the population has been revealed in numerous epidemiological studies. For example, it was shown that 17% of all British general practitioners in their fifties have symptoms of COPD, as do 37.8% of older physicians (Pride 1977); Hutli from Finland found COPD in 27% of the population surveyed (Press and Rufener-Press 1974) and the prevalence of COPD in Sweden is estimated to be less than 2% (Kirilog and Irnell 1974). Morbidity of chronic bronchitis in the US differs according to the areas surveyed and the chosen population groups: 23.5% in telephone companies' employees, 21.3% in the inhabitants of smaller industrial towns, 11% in rural areas (Mueller et al. 1971). Other American surveys have revealed that 27% of the male and 13% of the female populations have symptoms and/or spirometric abnormalities indicating COPD (Higgins and Keller 1970). Swiss data for the male population in Geneva found the occurrence of COPD to be between 2.7% in men 20-29 years old and up to 7.7% in men over 60 years old (Press and Rufener-Press 1974).

There have been several epidemiological studies of COPD prevalence in Czechoslovakia. In a group of 8298 men (52-67 years old) from Prague, 32% were found to have COPD. In rural areas, cases were found in 24.8% of men 40-64 years old and in 7% of the women (Boudik et al. 1969b). From 1971 to 1975, a group of 20000 men 15 years and older (in the Czechoslovak area along the Danube river) were checked for symptoms of COPD. Of this group, 18.4% were diagnosed as having chronic bronchitis (Virsik et al. 1976).

The data on COPD mortality does not adequately describe the importance of this disease in the population. Several facts can explain this situation:

- Frequent coincidence of COPD with other, especially cardiovascular, diseases;
- Many physicians tend to refer to complications of COPD rather than the main disorder as a primary cause of death (Fletcher et al. 1964);
- Sometimes the terminal bronchopneumonia or other concomittant disease is classified as a cause of death instead of COPD (Herles 1964).

According to Higgins (1973) 30-40 thousand deaths per year in the US are caused by chronic disorders of the pulmonary system. In Table 1 we summarize the number of people who died from COPD in different countries of the world in 1980 (WHO 1982).

Table 1. Number of deaths from COPD in different countries by sex and age (WHO 1982).

	Number of deaths					
	Absolute			Percent of all		
Country	Total	Male	Female	Total	Male	Female
Austria	1710	1069	641	1.8	2.4	1.3
Bulgaria	3647	2380	1267	3.7	4.4	2.8
FRG	22025	15079	6946	3.1	4.3	1.9
Hungary	7043	443	2612	4.8	5.8	3.8
Netherlands	2750	2147	603	2.4	3.4	1.1
Poland	10096	7229	2867	2.8	3.8	1.8
England and Wales	20735	14802	5933	3.6	5.0	2.0
Japan	12712	8022	4690	1.7	0.2	1.4

Very important are the data on other, indirect indices of COPD morbidity, since, for example, 10% of all sick-leave in the US is caused by COPD. 90 million US dollars are paid for sick leave to invalids with chronic bronchitis (Higgins 1973) and there were 4.3 cases of COPD related sick-leave per 100 employees in France in 1978 (Pedrizet et al. 1978). Recent estimates of COPD prevalence in the US give 10 million people and, collectively, chronic lung diseases account for more than half a million hospital admissions annually. The limitation of activity occurs later in life so that Medicare pays a large percentage of the health care costs (DHHS 1984). The increase in the number of sick-leave cases, hospitalizations, and hospital days for COPD in Czechoslovakia from the year 1971 is illustrated in Figure 1.

The continuous increase of all the indices is visible (CSSR zdravotnictvi 1983).

2. WHY A COPD MODEL?

Understanding the facts and recognizing the importance of the effects of chronic, noninfectious diseases on the health status of population led us to design a COPD mathematical model. The aim of the model is to facilitate the estimation of COPD prevalence and its development as the basis for creating a rational forecast of health care development. It could serve as a test for various hypotheses about the development of the illness in the affected population. The scope of the model is broader than the health care system itself, since it includes aspects of social care, environment, and economics. This integrative approach introduces quantitative expressions for new ideas that frequently appear in the literature on health care and clinical medicine. The model is based on an understanding of the causes and risk factors responsible for the onset and development of COPD.

3. CAUSES AND DEVELOPMENT OF COPD

Chronic obstructive pulmonary disease is one of the typical illnesses in the developed world. Its etiology is usually described as multicausal. The exact origin of this type of disease during the life span is often undetectable which complicates the exact disclosure of the causative factor. In their daily practice, medical doctors usually only face the full manifestations of clinical symptoms. The relationship of these manifestations to the primary causes is often impossible to analyze in detail (Bajan 1968).

Several epidemiological studies undertaken in various countries have furthered our understanding of many etiopathogenetic factors in COPD development. From the clinical and practical point of view, it is possible to divide these into four categories (Halak and Bajan 1976):

- biological
- physical
- chemical
- allergical.

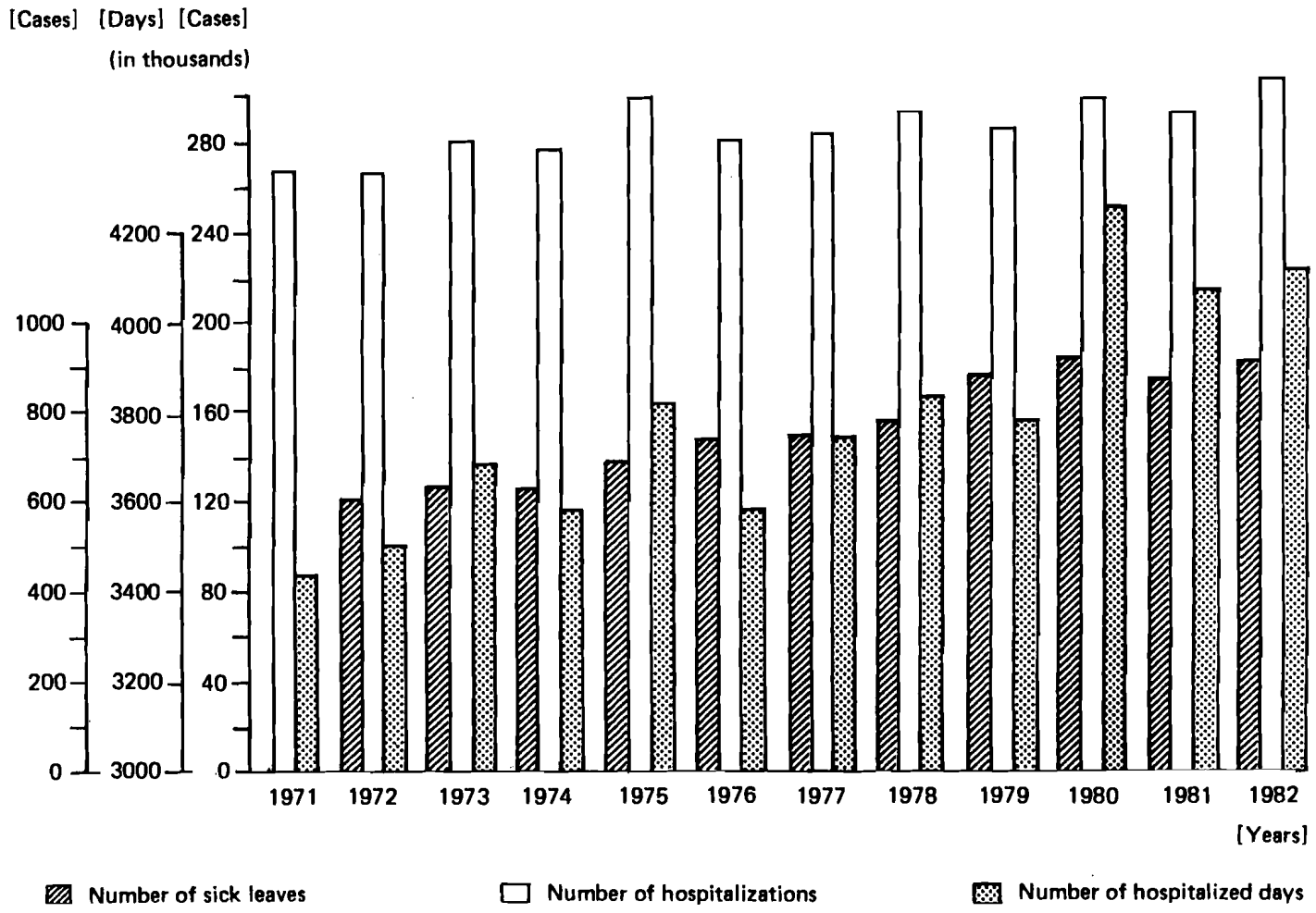


Figure 1. Number of COPD hospitalizations, hospital days, and sick-leave cases in Czechoslovakia, 1971-1982. (Source: Health Care Statistical Yearbooks, 1972-1984.)

All of these factors irritate and infiltrate mucosis of the pulmonary system or, through the antibody reaction (allergy), prepare conditions for the illness to develop (Bajan 1983).

The above categorization of etiopathogenetic factors is especially suitable for didactic purposes, but in practice, one usually faces the mixed effects of these factors. The effect of cigarette smoke, for example, is partly chemical, partly physical, and sometimes even allergical, which is why we include the complex effects of these harmful aspects in terms of a risk factor in the model.

3.1. Risk Factors for COPD Development

Thanks to the wide dissemination of warnings against smoking, cigarette smoking is generally a recognized factor in the development of chronic airways diseases. Much less is known of the other COPD risk factors.

SMOKING: Smoking is undoubtedly foremost in the etiopathology of COPD. The smoker creates an even higher contamination of air for himself by inhaling all the polluting substances from the smoke directly into his or her airways. The number of harmful substances in cigarette smoke is given in Sylla (1978) and exceeds 1300 various kinds. Nicotine alone is a strong enough poison, with lethal doses of about 50 milligrams for a human being.

Functional abnormalities of the lungs are present in a large number of smokers (Fletcher et al. 1976; Dosman et al. 1981; Tashkin et al. 1984). If they continue smoking, a significant number of them develop severe and clinically manifested obstructions of the bronchial tree. Conclusions from several long-term prospective studies suggest that cessation of smoking (at an early stage in chronic airflow development) can prevent progression to a clinically significant disease by reversing the established, related acceleration in annual decline in the lung function to or toward a normal rate of decline (Fletcher and Peto 1977; Bosse et al. 1981; Hjalmarson and Svardsudd 1981). Smoking significantly increases the mortality of smokers. For heavy smokers (more than 25 cigarettes per day), COPD related mortality is up to 25 times higher than for nonsmokers (Ferlinez 1974). This fact was demonstrated also by Hammond (1966) in his study of 1 million males and females. In Table 2 we summarize the results of this study and emphasize the difference in mortality between male smokers and nonsmokers. In the female population this difference was minimal.

Table 2. Mortality differences in smokers and nonsmokers by sex and age (Hammond 1966).

Number of COPD deaths				
	Non-smokers		Smokers	
	Age		Age	
Sex	45-64	65-79	45-64	65-79
Male	10	10	194	175
Female	6	-	7	-

Cigarette smoking acts alone and synergically with the other risk factors of COPD. In the developed countries, it is the single most significant causal entity and its importance is several times greater than air pollution (WHO 1979).

AIR POLLUTION: People who live in urban areas have a significantly higher incidence of COPD than those from rural areas, as proved several times in epidemiological surveys. The frequency of illnesses in airways is much higher in the inhabitants of towns with extreme air pollution (Holland and Reid 1965). Air pollution at the place of work plays a very important role, with symptoms of disturbances of respiratory functions being very frequent in miners and in workers in dusty environments. Several studies have proved the dependence of COPD incidence on the length of employment in certain high-risk professions. The results of investigation of COPD prevalence in the nonsmoking population of workers in coal/electric power plants, unambiguously support this fact (Stejskal et al. 1983).

The reversibility of the effects of this factor was proved in a study of migrants from towns with significantly polluted air in Great Britain to towns with less air pollution in the US (Reid and Fletcher 1971).

A significant degree of air pollution can be achieved by smoking in closed rooms. Nonsmokers, being present in such an environment, inhale cold cigarette smoke with all the dangerous constituents, a phenomenon usually called passive smoking. Children with parents who smoke appear to have small, but measurable, differences in a test of lung function. These children also have an increased frequency of bronchitis, pneumonia, or other respiratory symptoms, when compared to children from homes with nonsmoking parents.

INFECTION: Despite the fact that infection is not among the leading causes of COPD, its role in the increase and development of the sickness is generally recognized and proved (Tager and Speizer 1975). The role and significance of bacterial and viral infection, from the etiopathogenetical point of view, is demonstrated not only in the origin of, but also in the maintenance of the chronic character of the COPD process. Several authors stress the significance of infections of the lower respiratory tract in childhood (Holland et al. 1969; Reid 1969). They take into consideration the higher sensitivity in these children to the development of COPD. According to the results of a study of 2228 schoolboys between the ages of 7 and 11 years (from South Wales and West England), up to 10% of the boys and 6% of the girls had infected airways. In the majority of these children, significant impairment of lung functions was found (Yarnell and Leger 1981).

ALLERGY: These factors present independent and, until now, ambiguously solved problems. Despite plenty of new evidence, there is no known mechanism that can explain the complex of changes that lead to the final state of irreversible obstructive changes on the basis of atopy alone (Orie and von der Lende 1970; Schmidt 1979).

AGE: In connection with the etiological factors that affect the development of COPD, it is necessary to mention one more very important factor--age. COPD frequently occurs in the older age category of the population. Conditions for the onset of the manifestation of COPD create, besides other factors, physiological, structural, and pathophysiological changes in the lungs and in the bronchial tree during the later phases of life, as well as a general decrease of resistance.

GENETICS: In the early 1960s, scholars announced a new syndrom--alpha 1 protease inhibitor deficiency being genetically transferred, later known under the term alpha 1 antitrypsin deficiency (Laurel and Eriksson 1963). Later on, the relationship between this disorder and a significant increase in the size and reduction of the number of alveoli, a diminution of the internal lung surface, and a rearrangement of the lung tissue was stated (Snider and Korthy 1978). It is more than probable that these mechanisms play an important role in the course of COPD (Snider 1984).

ALCOHOL: Burch and DePasquale (1967) hypothesized the existence of an "alcoholic lung disease" and Rankin called attention to the high prevalence of airways obstruction in alcoholics who also smoke (Rankin et al. 1969). Indeed, heavy alcohol consumption is associated with chronic cigarette smoking in a large number of epidemiologic surveys. The association of heavy drinking with increased cough-

ing, excessive mucous hypersecretion, and frequent episodes of non-specific respiratory illnesses has led numerous investigators to speculate that alcohol exacerbates the effects of smoking and contributes to the development of chronic bronchitis (Lebowitz 1981; Krumpe et al. 1984). Despite all of the research being done in this field, the question still remains open.

Etiopathogenetic factors of COPD represent a complicated set of agents of exogenous or endogenous character. They cooperate in their originating changes in the bronchial tree and in creating the conditions for the individual forms and features of COPD manifestation. There are still many open and unsolved questions in the etiopathogenesis of COPD, despite the evident progress of research into the effects and influence of different COPD risk factors. New discoveries in this field will not only have a theoretical impact, but also a significant impact on prevention, early diagnosis, and effective therapy.

4. COPD MODEL OBJECTIVES

Health care statistics adequately describe the prevalence of tuberculosis, venereal diseases, and communicative diseases in a majority of countries. This is because when medical statistics were developed, tuberculosis and infectious diseases were the most important health problems. This conjunction of medical statistics and infectious diseases is described in the *curriculum vitae* of Florence Nightingale, who helped to pioneer the revolutionary notion that social phenomena could be objectively measured and subjected to mathematical analysis (Cohen 1984). Besides this historical relationship, there is another feature of routine medical statisitics: the majority of them are episode based (case based), not based on the individual (Zacek 1984); modern health care calls for knowledge of the latter type of information. The prevalence image is governed by chronic, noncommunicable diseases. However, no current routine source of medical information treats the whole history of the development of a chronic disease from its onset until its end. Thus, health care managers frequently do not know the prevalence of a particular illness in the population. However, they must have this knowledge in order to effectively fight against the illnesses.

Prevalence studies are often used to examine occurences of chronic diseases, involving a limited proportion of the population who suffer from a particular disease, disability, syndrome or studied symptom within a short period of time. Of course, such surveys are usually very expensive, which is why they are restricted

in time and area. The description of the allocation of a number of sick people is the usual result and such information is of great value for health care management. However, it is insufficient for the purpose of setting objective targets and procedures for the health care system and for short- and long-term planning, especially the latter.

In a recent paper, we drew attention to the possibilities of using mathematical modeling to transform static information--which has been accumulated by means of different surveys--into a dynamic tool in the hands of health care managers (Koonce et al. 1984). During the designing of the COPD model, we have kept in mind this target. The design is based on understanding the current situation and defining our aims. The general aim, remaining after all of our efforts, is to decrease the prevalence of COPD. To further this, the model can aid the task of discovering the effects of different risk factors on COPD prevalence diminution. There is probably no need for explaining that it has, besides the ethical consequences, such a diminution would have a serious economic effect with respect to the whole of society. The model also follows this aspect of health care.

We assume that after some time the model will find its place within the system of continuous health-care development forecasts. Even more, we suggest that this model could be used in postgraduate training of health care managers at different levels. Research into COPD could profit from utilizing this model as well.

5. COPD MODEL STRUCTURE

The COPD prevalence model consists of two major blocks of logic:

- prognosis for population development
- prognosis for the development of COPD risk factors development.

The possibility of testing different scenarios allows a holistic approach to the model.

5.1. Population Development Prognosis

The forecast is based on a simplified view of population development. Data for the demographic prognosis are estimated from the number of newborns, the number of all deaths, and the number of transitions between age groups. Such factors as migration, male/female ratio, and so on, were not considered in the fore-

cast. The overall prognosis is based on data from Slovakia, 1983.

5.2. COPD Risk Factors Prognosis

This module represents the key point of the whole model. We have employed our knowledge of COPD etiopathogenesis in this area. The whole model construction is according to the following three population divisions: (1) healthy individuals, (2) those in COPD risk, and (3) those suffering from COPD.

Healthy Individuals: We define the members of this group as those individuals without COPD symptoms and those not under the influence of any of the risk factors. We ignore the possibility of genetic transfer of COPD, which means that, from our point of view, all newborns are healthy, having the same probability of staying healthy or entering any of the risk groups.

Individuals with COPD Risk Factor: We begin from the point of view of the model targets in analyzing the problem of risk group selection. The only ones we selected were those with important etiopathogenesis and the real possibility for intervention by a health care system or society. That is why we chose the following factors:

- *smoking*: one of the most important factors, influencing the largest part of the population, but the effect of intervention by health care or society is still dubious.
- *air pollution*: compared with smoking this factor is of secondary importance. The role of securing a diminution or even elimination of this is of primary importance.
- *frequent respiratory infections*: the role of this factor in the development of COPD in adulthood is still not clearly defined. In the struggle against this factor, the last word has not yet been said.

COPD sick individuals: Individuals who fulfil the WHO criteria for chronic bronchitis, lung emphysema, or bronchial asthma were placed in this group (WHO 1980).

The overall model structure is shown in Figure 2.

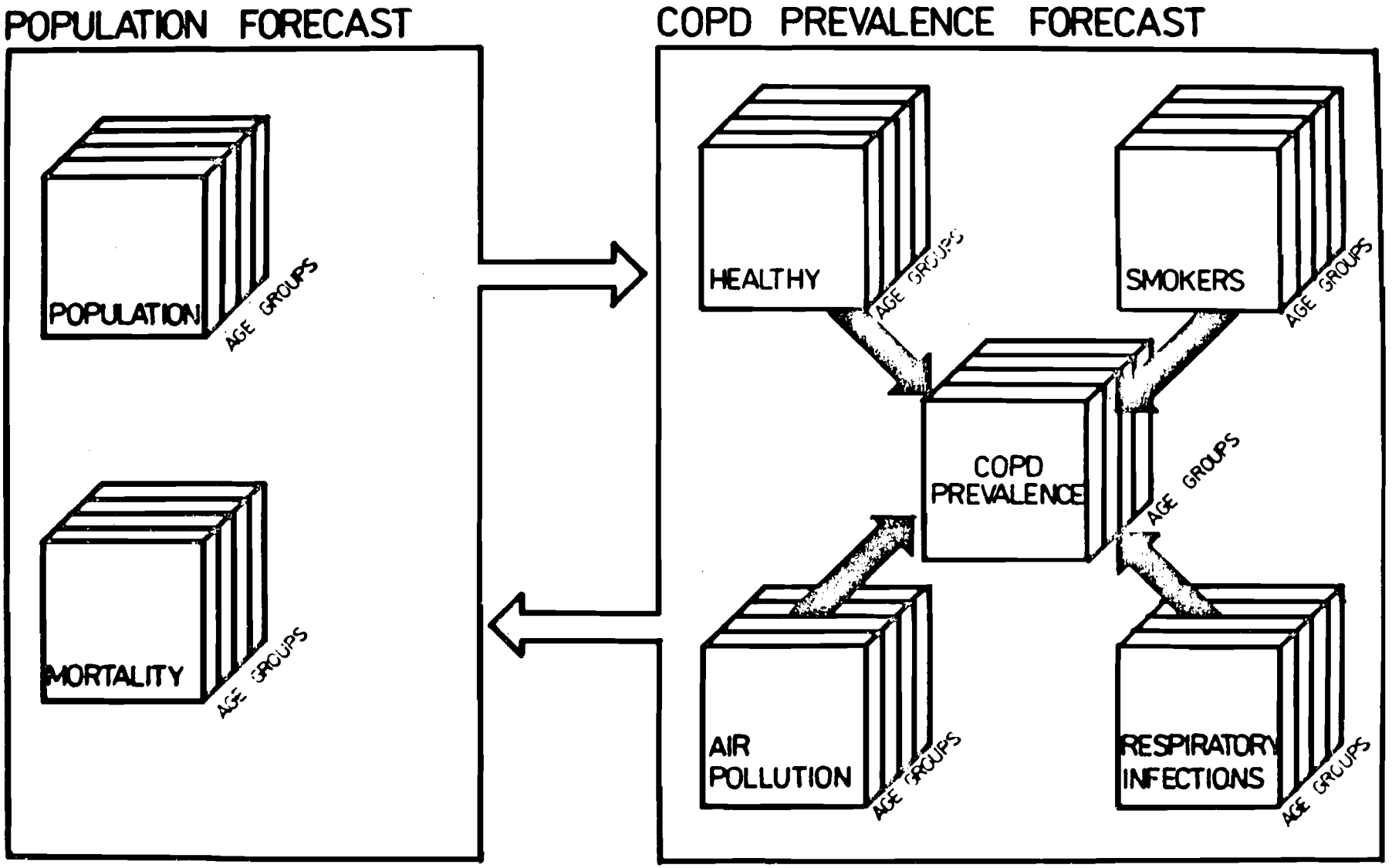


Figure 2. COPD model structure.

6. COPD MODEL REALIZATION

6.1. Model Derivation

The part of the COPD model that describes population dynamics is based on the concept of people flow from one age category to another in each year. A certain number of people are born and a certain number die. Denoting as $n_1(t)$ the number of people in the group aged younger than 20 years, $b(t)$ the number of newborns, and μ_1 the death rate for the first age category in year t, one can describe the population as follows:

$$n_1(t) = n_1(t-1) + b(t) - 0.05n_1(t-1) - \mu_1 n_1(t-1) \quad . \tag{1}$$

The constant 0.05 represents that part of the population which enters the next age group (20-year age groups in this case). We do not have newborns in the older ages, but instead we have an inflow of people from the previous age groups. Because we have 20-year age groups we use the 0.05 constant again.

$$n_2(t) = n_2(t-1) + 0.05n_1(t-1) - 0.05n_2(t-1) - \mu_2 n_2(t-1) \tag{2}$$

$$n_3(t) = n_3(t-1) + 0.05n_2(t-1) - 0.05n_3(t-1) - \mu_3 n_3(t-1) \quad . \tag{3}$$

We have only one transition from the last group, so we can write the equation as follows:

$$n_4(t) = n_4(t-1) + 0.05n_3(t-1) - \mu_4 n_4(t-1) \quad . \tag{4}$$

The second part of the COPD model describes the dynamics of the population under different risk factors. As already mentioned, the people not subjected to extra risk of COPD development were considered as a special type. Denoting j for the age categories, one can describe this group as follows:

$$n_j^1(t) = n_j^1(t-1) + p - (\rho + \lambda_j^1)n_j^1(t-1) - \bar{\mu}_j n_j^1(t-1) \quad . \tag{5}$$

Assuming four age categories, we have $j = 1, \ldots, 4$. For those under 20 years of age, p denotes the number of newborns. For others it denotes the part of the population that enters the category from the previous one:

$$j = 1 \qquad p = b$$

$$j > 1 \qquad p = 0.05\, n_{j-1}^1(t-1) \quad . \tag{6}$$

People from this group may enter one of the other risk groups, become ill from

COPD, or even die. The process of transition to other health hazards is represented by the sum of the transition coefficients ρ

$$\rho = \sum_{i=1}^{3} \rho^i \quad . \tag{7}$$

The possibility of the onset of a COPD disease in healthy individuals is depicted by the coefficient λ_j^1. The number of people dying in this group from causes other than COPD is:

$$\bar{\mu}_j n_j^1(t-1) \quad , \tag{8}$$

when $\bar{\mu}$ stands for mortality rate.

Applying similar notation, one can derive equations for estimating the number of cigarette smokers:

$$n_j^2(t) = n_j^2(t-1) + \rho^1 n_j^1(t) + p - \lambda_j^2 n_j^2(t-1) - 0.05 n_j^2(t-1) - \bar{\mu}_j n_j^2(t-1)$$

$$j = 1 \qquad p = 0 \tag{9}$$

$$j > 1 \qquad p = 0.05\, n_{j-1}^2(t-1) \quad ,$$

- the number of people in an air-polluted environment:

$$n_j^3(t) = n_j^3(t-1) + \rho^2 n_j^1(t) + p - \lambda_j^3 n_j^3(t-1) - 0.05 n_j^3(t-1) - \bar{\mu}_j n_j^3(t-1)$$

$$j = 1 \qquad p = 0 \tag{10}$$

$$j > 1 \qquad p = 0.05\, n_{j-1}^2(t-1) \quad ,$$

- and the number of children with frequent respiratory infections:

$$n_1^4(t) = n_1^4(t-1) + \rho^3 n_1^1(t) - \lambda_j^4 n_a^4(t-1) - 0.05\, n_1^4(t-1) - \bar{\mu}_1 n_1^4(t-1) \quad . \tag{11}$$

Since the aim of the model is to estimate the number of people with COPD, the following equation was derived:

$$n_j^5(t) = n_j^5(t-1) + \lambda_j^1 n_j^1(t-1) + \lambda_j^2 n_j^2(t-1) + \lambda_j^3 n_j^3(t-1) + \lambda_j^4 n_j^4(t-1)$$

$$+ p - \bar{\mu}_j n_j^5(t-1)$$

$$j = 1 \qquad p = 0 \tag{12}$$

$$j > 1 \qquad p = 0.05\, n_{j-1}^5(t-1)$$

to depict the COPD prevalence dynamics.

6.2. Implementation

The following items were used for the initialization of the forecast:

- age structure of the population of the Slovak Socialistic Republic (SSR), 1983;
- general death rate for population of SSR, 1983;
- number of newborns in SSR, 1983;
- number of COPD related deaths in SSR, 1983;
- risk of COPD in individuals in SSR, 1983, with and without risk factors;
- COPD risk-factor prevalence in the population of SSR, 1983;
- coefficients of transition from the group without risk to that with risk;
- average length of stay in the hospital for a patient with COPD;
- coefficient of transition from the group of COPD ill to disabled;
- average number of days in sanatoriums.

We divide the population into four groups according to age:

- 0-19 years;
- 20-39 years;
- 40-59 years;
- 60 years and over.

We are aware of the error possibly arising from this rough age stratification, but restrictions due to the computer used did not allow us to probe this problem more deeply. The same situation occurs in the sex structure. The following may support our decision not to consider sex differences: in developed countries smoking started as a predominantly male phenomenon and women started to smoke much later. While men were usually the first to stop smoking, smoking continued to increase among women (although it has now started to level off, apparently as a result of smoking control activities) at a much lower rate than that reached by men (WHO 1979). So we tried to select data describing the whole population regardless of sex.

In our model design, we employed the assumption that each of the risk factors affects the population independently. We abstracted their synergistic effects, having in mind the following ranking:

(1) smoking

(2) air pollution

(3) frequent respiratory diseases in childhood.

So, if an individual is a smoker, it is possible to neglect the influence of other contaminating factors. Similar hypotheses were also accepted for the other combinations of factors, since the lack of data on the effects of the combinations of risks forces this simplification.

We implemented the COPD model on an APPLE-IIe microcomputer in APPLESOFT BASIC. The program is assembled from several modules, as shown in Figure 3. Data specifying the starting conditions of the model are incorporated into the program by means of the command DATA.

6.3. Demographic Data

The demographic data for the SSR population (age structure, death rate, newborns) were extracted from the *Statistical Health Care Year Book* for Czechoslovakia (CSSR zdravotnictvi 1983)*. Specific mortality for COPD was derived from a statistics book (Pohyb obyvatelstva v SSR, 1983). The number of COPD deaths is not enumerated in this book, which is why we considered the sum of deaths from following diagnostic categories, according to the *International Classification of Diseases* (VIII-th revision):

- ICD 491 chronic inflammation of bronchi-chronic bronchitis
- ICD 492 lung emphysema
- ICD 493 bronchial asthma.

There are three types of mortalities considered in this model: overall mortality, specific COPD mortality, and mortality without COPD. This distinction was employed partly to verify the different effects on mortality and partly because of the possibility of extending the model to other diseases.

*These data are included in Table 3.

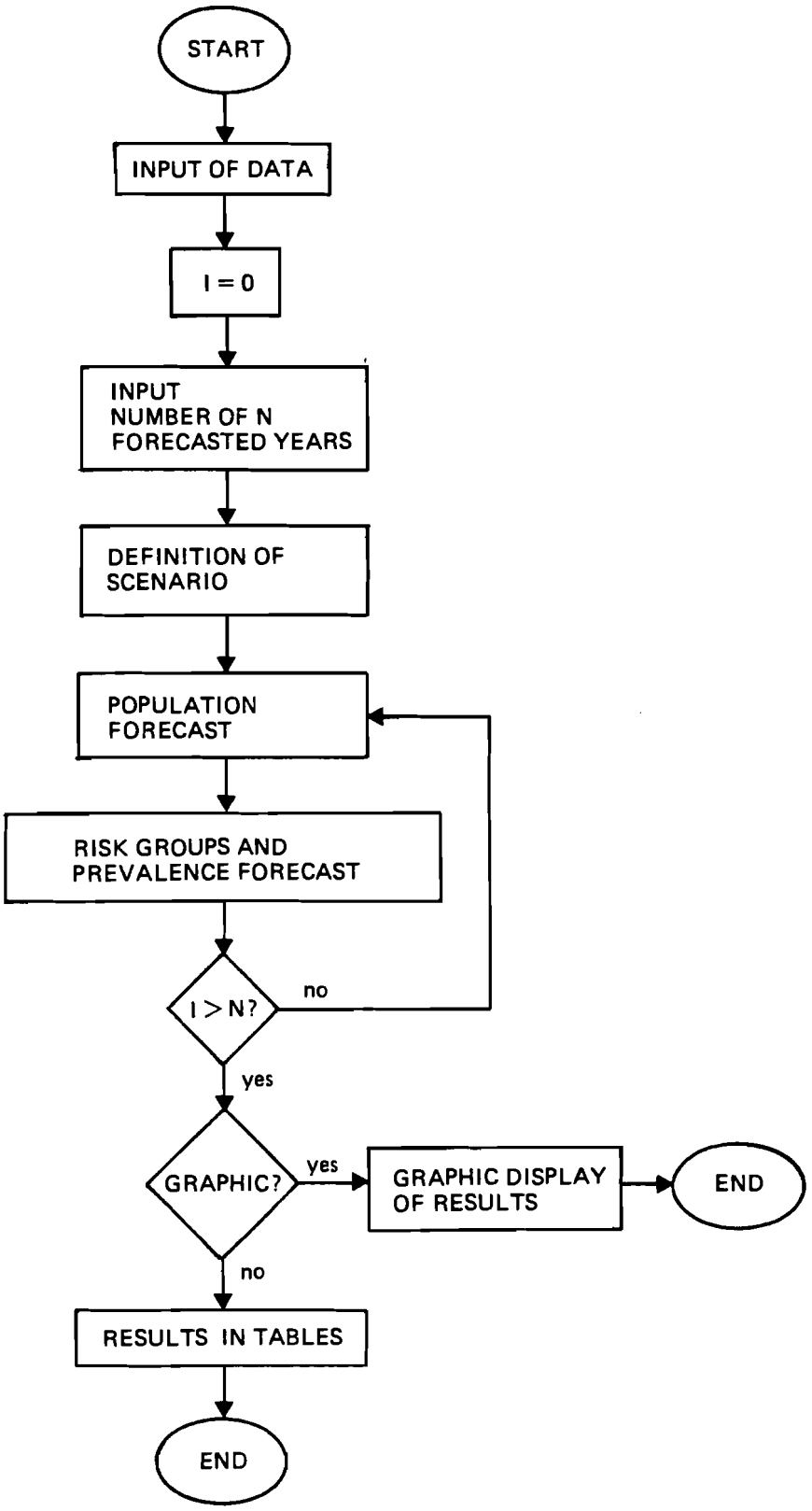


Figure 3. The COPD model realization in computer program.

Table 3. Population, number of deaths without COPD, and number of deaths from COPD (Pohyb obyvateslstva v SSR 1983).

Initial demographic data for Slovakia, 1983			
Age group	Population in SSR	Deaths without COPD	COPD deaths
0-19	1736000	3342	1
20-39	1619000	1912	7
40-59	1070000	6735	123
60-	731000	33269	1760

6.4. Number of COPD Cases

There are two main channels of information about general morbidity data:

- routine statistics
- special investigations

Most countries do not supply routine data on COPD prevalence (Shigan 1977). However, the data do describe hospitalizations, sick-leave cases and days, disease specific mortality, and other indirect measures of prevalence. The most appropriate source of prevalence data are special investigations. We based the estimation (of the initial COPD prevalence in the SSR) on the results of a study of 20000 inhabitants, 15 years and older, in the Danube area (Virsik et al. 1976) along with an investigation of about 60000 people (male and female) in the West of Slovakia (Kruty et al. 1975). For a more precise estimation of prevalence, we plan an extension of the model with a module of prevalence estimation based on some indirect prevalence indices. The values of the initial COPD prevalence are summarized up in Table 4.

6.5. Risk of Developing COPD

We consider the estimation of coefficients--for expressing the risk of COPD development in an individual sorted by risk factors--to be of crucial value for the success of the model. The way that we perceive the risk considers the pathogenic power of the etiology factor, which means the magnitude of risk for an individual

Table 4. Initial COPD prevalence in Slovakia, 1983.

Age	Number of cases
0-19	143000
20-39	298000
40-59	276000
60-	382000

subject to one of the factors that increases the likelihood of developing the considered disease. According to Zacek (1984), the measure of risk is according to the magnitude and duration of the association. Mathematical statistics offer a number of different techniques for establishing this (Armitage 1971). Case-control and cohort studies are used to measure the increased risk of incurring a particular disease if a certain factor is present. In cohort studies, such estimations can be done directly by observing the experience of groups of subjects with and without the factor. In a case-control study the data do not present an immediate answer to this type of question. The association between the factor and the disease could be measured by the ratio of the risks of the disease being positive for those with and those without the factor.

We processed coefficients of relative COPD risk according to data from COPD epidemiological studies carried out in the SSR during recent years. Because we could not find a survey of the consequences of all three risk factors used, we combined data from several sources. Numerous surveys were conducted to compare the incidence and risk of COPD in the inhabitants of rural and urban areas (Olejnicek et al. 1974; Kubik et al. 1978; Coufal et al. 1973). We solved the question of COPD incidence in youths with the help of a study oriented selectively on younger age categories (Vyslouzil et al. 1975). For the risk of COPD development in individuals with frequent respiratory infections during childhood we used the results of the study on the population of South Wales and the West of England (Yarnell and Leger 1981), because we do not know of any such survey in Slovakia. Table 5 contains values of coefficients used.

Table 5. Risk of COPD onset for people under different risks, by age.

	Age			
Risk group	0-19	20-39	40-59	60-
Without risk	0.02	0.1	0.2	0.25
Smokers	0.05	0.3	0.6	0.75
Air pollution	0.1	0.2	0.25	0.3
Respiratory infections	0.39	0	0	0

6.6. Risk Factors Prevalence

The estimation of risk-factor incidences in the population could, at first glance, be regarded as an easier task compared to previous ones, but we have to state rational limitations to be successful. There are a large number of surveys that consider the distribution of smokers in the population. Different clusters of smokers are used according to, e.g., what is smoked, for how long, how deeply the smoke is inhaled, etc. We made use of the following stratification of people according to their smoking habits:

- smokers
- nonsmokers

We restricted our attention to cigarette smoking alone. We received some valuable data on smoking from the Special Study of Tobacconism in Slovakia (Katriak 1983). They investigated the smoking habits of 1700 inhabitants of the SSR 14 years and older, chosen at random according to sex, age, social status, and residential area. The study represents a complex view on smoking epidemiology in the SSR.

More problems were faced in estimating the number of people affected by polluted air. For Slovaks living in areas with good quality air and for those living in towns with proved air pollution, no appropriate data were found. We understand that this division is too general, but until better data is available we can use nothing else. The data on air pollution were estimated according to Kühnl (1982).

The frequency of respiratory diseases in children is about 3-4 illnesses per year, but is usually less frequent for children in rural areas. We estimated the number of children with frequent respiratory infections with the help of a pediatrician's expert estimation. Our opinion, based on experience with this data

estimation, is that no more precise data on that problem will be available in the near future. The proportions of people subject to different risks are summarized in Table 6.

Table 6. Proportions of people subject to different risk factors by age.

	Age			
Risk group	0-19	20-39	40-59	60-
Smokers	0.144	0.277	0.245	0.07
Air pollution	0.273	0.161	0.130	0.045
Respiratory infections	0.264	0	0	0

6.7. Transition Coefficients Between Groups

These coefficients describe how an individual subject to none of the mentioned health risks can enter one of the risk groups. Derivation of these coefficients is based on the assumption that a newborn child is under no risk, which excludes the possibility of hereditary defects. Numbers for transitions to the group of smokers were derived from the already mentioned study of cigarette smoking epidemics in Slovakia (Katriak 1983). The estimation of coefficients for air pollution was done using migration data for Slovakia (Kühnl 1982). Because of the lack of statistical data on children's respiratory infections, expert opinions were used. The experts (physicians) were familiar with the epidemiological situation among the children in Slovakia. Table 7 comprises the described coefficients for Slovakia.

Table 7. Coefficients of transitions between the group of people with no mentioned health hazard compared to those with hazard.

	Age			
Risk group	0-19	20-39	40-59	60-
To smokers	50	30	30	30
To air-polluted areas	0.025	0.06	0.06	0.165
To respiratory infections	0.08	0	0	0

7. RESULTS AND DISCUSSION

The COPD model allows the user to forecast the prevalence development to the year 2003 (20 years forecast). Figure 4 depicts the basic development of prevalence, when no interventions are assumed. Notice the steady increase in quantity of sick people, especially in age categories, which. should strike the attention of health care managers and policymakers.

Several scenarios were used to test different approaches and their impacts on the population. The transformation of an expert's ideas into scenarios represents a quantification of the hypothesis, and it is possible to express such a quantification in several different ways. The model uses percentages as a measure of change between the original state and the hypothetical situation. The change could be introduced in any year of the forecasted period.

Tested hypotheses were chosen in order to highlight the answers to these questions:

- how would a change in the amount of people affected by different risks influence COPD prevalence in the future?
- how would the prevalence react to a change in more effective COPD therapy and prevention?

Figures 5 and 6 depict situations in which different changes in the smoking situation are assumed. The hypotheses of reducing the number of smokers and of increasing the smoker population were tested. Reducing the amount of people affected by cigarette smoke has long been a task of many, not necessarily health care, authorities all over the world, with several programs of WHO and other institutions trying to treat this problem. US smoking habits are shown in Table 8.

Column (1) of Table 8 shows that the total US cigarette consumption has increased fairly steadily over the past two decades, but the growth in total consumption has not kept pace with the growth in the smoking population. This is reflected in column (3) in which the aggregate data are converted into cigarettes per adult (individuals over 17 years of age). As columns (3) and (4) show, by this measure cigarette consumption has fallen steadily, if gradually, since 1973 (Warner 1983).

Figure 5 shows the prevalence change after the effective antismoking campaign was introduced (dimunition of smokers to 80% in people younger than 20 years and to 70% between 20-39 years of age in the year 1985).

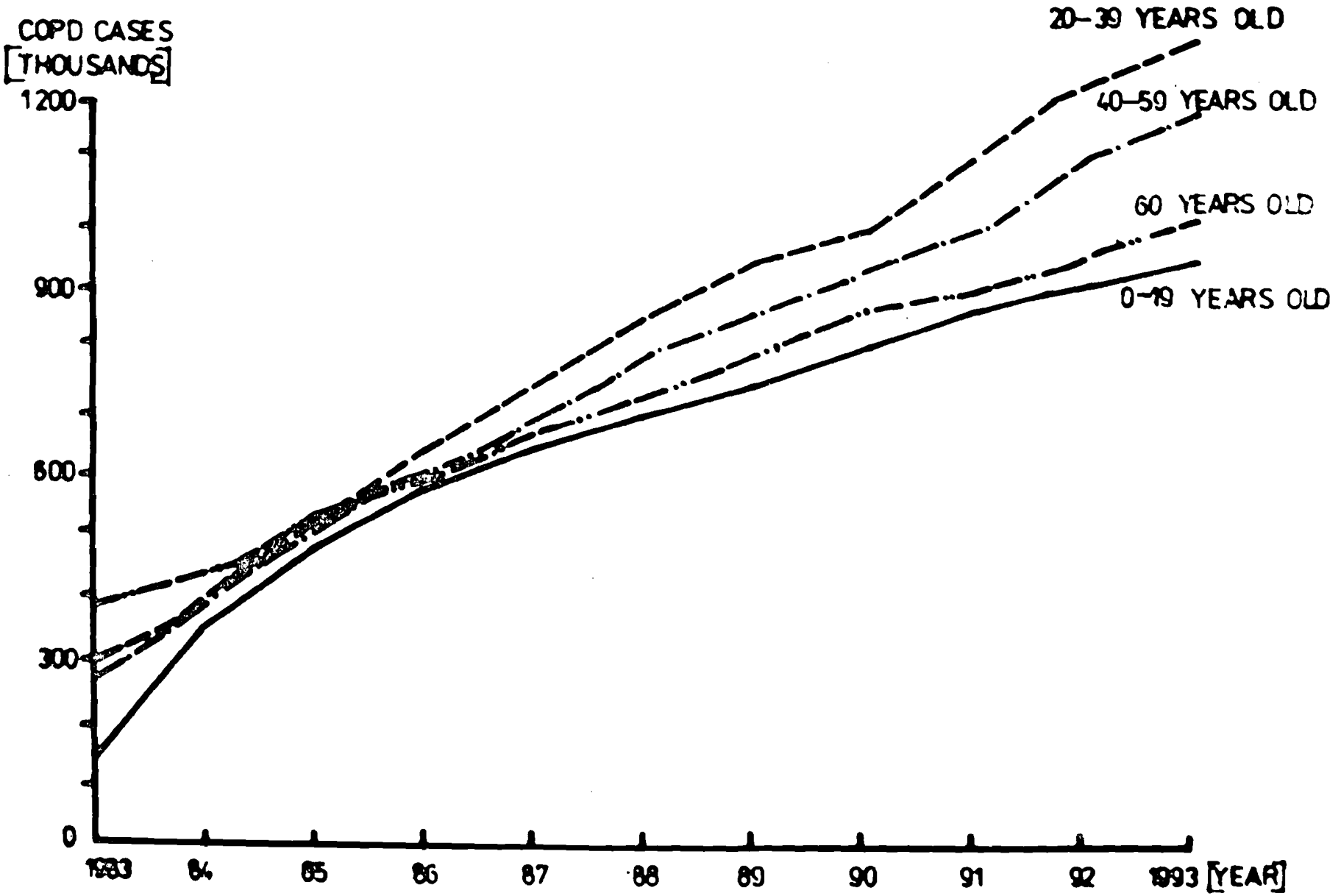


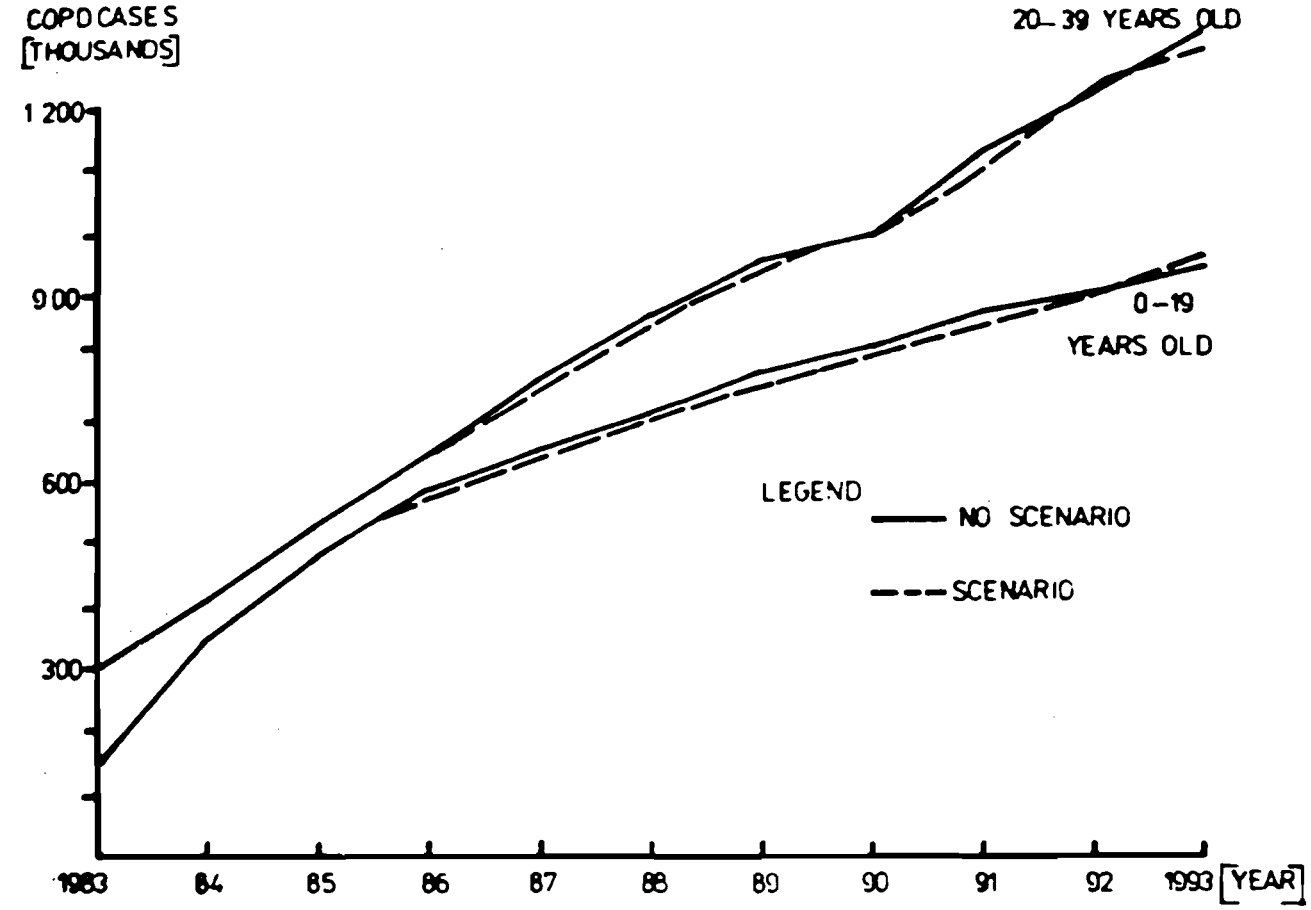
Figure 4. Forecast of COPD prevalence.

Table 8. Total and adult per capita cigarette consumption by year (Warren 1983).

Year	Total consumption (billions of cigarettes) (1)	Percentage increase (decrease) from preceding year (2)	Consumption per adult (3)	Percentage increase (decrease) from preceding year (4)
1960	484.4	--	4171	--
1961	502.7	3.8	4266	2.3
1962	508.4	1.1	4265	--
1963	523.9	3.0	4345	1.9
1964	511.2	(2.4)	4194	(3.5)
1965	528.7	3.4	4263	1.6
1966	541.2	2.4	4287	0.6
1967	549.2	1.5	4280	(0.2)
1968	545.7	(0.6)	4186	(2.2)
1969	528.9	(3.1)	3993	(4.6)
1970	536.4	1.4	3985	(0.2)
1971	555.1	3.5	4037	1.3
1972	566.8	2.1	4043	0.1
1973	589.7	4.0	4148	2.6
1974	599.0	1.6	4141	(0.2)
1975	607.2	1.4	4123	(0.4)
1976	613.5	1.0	4092	(0.8)
1977	617.0	0.6	4051	(1.0)
1978	616.0	(0.2)	3967	(2.1)
1979	620.0	0.6	3924	(1.1)
1980	630.0	1.6	3880	(1.1)

The awaited effect of dimunition of COPD cases was not as significant as one might have hoped. Nevertheless, a decrease of 1000 COPD sick people in the younger age groups will have a more significant effect on their health when they become older. Practically no change was achieved in the older age categories, which reflects the time delay required for an individual from one age group to reach the older age category. As we have 20-year age groups, only a half of the population will reach the older age groups during the forecasted period (10 years).

The scenario with increasing numbers of smokers in two younger-age categories was tested (the number of people younger than 20 years who start smoking is increased by 140% and that of people between 20-39 years of age by 130% of the 1985 figures). The results were just the opposite to those derived from the first scenario, as shown in Figure 6.


COPD CASES
[THOUSANDS]
1 200
900
600
300
20-39 YEARS OLD
0-19
YEARS OLD
LEGEND
NO SCENARIO
SCENARIO
1983
84
85
86
87
88
89
90
91
92
1993
[YEAR]

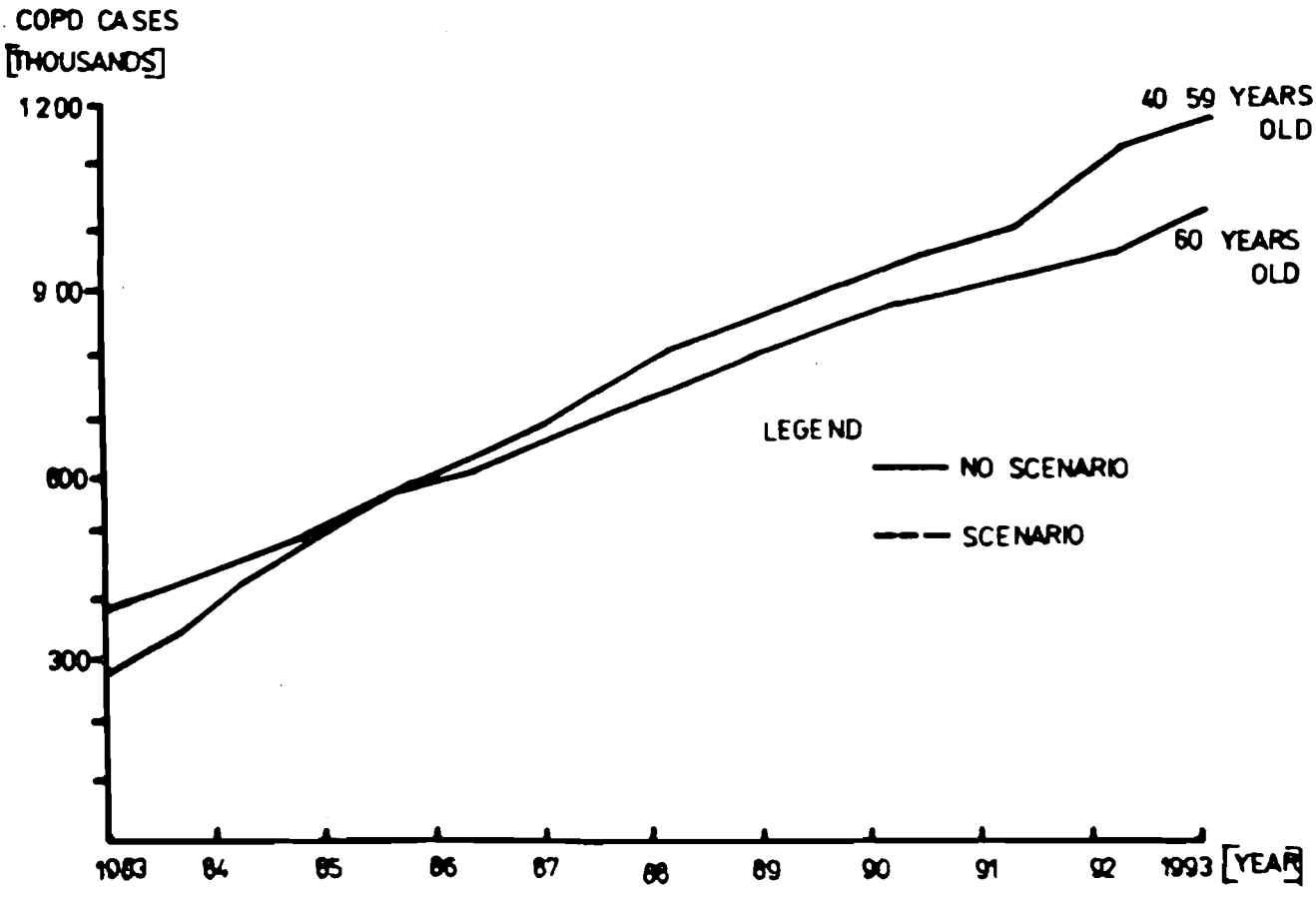

COPD CASES
[THOUSANDS]
1200
900
600
300
40 59 YEARS
OLD
60 YEARS
OLD
LEGEND
NO SCENARIO
SCENARIO
1983
84
85
86
87
88
89
90
91
92
1993
[YEAR]

Figure 5.

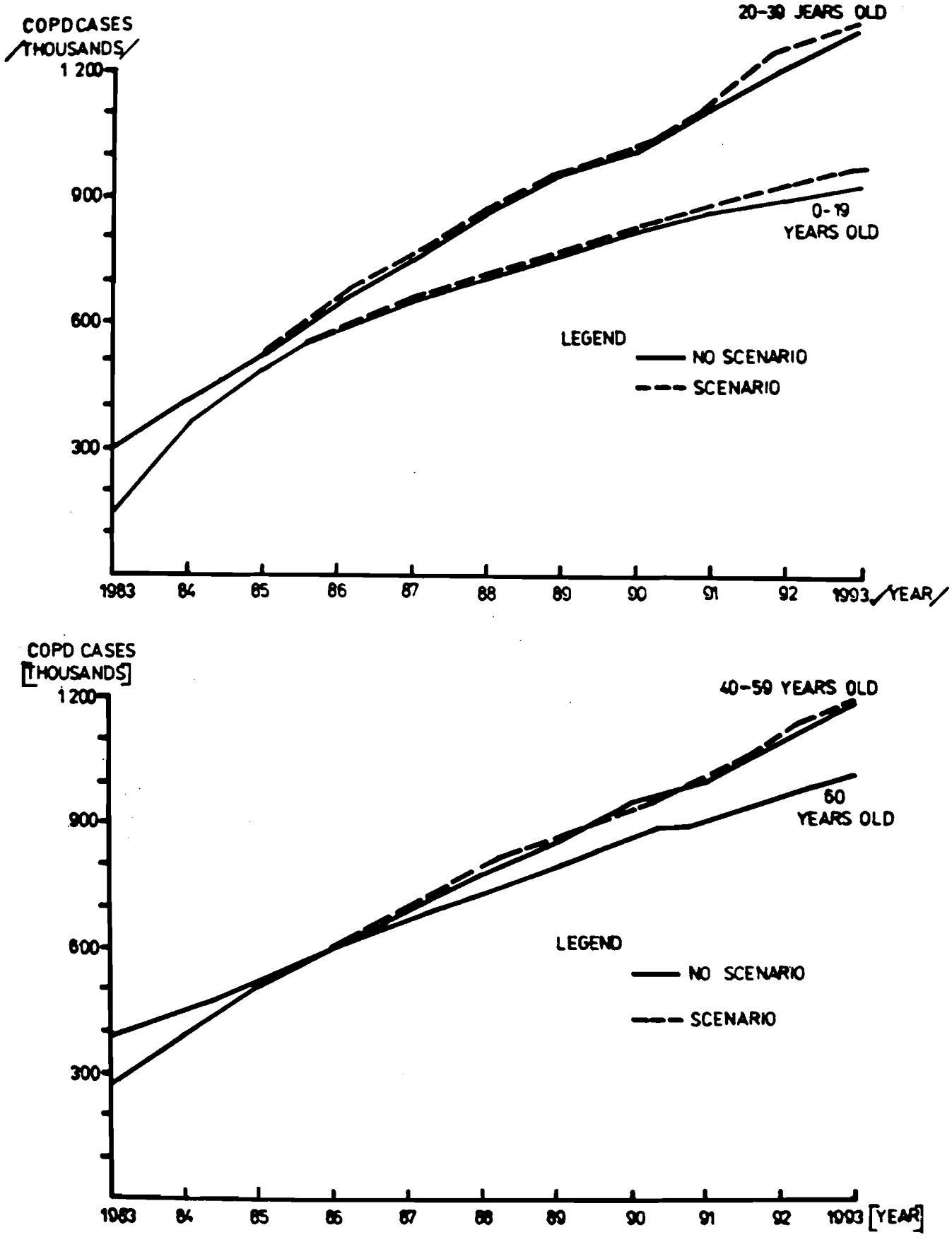

COPD CASES
/THOUSANDS/
1 200
900
600
300
20-39 JEARS OLD
0-19
YEARS OLD
LEGEND
NO SCENARIO
SCENARIO
1983
84
85
86
87
88
89
90
91
92
1993
/YEAR/
COPD CASES
[THOUSANDS]
1 200
900
600
300
40-59 YEARS OLD
60
YEARS OLD
LEGEND
NO SCENARIO
SCENARIO
1983
84
85
86
87
88
89
90
91
92
1993
[YEAR]

Figure 6.

The most affected population would be in the younger-age categories. The hypothesis used reflects a situation that is common in nearly all the developed countries. The number of heavy smokers among men in twenties and their thirties increased, but the number among older men decreased. The number of female heavy smokers, on the other hand, has continued to increases until women were in their fifties (Stoto 1985).

Another two scenarios tested the situations in which substantial changes in COPD prevention and treatment effectiveness occur. Screening for initial lung function impairments and complex treatment of acute upper respiratory infections together with decreasing air pollution, are the main possibilities for preventing COPD illness. Figure 7 reflects the situation with increased effectiveness of preventive programs. The usual target of such programs is the adult part of the population--up to 60 years of age. The hypothesis employed suggests the rise in prevention effectiveness will be 140% in the age groups 20-39 and 40-59 years. The response of the model was immediate. The change was introduced in 1984 and a significant decrease in COPD prevalence appeared during the same year in the second and third age categories and after four years in the older age categories.

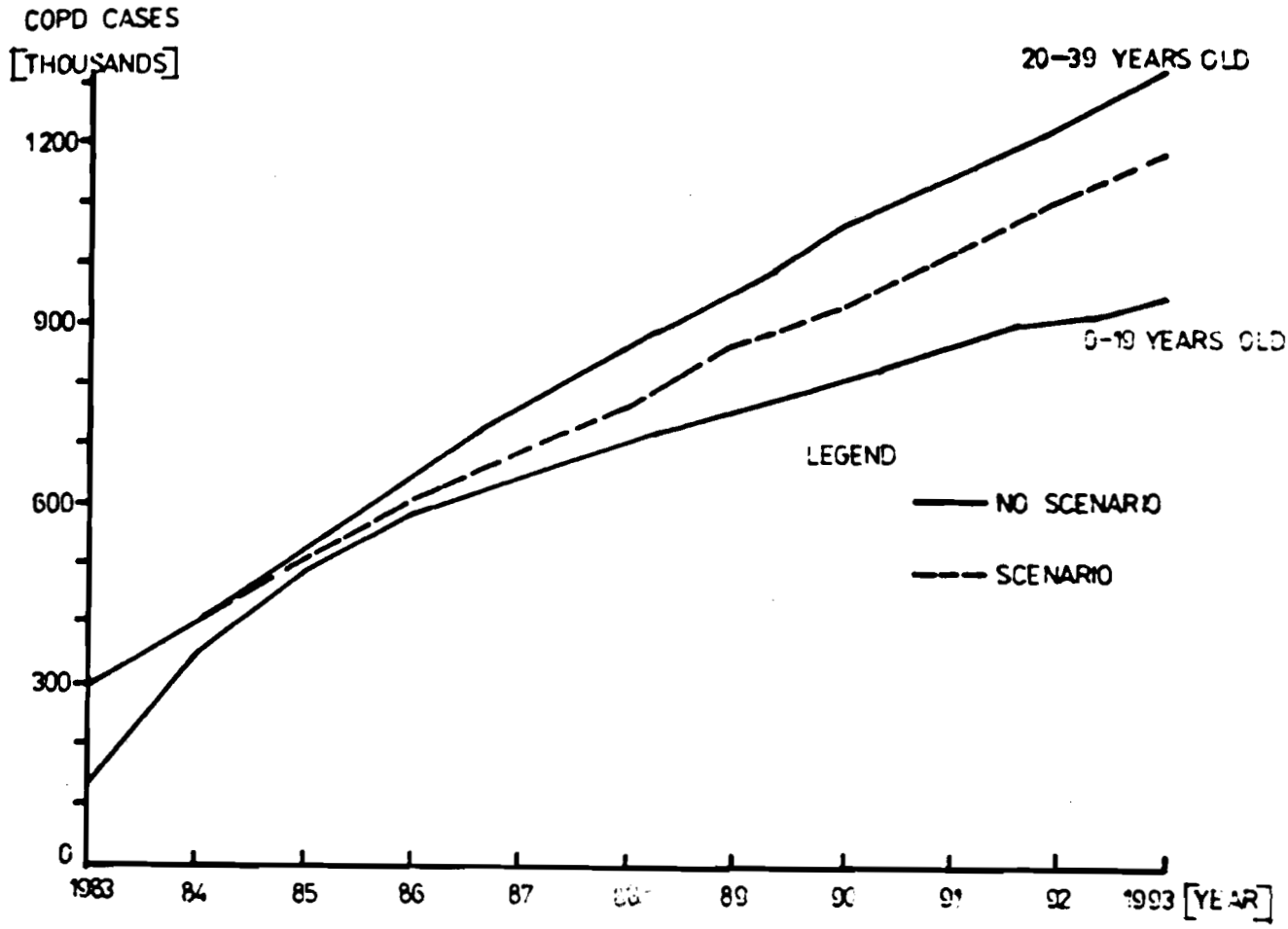
Assuming that the health authorities will not pay appropriate attention to the preventive activities, the hypothesis of a decline to 60% of the current status in the same age groups as the previous scenario was tested. The change was introduced in 1984 and the results of the forecast are shown in Figure 8.

CONCLUSIONS

More hypotheses than mentioned here were checked, most striking results being given as examples of the model's runs. The described model is one of the first steps in our efforts to model sociodemographic impacts of chronic diseases on populations. The results showed us what we should use in future population models.

Based on the experiments with the model, the following conclusions can be drawn:

- the model provides the experimentor with meaningful forecasts and enables him or her to test different types of scenarios;
- the COPD model is sensitive enough to simulate assumed changes;


COPD CASES
[THOUSANDS]
20-39 YEARS OLD
1200
900
600
300
0
0-19 YEARS OLD
LEGEND
NO SCENARIO
SCENARIO
1983
84
85
86
87
89
90
91
92
1993 [YEAR]

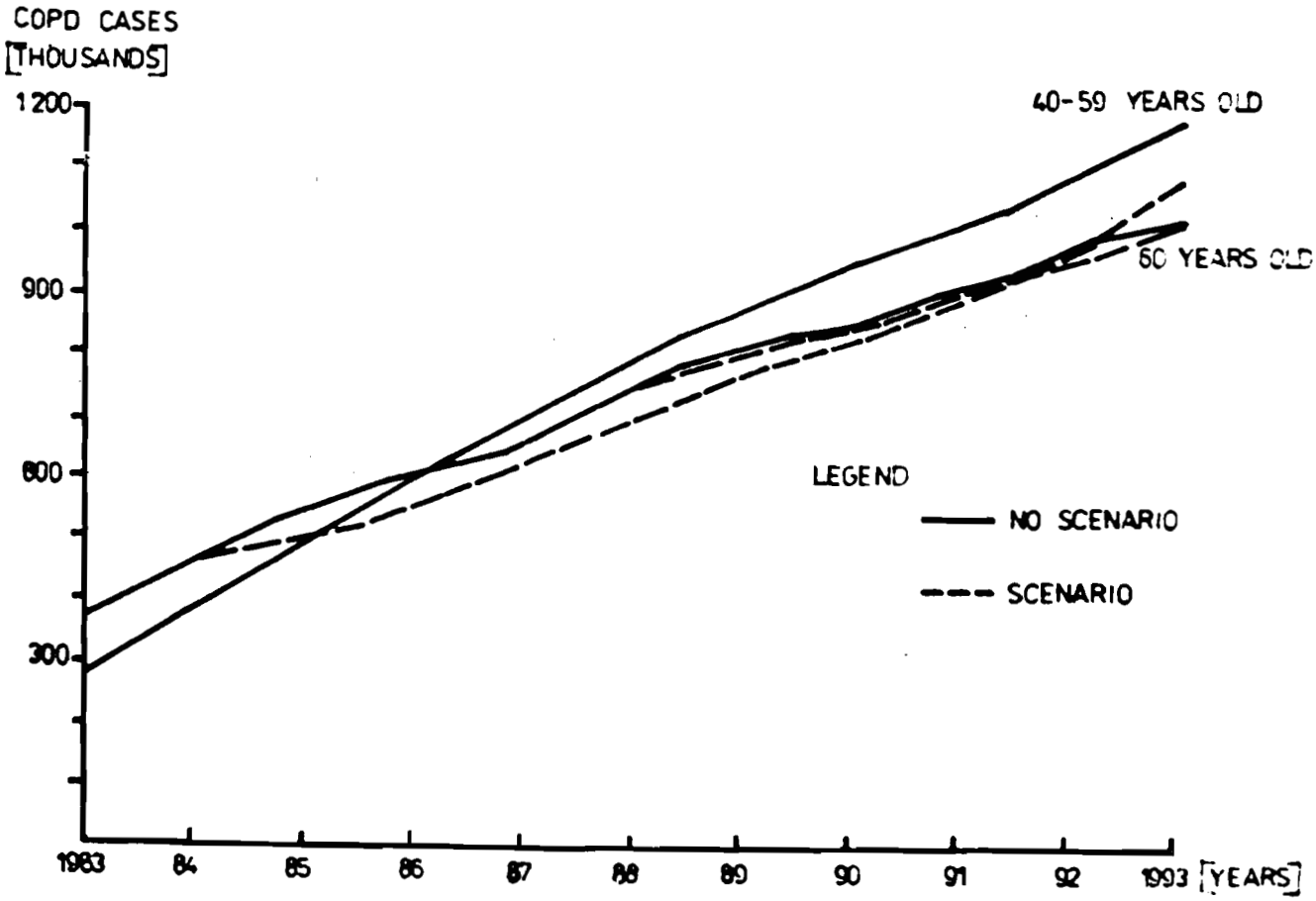
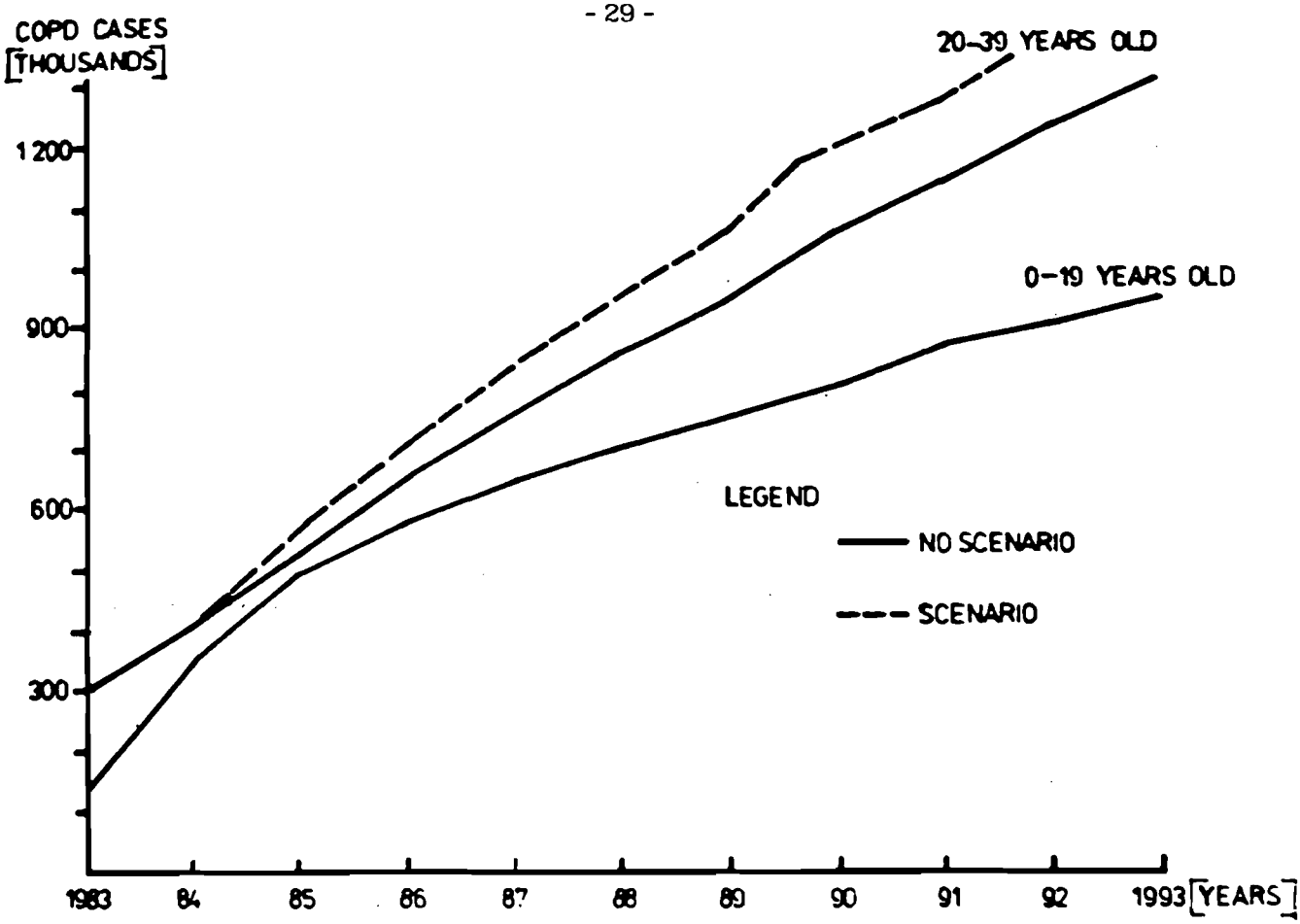

COPD CASES
[THOUSANDS]
1200
40-59 YEARS OLD
900
60 YEARS OLD
600
LEGEND
NO SCENARIO
SCENARIO
300
1983
84
85
86
87
89
90
91
92
1993 [YEARS]

Figure 7.


COPD CASES
[THOUSANDS]
1200
900
600
300
20-39 YEARS OLD
0-19 YEARS OLD
LEGEND
NO SCENARIO
SCENARIO
1983
84
85
86
87
88
89
90
91
92
1993
[YEARS]

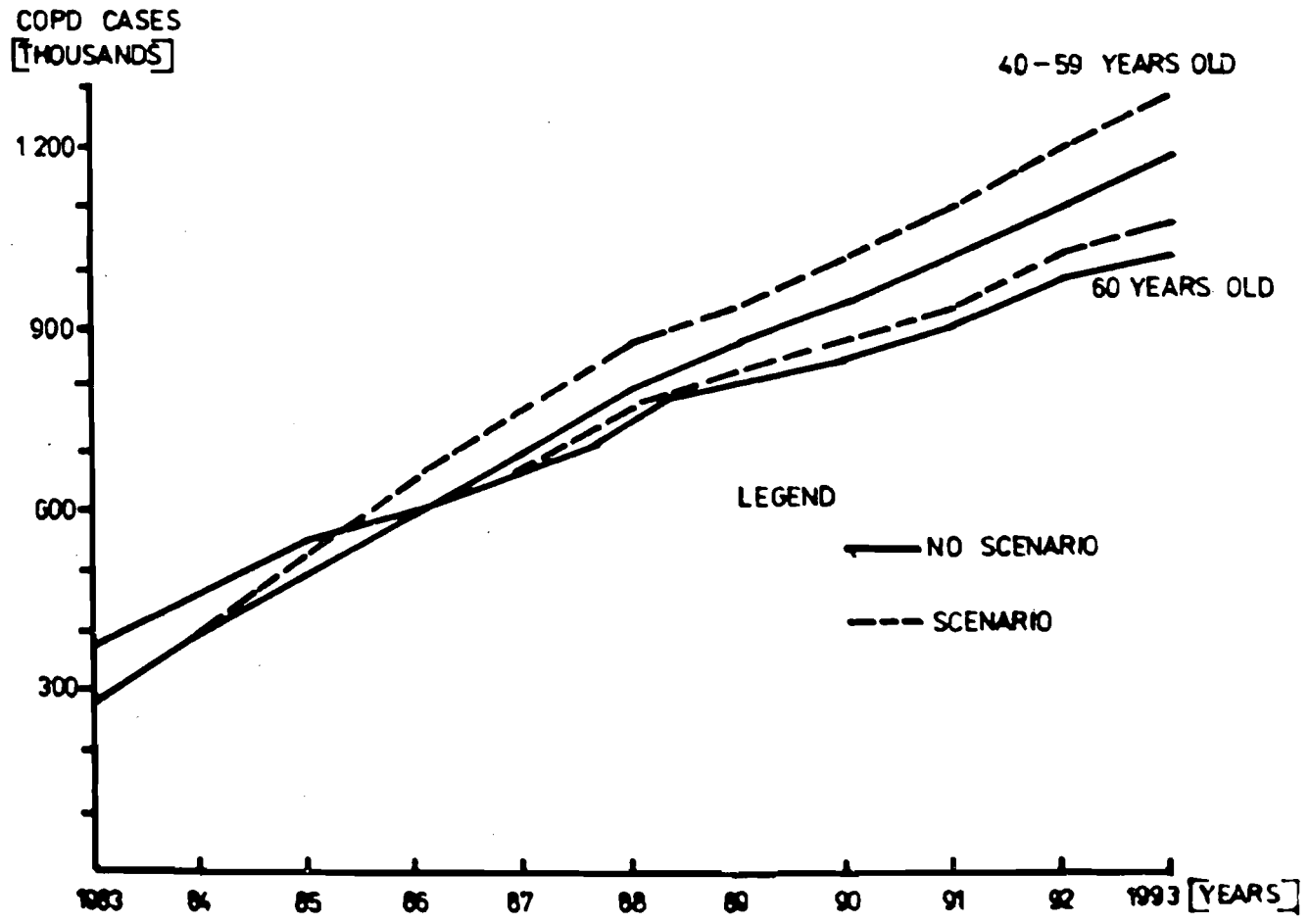

COPD CASES
[THOUSANDS]
1200
900
600
300
40-59 YEARS OLD
60 YEARS OLD
LEGEND
NO SCENARIO
SCENARIO
1983
84
85
86
87
88
89
90
91
92
1993
[YEARS]

Figure 8.

- the data aggregation by four age groups seems to us to be too coarse, disaggregation into more age groups and by sex would probably bring better insights into the system dynamics;
- the disaggregation would certainly require more computer memory than 64 K bytes and the use of some compilor seems to be necessary, because of the increased time for computation;
- the interactive, user-friendly mode of the model's operation allows its utilization by those unskilled with computers.

The incomplete information on COPD prevalence and on the transitions between different population groups does not allow to use the traditional model-tuning procedures. The results of modeling, however, were discussed with experts in COPD, whose opinion was that the model is realistic and can help in understanding the mechanisms of COPD development. The new data on various aspects of COPD will allow us to develop a more detailed version of the model.

Based on these facts, the new version of this model is under preparation. The authors hope that it will be of substantial help to other scholars in this field.

REFERENCES

Armitage, P. (1971) *Statistical Methods in Medical Research*. Blackwell Scientific Publications. 504 pp.

Bajan, A. (1968) Choroby Dychacieho Ustrojenstva Medicinsky a Ekonomicky Problem. *Lek. Obzor*. 17(8):493-498.

Bajan, A. (1983) *Bronchitis Chronica*. Osveta. 214 pp.

Bosse, R., D. Sparrow, C.L. Rose, and S.T. Weiss (1981) Longitudinal Effect of Age and Smoking Cessation on Pulmonary Function. *American Review of Respiratory Diseases* 123:378-381.

Boudik, F., F. Herles, V. Teichmann, F. Macholda, F. Horacek, and P.C. Kaufmann (1969a) Vyskyt Chronicke Bronchitidy v Mestskem Obvodu. *Cas. lek. ces.* 108(1):17-19.

Boudik, F., V. Teichmann, K. Novak, and J. Jencovsky (1969b) Vyskyt Vlekle Bronchitidy Medzi Venkovskym Obzvatelsttvem. *Vnitrni lek.* 15(8):736-744.

Burch, G.E. and N.P. DePasquale (1967) Alcoholic Lung Disease--An Hypothesis. *American Heart Journal* 73:147-148.

Cohen, I.B. (1984) Florence Nightingale. *Scientific American* 250(3):128-137.

Coufal, K., J. Palka, and J. Krchakova (1973) Chronicka Bronchitida Medzi Venkovskym Obyvatelstvem v Okresu Blansko. *Studia Pneumol. Phtiseol. Cechoslov.* 33(7):453-464.

CSSR zdravotnictvi (1983) *Ustav Zdrav. Informaci a Statistiky* (Praha). 514 pp.

Dosman, J.A., D.J. Cotton, B.L. Graham, et al. (1981) Sensitivity and Specifity of Early Diagnostic Tests of Lung Function in Smokers. *Chest* 79:6-11.

DHHS (1984) *Surgeon General's Report on the Health Consequences of Smoking* (Washington, D.C.: U.S. Department of Health and Human Services, National Institute of Health).

Ferlinez, R. (1974) *Lungen und Bronchialerkrankungen* (Stuttgart: G. Thieme). 710 pp.

Fletcher, C.M. and R. Peto (1977) The Natural History of Chronic Airflow Obstruction. *British Medical Journal* 1:1645-1648.

Fletcher, C.M., N.L. Jones, B. Burrows, and A.M. Nidden (1964) American Emphysema and Chronic Bronchitis. *American Review of Respiratory Diseases* 96(1):1-13.

Fletcher, C.M., R. Peto, C.M. Tinker, and F.E. Speizer (1976) *The Natural History of Chronic Bronchitis and Emphysema. An Eight Year Study* (Oxford University Press). 271 pp.

Halak, O. and A. Bajan (1976) Sucasny Pohlad na Vyvoj Bronchitidy Dospelych. *Stud. Pneumol. Phtiseol. Cechoslov.* 36(9):595-600.

Hammond, E.C. (1966) Smoking in Relation to the Death Rates of One Million Men and Women. *National Cancer Institute Monographs* (19):127-204.

Herles, F. (1964) Problematika a Zdravotnicky Vyznam Chronicke Bronchitidy. *Prakt. Lek.* 44(19):721-722.

Higgins, T.T. (1973) The Epidemiology of Chronic Respiratory Disease. *Preventive Medicine* 2(2):14-33.

Higgins, M.V. and Y.B. Keller (1970) Predictors of Mortality in the Adult Population of Tecumseh (Respiratory Symptoms, Chronic Respiratory Diseases and Ventilatory Lung Function). *Arch. Environ. Health* 21:418-420.

Hjalmarson, A. and K. Svardsudd (1981) Health Consequences of Giving Up Smoking in a Prospective Population Study of Middle-Aged Swedish Men. *Acta Med. Scand.* 210:93-96.

Holland, W.W. and D.D. Reid (1965) The Urban Factor in Chronic Bronchitis. *Lancet* 1:445.

Holland, W.W., T. Hallit, A. Benett and A. Elliott (1969) Factors Influencing the Onset of Chronic Respiratory Disease. *British Medical Journal* 2:205.

Katriak, M. (1983) Sociologicke Aspekty Tabakismu. *Ustav Zdrav. Vychovy* (Bratislava). 97 pp.

Kirilog, Y. and L. Irnell (1974) The Prevalence of Bronchial Asthma and Chronic Bronchitis in Uppsala. *Sweden. Scand. J. Resp. Dis.* (Supplement) 89:35-40.

Koonce, J., A.I. Yashin, C.J. Walters, and M. Rusnak (1984) *Modeling of Public Health: Call for Interdisciplinary Actions.* CP-84-1 (Laxenburg, Austria: International Institute for Applied Systems Analysis).

Krumpe, P.E., J.M. Cummiskey and G.A. Lillington (1984) Alcohol and the Respiratory Tract. *Medical Clinics of North America* 68(1):201-219.

Kruty, R., E. Hulova, E. Roch, G. Hudakova, and J. Varsnyi (1975) Hromadna Depistaz Chronickych Bronchitid. *Studia Phtiseol. Cech.* 35(4):479-483.

Kubik, A. et al. (1978) Epidemiologie Chronicke Bronchitidy v Praze a Zatci. *Studia Pneumol. Phtiseol. Cech.* 35(9):596-605.

Kühnl, K. (1982) *Migration and Settlement: 16. Czechoslovakia.* RR-82-32 (Laxenburg, Austria: International Institute for Applied Systems Analysis).

Laurel, C.B. and S. Eriksson (1963) The Electrophoretic Alpha-1 Globulin Pattern of Serum in Alpha-1 Antitrypsin Deficiency. *Scand. J. Clin. Lab. Invest.* 15:132-140.

Lebowitz, M.D. (1981) Respiratory Symptoms and Disease Related to Alcohol Consumption. *American Review of Respiratory Diseases* 123:16-19.

Mueller, R.E., D.L. Kelbe, J. Plummer, and S.H. Walker (1971) The Prevalence of Chronic Bronchitis, Chronic Airway Obstruction and Respiratory Symptoms in a Colorado City. *American Review of Respiratory Diseases* 103:209-228.

Olejnicek, M., J. Meluzin, A. Pokorny, and D. Tlach (1974) Problematika Vlekleho Zapalu Priedusiek u Mestskeho Obyvatelstva. *Vnitrni lek.* 20(2):116-123.

Orie, N.G.M. and R. van der Lende (1970) *Bronchitis III,* Ch.C. Thomas (Ed.) (FRG: Assen).

Pedrizet, S., O. Strauss, and J. Cooreman (1978) Apport de la Methode Epidemiologique dans la Conaissance des Bronchités Chroniques. *Rev. Pract.* 28(9):653-665.

Pohyb obyvatelstva v SSR (1983) *Slov. Statisticky Urad* (Bratislava). 325 pp.

Press, P. and C. Rufener-Press (1974) Epidemiologie de la Bronchité Chronique. *Dev. Therap.* 34(3):140-144.

Pride, N.B. (1977) Chronic Bronchitis and Emphysema: Recent Trends. *Practitioner* 219(12):640-647.

Protivinski, R. (1968) Morbidität und Mortalität der Chronischen Bronchitis in Verschiedenen Europäischen Ländern. In K.Th. Bopp and T.A. Hertle (eds.) *Chronische Bronchitis* (Stuttgart and New York: F.K. Schattaner). 537 pp.

Rankin, J.G., G.S. Gale, P. Wilkinson et al. (1969) Relationship Between Smoking and Pulmonary Disease in Alcoholism. *American Review of Respiratory Diseases* 99:390-398.

Reid, D.D. (1969) The Beginning of Bronchitis. *Proc. R. Soc. Med.* 62:311.

Reid, D.D. and C.M. Fletcher (1971) International Studies in Chronic Respiratory Disease. *British Medical Bulletin* 27(59).

Schmidt, P.O. (1979) Fazit für Praxis. In *Obstructive Atemwegserkrankungen* (G. Witztrock). 159 pp.

Shigan, E.N. (1977) *Alternative Analysis of Different Methods for Estimating Prevalence Rate.* RR-77-40 (Laxenburg, Austria: International Institute for Applied Systems Analysis).

Snider, G.L. (1984) Two Decades of Research in the Pathogenesis of Emphysema. *Schweiz. med. Wschr.* 114:898-906.

Snider, G.L. and A.L. Korthy (1978) Internal Surface Area and Number of Respiratory Air Spaces in Edlastase-Induced Emphysema in Hamsters. *American Review of Respiratory Diseases* 117:685-693.

Stejskal, J., J. Kaupa, and M. Sedlak (1983) The Occurrence of Chronic Bronchitis in a Group of Inhabitants of District Prievidza. *Studia Pneum. Phtiseol. Cech.* 43(4):244-251.

Stoto, M. (1985) *Changes in Adult Smoking Behavior in the United States: 1955 to 1983.* WP-85-53 (Laxenburg, Austria: International Institute for Applied Systems Analysis).

Sylla, A. (1978) *Lungenkrankheiten Bd. i.* (Leipzig: G. Thieme). 513 pp.

Tager, J. and F. Speizer (1975) Role of Infection in Chronic Bronchitis. *England Journal of Medicine* 292(13):563-571.

Tashkin, D.P. et al. (1984) The UCLA Population Studies of COPD. *American Review of Respiratory Diseases* 130:707-715.

Virsik, K., A. Bajan, l. Badalik, M. Vagac, and S. Litomericky (1976) Chronicka Bronchitida v Podunajskej Oblasti. *Studia Pneumol. Phtiseol. Cechoslova.* 36(7):447-452.

Vyslouzil, Z. et al. (1975) Vyskyt Chronicke bronchitidy u Maldistvych. *Studia Pneumol. Phtiseol. Cech.* 35(9):591-595.

Warner, K.E (1983) Reactions to Perceived Risk: Changes in the Behavior of Cigarette Smokers. In V.T. Carello, W.B. Flamm, J.V. Rodricks, and R.G. Tardiff (Eds.) *The Analysis of Actual Versus Perceived Risks* (New York: Pleinum Press). 262 pp.

WHO (1979) *Controlling the Smoking Epidemics* Technical Report Series, No. 636 (Geneva: World Health Organization). 87 pp.

WHO (1980) *Early Detection of Chronic Lung Diseases.* Report on a Meeting (Copenhagen: World Health Organization). 32 pp.

WHO (1982) *Vital Statistics and Causes of Death* (Geneva: World Health Organization). 471 pp.

Yarnell, J.W.G. and A.S. Leger (1981) Respiratory Infections and their Influence on Lung Function in Children: A Multiple Regression Analysis. *Thorax* 36:847-851.

Zacek, A. (1984) *Metody studia Zdravi a Nemoci v Populaci* (Avicenum). 402 pp.

Appendix: Program Listing

```
]LIST

5  REM  NEW VERSION OF COPD PROGR
     AM
10  REM  IIASA NOVEMBER 26,1984
20  DIM P(21,4),M1(4),M2(4),N5(21
     ,4),U(20)
30  DIM L1(4,4),N(4,4),R1(3,4),N1
     (21,4),N2(21,4),N3(21,4),N4(
     21,4)
31  DIM D4(4),M(4)
33  DIM MX(4),RX(3,4),LX(4)
35  FOR I = 1 TO 4:MX(I) = 100:LX
     (I) = 100: NEXT I
36  FOR I = 1 TO 3: FOR J = 1 TO
     4:RX(I,J) = 100: NEXT J: NEXT
     I
40 A =  FRE (0): PRINT "VOLNA PAM
     AT:";A
50  REM  ------------------------
     ---
51  REM  P(T,J) SLOVAK POPULATION
      BY AGE
52  REM  B  NUMBER OF NEWBORNS IN
      SSR 1983
53  REM  M1(J) COPD MORTALITY IN
     SSR 1983
54  REM  M2(J) GENERAL MORTALITY
     WITHOUT
55  REM         COPD CASES IN SSR,
     1983
56  REM  U(T) % OF CHANGE IN MORT
     ALITY
57  REM        IN FORCASTED YEARS
58  REM  N(I,J) PROPORTION OF SLO
     VAK POPU
59  REM          LATION IN RISK
60  REM     I=1 WITHOUT RISK
61  REM     I=2 CIGARETTES SMOKING

62  REM     I=3 LIVING/WORKING IN
     AIR-POLL
63  REM         UTED ENVIRONMENT
64  REM     I=4 FREQUENT UPPER-AIR
     WAYS IN
65  REM         FECTIONS
66  REM  R1(I,J) COEF.OF TRANSITI
     ON FROM
67  REM           GROUP WITHOUT AN
     Y RISK
68  REM           TO THE RISK GROU
     PS
```

```
69  REM     I=1 SMOKING
70  REM     I=2 INFECTIONS
71  REM     I=3 AIR-POLLUTION
72  REM  L1(I,J)RISK OF GETTING S
     ICK FROM
73  REM          COPD FOR PEOPLE
74  REM     I=1 WITHOUT RISK
75  REM     I=2 CIGARETTES SMOKING

76  REM     I=3 INFECTIONS
77  REM     I=4 AIR-POLLUTION
78  REM  N1(T,J) FORECAST OF PEOP
     LE WITHO
79  REM           OUT RISK
80  REM  N2(T,J) FORECAST OF SMOK
     ERS IN
81  REM           SSR
82  REM  N3(T,J) FORECAST OF PEOP
     LE WITH
83  REM           FREQUENT INFECTI
     ONS
84  REM  N4(T,J) FORECAST OF PEOP
     LE IN
85  REM           AIR-POLLUTION
86  REM  T TIME OF FORECAST IN YE
     ARS
87  REM  J AGE GROUPS:0-19;20-39;
     40-59;
88  REM                 60&MORE
90  REM  MX(I) I=4 FACTOR TO CHAN
     GE MORTALITY
91  REM  RX(I) I=4 FACTOR TO CHAN
     GE TRANSITION INTO RISK GROU
     PS
92  REM  LX(I) I=4 FACTOR TO CHAN
     GE TRANSITION INTO GROUP OF
     ILL
100  REM ----------------------
     ---------
105  PRINT "TEST SCENARIOS?(Y/N)"
     ;: INPUT SC$: IF  MID$ (SC$,
     1,1) = "Y" THEN  GOSUB 4000
110  DATA  1736,1619,1070,731: REM
      SLOVAK POPULATION IN 1983 I
     N THOUSANDS
120  FOR I = 1 TO 4: READ P(1,I):
     P(1,I) = P(1,I) * 1000: NEXT
     I
122 N1(1,1) = P(1,1) * 0.51: FOR
     I = 2 TO 4:N1(1,I) = P(1,I) *
     0.34: NEXT I: REM  INITIAL N
     UMBER OF PEOPLE WITHOUT ANY
     RISK
125  REM PRINT TAB( 7);P(1,1); TA
     B(              22);P(1,2); T
     AB( 38);P(1,3);
     TAB( 48);P(1,4)
```

```
130 B = 112750: REM  #OF BIRTH IN
     1983
140  REM  M1-#OF COPD DEATHS;M2-#
    TOTAL # OF DEATH WITHOUT COP
    D CASES
150  DATA  167,96,336,1663: REM
    #OF COPD DEATHS
160  FOR I = 1 TO 4: READ M1(I):D
    5(I) = M1(I):M1(I) = M1(I) /
    P(1,I): NEXT I
161  REM  D5(I) - NUMBER OF DEATH
    S FROM COPD IN AGE GROUPS
170  DATA  3342,1912,6735,33269: REM
     NUMBER OF ALL DEATHS
180  FOR I = 1 TO 4: READ M2(I):D
    4(I) = M2(I):M2(I) = M2(I) /
    P(1,I):M(I) = M2(I) - M1(I):
     NEXT I
190  REM  -----CYCLE TO COUNT POP
    ULATION FORECAST--------
192  DATA 0.99,1,1,1.01,1.01,1.02
    ,1.02,1.02,1.02,1.03,1.03,1.
    03,1.03,1.04,1.04,1.04,1.04,
    1.04,1.04,1.04
194  FOR I = 1 TO 20: READ U(I): NEXT
    I: REM  %OF MORTALITY DEVELO
    PMENT FORECAST
200  FOR J = 2 TO 21: REM  20 YEA
    RS FORECAST
202  IF MY = J THEN  FOR K = 1 TO
    4:M1(K) = M1(K) * MX / 100: NEXT
    K
210 P(J,1) = P(J - 1,1) + B - (0.
    05 * P(J - 1,1)) - (M2(1) *
    U(J - 1) + (M1(1) * X / 100)
    ) * P(J - 1,1): REM   POPULA
    TION IN AGE 0-19
220  FOR K = 2 TO 3: REM  FOR THE
     2ND AND THE 3RD AGE GROUP
230 P(J,K) = P(J - 1,K) + (0.05 *
    P(J - 1,K - 1)) - (0.05 * P(
    J - 1,K)) - (M2(K) * U(J - 1
    ) + M1(K)) * P(J - 1,K): NEXT
    K
240 P(J,4) = P(J - 1,4) + (0.05 *
    P(J - 1,3)) - (M2(4) * U(J -
    1) + M1(4) * X / 100) * P(J -
    1,4): REM    POPULATION IN A
    GE 60 & OVER
242  REM PRINT TAB( 7);P(J,1); TA
    B(          22);P(J,2); T
    AB( 38);P(J,3);
    TAB( 48);P(J,4)
250  NEXT J
260  DATA   143,298 ,276,382: REM
    #OF COPD CASESIN SSR IN 1983
```

```
270  FOR I = 1 TO 4: READ N5(1,I)
     :N5(1,I) = N5(1,I) * 1000: NEXT
     I
280  DATA  0.02,0.1,0.2,0.25: REM
      RISK OF GETTING SICK FROM T
     HE GROUP OF HEALTHY
290  DATA  0.05,0.3,0.6,0.75: REM
      RISK OF GETTING SICK FROM S
     MOKING
300  DATA  0.1,0.2,0.25,0.3: REM
      RISK OF GETTING SICK FROM A
     IR-POLLUTION
310  DATA  0.39,0,0,0: REM  RISK
     OF GETTING SICK FROM REPEATE
     D RESPIRATORY INFECTIONS IN
     CHILDHOOD
320  FOR I = 1 TO 4: FOR J = 1 TO
     4: READ L1(I,J): NEXT J: NEXT
     I
329  DATA  0.88,0.52,0.42,0.14
330  DATA  0.164,0.5327,0.5848,0.
     4938: REM  PROPORTION OF POP
     ULATION WITH SMOKING
340  DATA  0.31,0.31,0.31,0.32: REM
      PROPORTION OF POPULATION UN
     DER AIR-POLLUTION
350  DATA  0.30,0,0,0: REM  PROPO
     RTION OF POPULATION WITH FRE
     QUENT RESPIRATORY DISEASES I
     N CHILDHOOD
356  FOR K = 1 TO 4: READ X(K): NEXT
     K
360  FOR I = 1 TO 3: FOR J = 1 TO
     4: READ N(I,J)
362 N(I,J) = N(I,J) * X(J)
364  NEXT J: NEXT I
370  FOR J = 1 TO 4:N2(1,J) = N(1
     ,J) * P(1,J):N3(1,J) = N(2,J
     ) * P(1,J):N4(1,J) = N(3,J) *
     P(1,J): NEXT J: REM  ABSOLUT
     EPLE IN RISK
379  DATA  50,30,30,30
380  DATA  0.69,0.06,0.09,0.04: REM
      COEF.OF TRANSITION FROM GRO
     UP WITHOUT SMOKING TO SMOKIN
     G
390  DATA  0.0005,0.002,0.002,0.0
     055: REM  COEF.OF TRANSITION
      FROM GROUP WITHOUT RISK TO
     AIR-POLLUTION
400  DATA  0.08,0,0,0: REM  COEF.
     OF TRANSITION FROM GROUP WIT
     HOUT RISK TO NURSERY
405  FOR K = 1 TO 4: READ X(K): NEXT
     K
```

```
410  FOR I = 1 TO 3: FOR J = 1 TO
     4: READ R1(I,J)
412  IF I = 2 THEN R1(I,J) = R1(I
     ,J) * X(J)
418  NEXT J: NEXT I
500  REM  ------# OF SICK,# OF PE
     OPLE IN RISK-------------
510  FOR T = 2 TO 21: REM   YEARS

511  IF LY = T THEN  FOR K1 = 1 TO
     4: FOR K2 = 1 TO 4:L1(K1,K2)
      = L1(K1,K2) * LX(K2) / 100:
      NEXT K2: NEXT K1
514  IF RY = T THEN  FOR K1 = 1 TO
     3: FOR K2 = 1 TO 4:R1(K1,K2)
      = R1(K1,K2) * RX(K1,K2) / 1
     00: NEXT K2: NEXT K1
520  FOR J = 1 TO 4: REM  AGE GRO
     UPS
530 R = R1(1,J) + R1(2,J) + R1(3,
     J)
531 P = B: IF J > 1 THEN P = 0.05
      * N1(T - 1,J - 1)
533 N1(T,J) = N1(T - 1,J) + P - (
     R + L1(1,J)) * N1(T - 1,J) -
     (0.05 * N1(T - 1,J)) - (M2(J
     ) * N1(T - 1,J))
534  IF J = 2 THEN N1(T,J) = N1(T
     ,J) + 0.05 * N4(T - 1,J): REM
      RECOVERING FROM RESP.DISEAS
     ES
536  IF J = 1 THEN P = 0
537  IF J > 1 THEN P = 0.05 * N2(
     T - 1,J - 1)
538  IF J = 2 THEN P = P + 0.05 *
     N4(T - 1,J - 1)
540 N2(T,J) = N2(T - 1,J) + R1(1,
     J) * N1(T,J) + P - (L1(2,J) *
     N2(T - 1,J)) - (0.05 * N2(T -
     1,J)) - (M(J) * N2(T - 1,J))

542  IF J > 1 THEN P = 0.05 * N3(
     T - 1,J - 1)
550 N3(T,J) = N3(T - 1,J) + R1(2,
     J) * N1(T,J) + P - (L1(3,J) *
     N3(T - 1,J)) - (0.05 * N3(T -
     1,J)) - (M(J) * N3(T - 1,J))
552  IF J > 1 THEN N4(T,J) = 0: GOTO
     562
560 N4(T,J) = N4(T - 1,J) + R1(3,
     J) * N1(T,J) + P - (L1(4,J) *
     N4(T - 1,J)) - (0.05 * N4(T -
     1,J)) - (M(J) * N4(T - 1,J))
```

```
562  IF J > 1 THEN P = 0.05 * N5(
     T - 1,J - 1)
570 N5(T,J) = N5(T - 1,J) + L1(1,
     J) * N1(T - 1,J) + L1(2,J) *
     N2(T - 1,J) + L1(3,J) * N3(T
      - 1,J) + L1(4,J) * N4(T - 1
     ,J) + P - (N5(T - 1,J) * (M1
     (J) + M(J)))
600  NEXT J: NEXT T
610  HOME
620  PRINT "PRESS";: INVERSE : PRINT
     " G ";: NORMAL : PRINT "IF Y
     OU PREFER RESULTS IN GRAPHS"

630  INPUT A$:PP$ =  MID$ (A$,1,1
     ): IF PP$ = "G" THEN  GOTO 1
     500
1000  REM -------PRINTOUTS-------
     --
1001  PR# 1: PRINT
1010  PRINT  TAB( 11)"I N P U T
     D A T A": PRINT  TAB( 11);"F
     OR SLOVAKIA 1983": FOR I = 1
      TO 40: PRINT "^";: NEXT I: PRINT
     : PRINT : PRINT
1011  PRINT "NOTE": PRINT  TAB( 6
     );"RISK GROUP#1 - WITHOUT RI
     SK": PRINT  TAB( 17);"#2 - S
     MOKERS": PRINT  TAB( 17);"#3
      - AIR - POLLUTION ": PRINT
      TAB( 17);"#4 - RESPIRATORY
     INFECTIONS":
1012  PRINT : PRINT : PRINT  TAB(
     10);"P O P U L A T I O N": FOR
     I = 1 TO 40: PRINT ".";: NEXT
     I:: PRINT : PRINT
1014  PRINT  TAB( 6);"0 - 19"; TAB(
     17);"20 - 39"; TAB( 29);"40
     - 59"; TAB( 39);"   60 -"
1015  PRINT  TAB( 4);P(1,1); TAB(
     15);P(1,2); TAB( 27);P(1,3);
      TAB( 39);P(1,4)
1016  PRINT : PRINT : PRINT "NUMB
     ER OF N E W B O R N S:";B: PRINT
     : PRINT
1017  PRINT  TAB( 11);"M O R T A
     L I T Y": PRINT  TAB( 11);"W
     ITHOUT COPD CASES": FOR I =
     1 TO 40: PRINT ".";: NEXT I:
      PRINT
1018  PRINT  TAB( 6);"0 - 19"; TAB(
     17);"20 - 39"; TAB( 29);"40
     - 59"; TAB( 39);"   60 -"
1019  PRINT  TAB( 4);D5(1); TAB(
     15);D5(2); TAB( 27);D5(3); TAB(
     39);D5(4)
```

```
1020  PRINT : PRINT : PRINT  TAB(
     11);"M O R T A L I T Y": PRINT
      TAB( 11);"SPECIFIC FOR COPD
     ": FOR I = 1 TO 40: PRINT ".
     ";: NEXT I: PRINT
1021  PRINT  TAB( 6);"0 - 19"; TAB(
     17);"20 - 39"; TAB( 29);"40
     - 59"; TAB( 39);"   60 -"
1022  PRINT  TAB( 4);D4(1); TAB(
     15);D4(2); TAB( 27);D4(3); TAB(
     39);D4(4): PRINT : PRINT
1023  PRINT  TAB( 16);"R I S K": PRINT
      TAB( 12);"OF GETTING SICK":
      FOR I = 1 TO 40: PRINT ".";
     : NEXT I: PRINT : PRINT
1025  PRINT "RISK"; TAB( 6);"0 -
     19"; TAB( 17);"20 - 39"; TAB(
     29);"40 - 59"; TAB( 39);"
     60 -"
1027  PRINT "#1"; TAB( 4);L1(1,1)
     ; TAB( 15);L1(1,2); TAB( 27)
     ;L1(1,3); TAB( 39);L1(1,4)
1029  PRINT "#2"; TAB( 4);L1(2,1)
     ; TAB( 15);L1(2,2); TAB( 27)
     ;L1(2,3); TAB( 39);L1(2,4)
1030  PRINT "#3"; TAB( 4);L1(3,1)
     ; TAB( 15);L1(3,2); TAB( 27)
     ;L1(3,3); TAB( 39);L1(3,4)
1031  PRINT "#4"; TAB( 4);L1(4,1)
     ; TAB( 15);L1(4,2); TAB( 27)
     ;L1(4,3); TAB( 39);L1(4,4): PRINT
     : PRINT
1035  PRINT  TAB( 3);"P R O P O R
      T I O N  OF POPULATION": PRINT
      TAB( 17);"IN RISK": FOR I =
     1 TO 40: PRINT ".";: NEXT I:
      PRINT : PRINT
1037  PRINT "RISK"; TAB( 6);"0 -
     19"; TAB( 17);"20 - 39"; TAB(
     29);"40 - 59"; TAB( 39);"60
     -"
1039  PRINT "#2"; TAB( 4);N(1,1);
      TAB( 15);N(1,2); TAB( 27);N
     (1,3); TAB( 39);N(1,4)
1040  PRINT "#3"; TAB( 4);N(2,1);
      TAB( 15);N(2,2); TAB( 27);N
     (2,3); TAB( 39);N(2,4)
1041  PRINT "#4"; TAB( 4);N(3,1);
      TAB( 15);N(3,2); TAB( 27);N
     (3,3); TAB( 39);N(3,4)
1042  PRINT : PRINT
1045  PRINT  TAB( 6);"COEF.OF T R
      A N S I T I O N": PRINT "FR
     OM GROUP WITHOUT RISK TO ONE
      WITH RISK": FOR I = 1 TO 40
     : PRINT ".";: NEXT I: PRINT
     : PRINT
```

```
1047  PRINT "RISK"; TAB( 6);"0 -
      19"; TAB( 17);"20 - 39"; TAB(
      29);"40 - 59"; TAB( 39);"60
      -"
1048  PRINT "#2"; TAB( 4);RX(1,1)
      ; TAB( 15);RX(1,2); TAB( 27)
      ;RX(1,3); TAB( 39);RX(1,4)
1049  PRINT "#3"; TAB( 4);R1(2,1)
      ; TAB( 15);R1(2,2); TAB( 27)
      ;R1(2,3); TAB( 39);R1(2,4)
1050  PRINT "#4"; TAB( 4);R1(3,1)
      ; TAB( 15);R1(3,2); TAB( 27)
      ;R1(3,3); TAB( 39);R1(3,4): PRINT
      : PRINT
1051  PRINT : PRINT : PRINT  TAB(
      2);"C O P D  M O R B I D I T
       Y";: FOR I = 1 TO 40: PRINT
      ".";: NEXT I: PRINT
1053  PRINT  TAB( 6);"0 - 19"; TAB(
      17);"20 - 39"; TAB( 29);"40
      - 59"; TAB( 39);"   60 -"
1055  PRINT  TAB( 4);N5(1,1); TAB(
      15);N5(1,2); TAB( 27);N5(1,3
      ); TAB( 39);N5(1,4)
1057  FOR I = 1 TO 40: PRINT "^";
      : NEXT I: PRINT : PRINT : PRINT

1060  IF SC$ <  > "Y" THEN 1110
1062  PRINT  TAB( 5)"S C E N A R
      I O  T E S T E D :"
1064  PRINT  TAB( 3);"CHANGE IN T
      REATMENT EFFECTIVENESS:"
1066  PRINT  TAB( 6);"0 - 19"; TAB(
      17);"20 - 39"; TAB( 29);"40
      - 59"; TAB( 39);"   60 -"
1068  PRINT  TAB( 4);MX(1); TAB(
      15);MX(2); TAB( 27);MX(3); TAB(
      39);MX(4)
1069  PRINT "YEAR OF CHANGE:";MY:
       PRINT : PRINT
1070  PRINT  TAB( 09);"CHANGE IN
      RISK FACTORS"
1072  PRINT "RISK"; TAB( 6);"0 -
      19"; TAB( 17);"20 - 39"; TAB(
      29);"40 - 59"; TAB( 39);"60
      -"
1075  PRINT "#3"; TAB( 4);RX(2,1)
      ; TAB( 15);RX(2,2); TAB( 27)
      ;RX(2,3); TAB( 39);RX(2,4)
1076  PRINT "#4"; TAB( 4);RX(3,1)
      ; TAB( 15);RX(3,2); TAB( 27)
      ;RX(3,3); TAB( 39);RX(3,4):
1078  PRINT "YEAR OF CHANGE:";RY
1080  PRINT : PRINT
1082  PRINT  TAB( 10);"CHANGE IN
      PREVENTION:"
```

```
1084  PRINT  TAB( 6);"0 - 19"; TAB(
      17);"20 - 39"; TAB( 29);"40
      - 59"; TAB( 39);"    60 -"
1086  PRINT  TAB( 4);LX(1); TAB(
      15);LX(2); TAB( 27);LX(3); TAB(
      39);LX(4)
1088  PRINT "YEAR OF CHANGE:";LY
1090  PRINT : PRINT : PRINT
1110  PRINT  TAB( 3);"FORECASTS O
      F PEOPLE UNDER DIFFERENT"
1112  PRINT  TAB( 10);"H E A L T
      H  R I S K"
1114  FOR I = 1 TO 40: PRINT "^";
      : NEXT I
1116  PRINT : PRINT : PRINT
1117  PRINT  TAB( 13);"W I T H O
      U T": PRINT : PRINT
1118  PRINT "YE"; TAB( 15);"AGE G
      ROUPS"
1120  PRINT "AR"; TAB( 6);"0 - 19
      "; TAB( 17);"20 - 39"; TAB(
      29);"40-59"; TAB( 39);"   60
       -"
1122  FOR I = 1 TO 49: PRINT ".";
      : NEXT I: PRINT
1200  FOR T = 1 TO 21
1210  PRINT T - 1; TAB( 4);N1(T,1
      ); TAB( 15);N1(T,2); TAB( 27
      );N1(T,3); TAB( 39);N1(T,4)
1220  NEXT T
1222  PRINT : PRINT : PRINT  TAB(
      8);"CIGARETTE  S M O K I N G
      ": PRINT : PRINT
1224  PRINT "YE"; TAB( 15);"AGE G
      ROUPS"
1226  PRINT "AR"; TAB( 6);"0 - 19
      "; TAB( 17);"20 - 39"; TAB(
      29);"40-59"; TAB( 39);"   60
       -"
1228  FOR I = 1 TO 49: PRINT ".";
      : NEXT I: PRINT
1230  FOR T = 1 TO 21
1232  PRINT T - 1; TAB( 4);N2(T,1
      ); TAB( 15);N2(T,2); TAB( 27
      );N2(T,3); TAB( 39);N2(T,4)
1234  NEXT T
1240  PRINT : PRINT : PRINT  TAB(
      7);"A I R - P O L L U T I O
      N": PRINT : PRINT
1242  PRINT "YE"; TAB( 15);"AGE G
      ROUPS"
1244  PRINT "AR"; TAB( 6);"0 - 19
      "; TAB( 17);"20 - 39"; TAB(
      29);"40-59"; TAB( 39);"   60
       -"
```

```
1246  FOR I = 1 TO 49: PRINT ".";
      : NEXT I: PRINT
1248  FOR T = 1 TO 21
1250  PRINT T - 1; TAB( 4);N3(T,1
      ); TAB( 15);N3(T,2); TAB( 27
      );N3(T,3); TAB( 39);N3(T,4)
1252  NEXT T
1260  PRINT : PRINT : PRINT  TAB(
      5);"FREQ.RESP.I N F E C T I
      O N S": PRINT : PRINT
1262  PRINT "YE"; TAB( 15);"AGE G
      ROUPS"
1264  PRINT "AR"; TAB( 6);"0 - 19
      "; TAB( 17);"20 - 39"; TAB(
      29);"40-59"; TAB( 39);"   60
       -"
1266  FOR I = 1 TO 49: PRINT ".";
      : NEXT I: PRINT
1268  FOR T = 1 TO 21
1270  PRINT T - 1; TAB( 4);N4(T,1
      ); TAB( 15);N4(T,2); TAB( 27
      );N4(T,3); TAB( 39);N4(T,4)
1272  NEXT T
1280  PRINT : PRINT : PRINT  TAB(
      14);"FORECAST OF": PRINT  TAB(
      7);"C O P D  M O R B I D I T
       Y"
1282  FOR I = 1 TO 40: PRINT "^";
      : NEXT I: PRINT : PRINT
1284  PRINT "YE"; TAB( 15);"AGE G
      ROUPS"
1286  PRINT "AR"; TAB( 6);"0 - 19
      "; TAB( 17);"20 - 39"; TAB(
      29);"40-59"; TAB( 39);"   60
       -"
1288  FOR I = 1 TO 49: PRINT ".";
      : NEXT I: PRINT
1290  FOR T = 1 TO 21
1292  PRINT T - 1; TAB( 4);N5(T,1
      ); TAB( 15);N5(T,2); TAB( 27
      );N5(T,3); TAB( 39);N5(T,4)
1294  NEXT T
1300  PRINT : PRINT : PRINT  TAB(
      14);"FORECAST OF": PRINT  TAB(
      5);"P O P U L A T I O N  I N
        S S R"
1302  FOR I = 1 TO 40: PRINT "^";
      : NEXT I: PRINT : PRINT
1304  PRINT "YE"; TAB( 15);"AGE G
      ROUPS"
1306  PRINT "AR"; TAB( 6);"0 - 19
      "; TAB( 17);"20 - 39"; TAB(
      29);"40-59"; TAB( 39);"   60
       -"
1308  FOR I = 1 TO 49: PRINT ".";
      : NEXT I: PRINT
```

```
1310  FOR T = 1 TO 21
1312  PRINT T - 1; TAB( 4);P(T,1)
     ; TAB( 15);P(T,2); TAB( 27);
     P(T,3); TAB( 39);P(T,4)
1314  NEXT T
1400  REM FORI = 1 TO 4: PRINT N1
     (20              ,I) + N2(20,I
     ) + N3(20,I) +              N
     4(20,I) + N5(20,I): NEXT I
1499  GOTO 2000
1500  REM
1505 MX = 0:MN = 200000000
1510  FOR T = 1 TO 21: FOR J = 1 TO
     4:P(T,J) =  INT (P(T,J) / 10
     0000)
1515  IF P(T,J) > MX THEN MX = P(
     T,J)
1520  IF P(T,J) < MN THEN MN = P(
     T,J)
1525  NEXT J: NEXT T
1530 D = 75 / MX
1539 MX = 0:MN = 2000000
1540  FOR T = 1 TO 21: FOR J = 1 TO
     4:N2(T,J) =  INT (N2(T,J) /
     10000)
1541  IF N2(T,J) > MX THEN MX = N
     2(T,J)
1542  IF N2(T,J) < MN THEN MN = N
     2(T,J)
1545  NEXT J: NEXT T
1546 D1 = 75 / MX
1549 MX = 0
1550  FOR T = 1 TO 21: FOR J = 1 TO
     4:N5(T,J) =  INT (N5(T,J) /
     100000)
1551  IF N5(T,J) > MX THEN MX = N
     5(T,J)
1555  NEXT J: NEXT T
1556 D2 = 75 / MX
1600  HGR : HCOLOR= 3: ROT= 0: SCALE=
     1
1610  HPLOT 0,0 TO 0,75 TO 130,75
      TO 130,0 TO 0,0
1620  HPLOT 149,0 TO 149,75 TO 27
     9,75 TO 279,0 TO 149,0
1630  HPLOT 0,84 TO 0,159 TO 130,
     159 TO 130,84 TO 0,84
1700 ZN$ = "POPULATION FORECAST":
     X = 15:Y = 7: GOSUB 1800
1710 ZN$ = "SMOKERS":X = 174:Y =
     7: GOSUB 1800
1720 ZN$ = "COPD CASES":X = 15:Y =
     91: GOSUB 1800
1790  GOTO 1900
1800  FOR I1 = 1 TO  LEN (ZN$):II
      =  ASC ( MID$ (ZN$,I1,1)) -
     31: IF II < 1 THEN II = 1
```

```
1810  DRAW II AT X + 6 * I1,Y: NEXT
     I1: RETURN
1900  FOR T = 1 TO 21: FOR J = 1 TO
     4
1905  IF J = 1 THEN  HCOLOR= 2
1906  IF J = 2 THEN  HCOLOR= 3
1907  IF J = 3 THEN  HCOLOR= 5
1908  IF J = 4 THEN  HCOLOR= 6
1910 X1 = (T - 1) * 6:X2 = T * 6:
     Y1 = 75 - (P(T - 1,J) * D):Y
     2 = 75 - (P(T,J) * D)
1950  HPLOT X1,Y1 TO X2,Y2
1960 X1 = 149 + (T - 1) * 6:X2 =
     T * 6 + 149:Y1 = 75 - (N2(T -
     1,J) * D1):Y2 = 75 - (N2(T,J
     ) * D1)
1963  HPLOT X1,Y1 TO X2,Y2
1970 X1 = (T - 1) * 6:X2 = T * 6:
     Y1 = 159 - (N5(T - 1,J) * D2
     ):Y2 = 159 - (N5(T,J) * D2)
1975  HPLOT X1,Y1 TO X2,Y2
1980  NEXT J: NEXT T
2000  PR# 0
2001  GOTO 10000
4000  REM  PREPAIR SCENARIOS
4001  HOME : PRINT : PRINT : PRINT
     : PRINT
4010  PRINT "FOLLOWING SCENARIOS
     ARE TO TEST:"
4011  PRINT  TAB( 15);"CHANGE IN
     TREATMENT EFFECTIVENESS"
4012  PRINT  TAB( 15);"CHANGE IN
     RISK FACTORS"
4013  PRINT  TAB( 15);"CHANGE IN
     PREVENTION"
4030  PRINT "ENTER % OF CHANGE OF
      TREATMENT:"
4031  PRINT " 0-19:";: INPUT A$: IF
     A$ = "" THEN 4034
4032 MX(1) =  VAL ( MID$ (A$,1,3)
     )
4034  PRINT "20-39:";: INPUT A$: IF
     A$ = "" THEN 4038
4036 MX(2) =  VAL ( MID$ (A$,1,3)
     )
4038  PRINT "40-60:";: INPUT A$: IF
     A$ = "" THEN 4042
4039 MX(3) =  VAL ( MID$ (A$,1,3)
     )
4042  PRINT "60- :";: INPUT A$: IF
     A$ = "" THEN 4050
4043 MX(4) =  VAL ( MID$ (A$,1,3)
     )
4045  PRINT "ENTER YEAR OF CHANGE
      (1-20)";: INPUT MY: IF MY >
     20 THEN 4045
```

```
4049  PRINT : PRINT
4050  PRINT "ENTER % OF CHANGE IN
      SMOKING HABITS:"
4051  PRINT " 0-19:";: INPUT A$: IF
     A$ = "" THEN 4054
4052 RX(1,1) =  VAL ( MID$ (A$,1,
     3))
4054  PRINT "20-39:";: INPUT A$: IF
     A$ = "" THEN 4058
4056 RX(1,2) =  VAL ( MID$ (A$,1,
     3))
4058  PRINT "40-60:";: INPUT A$: IF
     A$ = "" THEN 4062
4059 RX(1,3) =  VAL ( MID$ (A$,1,
     3))
4062  PRINT "60- :";: INPUT A$: IF
     A$ = "" THEN 4070
4063 RX(1,4) =  VAL ( MID$ (A$,1,
     3))
4070  PRINT "ENTER % OF CHANGE IN
      AIR-POLLUTION:"
4071  PRINT " 0-19:";: INPUT A$: IF
     A$ = "" THEN 4074
4072 RX(2,1) =  VAL ( MID$ (A$,1,
     3))
4074  PRINT "20-39:";: INPUT A$: IF
     A$ = "" THEN 4078
4076 RX(2,2) =  VAL ( MID$ (A$,1,
     3))
4078  PRINT "40-60:";: INPUT A$: IF
     A$ = "" THEN 4082
4079 RX(2,3) =  VAL ( MID$ (A$,1,
     3))
4082  PRINT "60-  :";: INPUT A$: IF
     A$ = "" THEN 4090
4083 RX(2,4) =  VAL ( MID$ (A$,1,
     3))
4089  PRINT : PRINT
4090  PRINT "ENTER % OF CHANGE IN
      FREQ.RESP.DIS.IN CHILDHOOD:
     "
4091  PRINT " 0-19:";: INPUT A$: IF
     A$ = "" THEN 4105
4092 RX(3,1) =  VAL ( MID$ (A$,1,
     3))
4105  PRINT "ENTER YEAR OF CHANGE
      (1-20):";: INPUT RY: IF RY >
     20 THEN 4105
4110  PRINT "ENTER % OF CHANGE IN
      EFFECTIVENESS": PRINT " IN
     PREVENTION"
4111  PRINT " 0-19:";: INPUT A$: IF
     A$ = "" THEN 4114
4112 LX(1) =  VAL ( MID$ (A$,1,3)
     )
```

```
4114  PRINT "20-39:";: INPUT A$: IF
     A$ = "" THEN 4118
4116 LX(2) =  VAL ( MID$ (A$,1,3)
     )
4118  PRINT "40-60:";: INPUT A$: IF
     A$ = "" THEN 4122
4119 LX(3) =  VAL ( MID$ (A$,1,3)
     )
4122  PRINT "60- :";: INPUT A$: IF
     A$ = "" THEN 4130
4123 LX(4) =  VAL ( MID$ (A$,1,3)
     )
4125  PRINT "ENTER YEAR OF CHANGE
      (1-20)";: INPUT LY: IF LY >
     20 THEN 4125
4130  REM
5000  RETURN
10000  END
```