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THE UNIVERSITY OF ALBERTA

A Demographic Analysis of Cancer Mortality in Canada: 1951-1981

by

Eddie Siu Man Ng

A THESIS

SUBMITTED TO THE FAGULTY OF GRADUATE STUDIES AND RESEARCH

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The undersigned certify that they have read, and recommend to the Faculty of Graduate Studies and Research, for acceptance, a thesis entitled A Demographic Analysis of Cancer Mortality in Canada: 1951-1981 submitted by Eddie Siu Man Ng in partial fulfilment of the requirements for the degree of Master of Arts in Demography.

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Supervisor

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October 17 1986

Date.....

Abstract

There are three objectives in this study. First, to examine the pattern of cancer mortality in Canada from 1951 to 1981. The mortality rates of male and female total cancer, lung, stomach, bladder, intestine, and female breast cancer are being studied using the crude and standardized rates, the geometric mean of age-sex rates, and the two components of cancer mortality rate, namely, the age effect and the cancer force.

Second, to set up statistic models to predict overall cancer death rates in Canada in the upcoming decades. The regression equations for age effect and cancer are constructed for males and females separetly. Estimates from these equations are then combined to predict cancer mortality rates for males and females, respectively. Predictions are computed for the years from 1982 to 2003.

Third, to analyze the factors that could possibily lead to high risk of cancer. A wide range of demographic, socioeconomic, and industrial variables are included in this study. Besides their individual effects, the interactions among these variables on cancer are also considered. The technique of best possible subset regression is used to pick out the most influential variables on cancer mortality.

The results from the trend analysis indicate that to the patterns of cancer mortality depicted by the crude and standardized death rates can be in times decptive. It is father often than not that the trends shown by the geometric

mean are different. The influence of individual cancer site on the overall cancer death rate is revealed, and this raises the question on the applicablity of results from overall cancer analysis to policy implementations.

The age effect and cancer force are better measures to describe trends overtime. Also, they contribute to our better understanding of the two broad elements behind cancer mortality that could be either improving or deteriorating overtime, or both. Their changes indicate the direction for policy formation, whether towards the aging of the population or the worsening of cancer induction factors.

Estimates of the age effect and cancer force show different patterns for the two sexes. Mortality predictions are in general satisfactory, and estimates for 1982 are particularly close to the reported death rates. Similar task can be done for specific cancer sites.

The demographic-socioeconomic analysis does not show the same income and education effect on male and female cancer, and they have rather strong interactive effect through other variables. The results do not comply totally with other studies. Among all ethnic groups, areas with large proportion of French Canadian have the highest impact on most cancer sites. Among the industrial variables, high manufacturing area and Ontario mining display strohg positive impacts. This study reveals the importance of simultaneous evaluation of cancer induction factors, and the need to controll statistically the confounding

relationships among variables in order to expose their true effect on cancer.

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1. Introduction

The level of Canadian mortality has improved substantially over the past decades. Crude death rate for both sexes dropped from a high of 22 per 1000 in 1851 to 6.2 in 1982. One of the immediate rewards of such progress is the remarkable increase in life expectancy for both males and females at all age groups. Between 1931 and 1981, Canadian male and female life expectancies at birth have realized some exciting gains of 12 and 16 years, respectively.

In addition, with life expectancy for both sexes at birth being 74 years in 1981, Canada has surpassed the United States and some European countries in preserving life. Despite the impressive progress, descent in mortality in this nation within the past few decades has been decelerating for quite a long period of time before regaining its strength since the last 10 years of so.

This stagnation of mortality decline, however, is not exclusive to Canada. In fact, this phenomenon is visible throughout the low mortality region, and lately in a few developing countries. Inquiry into this deceleration has traced it origin to the mid-1960's. Supporting evidence reveals that, while life expectancy in the developed regions gained by more than 4 years in 1955-1965, it increased by fewer than 1.6 years in the following decade. Concerns over the causes of such decline have prompted some studies to advocate the closing-in of a biological ceiling upon human longevity. Most researches, nevertheless, has stressed the

importance of epidemiological transition and the accompanying aging of the population.

The success in socioeconomic developments, improved nutrition, and advances in medical technology have enabled the developed countries to eradicte almost all of their infectious and parasitic diseases. Since these diseases strike primarily during the early stages of life, their control, on the one hand, allows the population to survive until advanced ages. On the other hand, the disappearance of communicable diseases, have altered the structure of causes of death.

Degenerative diseases, in particular heart disease and malignant neoplasms, have become the major killers. In countries where life expectancy is above 70 years, these two diseases are known to account for some 48 and 21 per cent of the total deaths, respectively. In contrast, only 1% of all deaths are due to infectious diseases. The seriousness of heart disease and cancer is further enchanced by the fact that they attack largely individuals of middle and older ages.

As populations in the developed regions are increasingly aging, it is perhaps not too surprising to record a slow-down in mortality reduction. Moreover, as most infectious diseases have already been brought to their lowest level, it is rather unlikely that suppressing them further will increase life expectancy by any significant margin. To extend life, it is obvious that efforts must be

directed towards the reduction of deaths from degenerative and chronic diseases.

While medical advancements have succeeded to some extent in controling heart disease, primarily by delaying fatality until later in life, most cancers still remain incurable. Given cancer's resitance to current technology, it seems that, unless major medical breakthroughs in treatment occur, human longevity is bounded by a techological limit, if not a biological ceiling.

In order to identify the causes of cancer, many studies have to look beyond biological factors to our exogeneous environment for clues. Findings have causally linked life-style, diet, occupational hazard, and environmental pollution to some specific cancer sites. Also, unequal risks of cancer are observed among various social strata. Such findings suggest that by improving certain social inequalities, cancer can be prevented to some extent.

Studies from various disciplines have called for changes in life-style, and tighter control over pollution in different environments to reduce cancer risks. However, it would be difficult for a single set of recommendations to be effective without considering individual's socioeconomic standing. After all, one's achievements in the social ladder do determine, to a large extent, one's life-style, diet, and exposure to possible carcinogens.

Given these conditions, there will be greater benefits if the influences on cancer by socioeconomic, demographic

and occupational factors can be analysized for the cancer sites individually. The details and specific information retrieved would be relevant for proper allocation and reallocation of finance and manpower to fight cancer. The purposes of the present study are to analyse cancer mortality trends in Canada for selected cancer sites, to predict future cancer death rates, and to assess the various kinds of influences on, or determinants of cancer mortality simultaneously.

1.1 Thesis Layout

In the first two Chapters, we will review evidence explaining how cancer emerged to become a leading cause of death as mortality structure and life expectancy changed and examine the factors behind such changes and their contribution to the new constraints that human mortality is facing.

Secondly, these new constraints, which have been found to be cancer inducible, will be examined in greater detail. Finally, socioeconomic differentials in cancer mortality will be reviewed since they can be regarded as the fundamental elements that condition one's life-style, or ones exposure to possible cancerous carcinogens. The review is based on both international and Canadian research findings.

In Chapter 4, the data required for the objectives of this study are discussed and the methods for data analysis

are reported also. In the following two chapters, we will study Canadian cancer mortality trends between 1951 and 1981. Equations for the estimation of future cancer death rates will be established. In Chapter 7, we will discuss our findings on the social-demographic analysis of cancer mortality. A smmary and suggestions for further analysis are provided in the final chapter.

2. The Emergence Of Cancer

2.1 Introduction

The absence of significant increases in life expectancy among the developed countries has raised serious concerns. Some investigators have suggested that a biological ceiling is encroaching on the human life span. Others consider the phenomenon a result of technological constraints that correspond to changes in the composition of causes of death. Degenerative diseases which dominate the present mortality structure are inelastic to the current levels of curative medical technology. This statement is particularly true of cancer. Despite the increasing success in delaying death from heart disease, cancer, which followed as the second leading cause of death, has remained largely incurable.

Further explorations on causes of degenerative diseases, including cancer, have added a third dimension to constraints on efforts to improve the present level of life expectancy. These constraints are directly related to personal behaviors or life styles in a broad sense. For cancer, they are actually risk factors. Such findings lead to the suggestions that cancer mortality can be prevented, if not cured, by appropriate modifications of hazardous behavior patterns.

Yet, the apparent obstacles, besides individual unwillingness to change, are to identify the risk factors and their functional network that interact with

socioeconomic and demographic elements. In order to better understand current human mortality and its social-demographic determinants, a review of the progress in the past is crucial. Canadian mortality experiences are selected for this purpose.

2.2 Canadian Mortality Trend

Canadian mortality in the 19th century has remained largely speculative as vital registration was not in force and data were not published until 1926 (Overebeek 1980). The oldest vital information reported occassionally could be dated back to 1921 only. Other sources of data that have been utilized to study the pre-registration period include records of baptisms, marriages, and burials secured by the Catholic church in Quebec from 1628 onwards (Overbeek 1980).

MaClean (D.B.S 1939) estimated levels of mortality using reports of death from the 1871 and 1881 Censuses. However, his figures were found unreliable because of inadequate coverage of the data base (McQuillan 1983). From figures produced by D'apres les Annuaires de Quebec in 1916, Kalbach and McVey (1979) state that Canadian mortality levels at no times, since 1867 have never exceeded that of the mid-1700's Catholic population, about 42 deaths per 1,000 population.

In his analysis of mortality patterns in Ontario between 1861-1921, McQuillan (1983) has added further insights into the historical trend of the Canadian death

rate. The author noticed that the pattern of mortality change in Ontario paralleled that reported earlier by Bourbeau and Legare (1982) for Canada as a whole.

The similarity lends credence to the notion that Canadian mortality decline was slow and steady in the first half of the 19th century, but was progressive since then. Female life expectancy, for example, increased from around 43 years in 1861 to about 50 years in 1901, and reached the 58 year mark by 1921 (McQuillan 1983).

Assessment of the post-1926 Canadian mortality trend is more reliable, as vital statistics are available. From the crude and standardized death rates (1956 base population) given in Table 2.1, male standardized death rate dropped from 13.3 per 1000 population in 1921 to 7.3 in 1981. Female death rate, which is always lower, fell from 12.9 to 4.2 in the same period.

Noteworthy in Table 2.1 is the small improvement in male mortality during 1956-1966. The percentages of decline were below those of the past and those that followed. Nevertheless, such decleration was not detected among the females. Although age adjusted rates are used in the comparison, they do not warrant bias-free results. In fact, the choice of the base population has been proven to be influential in across-time or -population comparisons, particularly when specific causes of death are considered (Schoen 1970; Shryock et al. 1976).

Table 2.1 Crude and Standardized Death Rates by Sex, Canada, 1921-1981

	Male		Female	
	(per 1000)			
Year	Crude	Standardized	Crude	Standardized
1921	11.9	13.3	11.2	12.4
1926	11.9	14.3	10.9	13.4
1931	10.5	12.7	9.6	11.7
1936	10.2	12.0	9.3	10.9
1941	10.8	12.0	9.1	10.2
1946	10.3	10.7	8.4	9.0
1951	10.1	10.0	7.8	8.0
1956	9.4	9.4	7.0	7.0
1961	9.0	9.0	6.5	6.3
1966	8.7	8.8	6.2	5.7
1971	8.5	8.4	6.1	5.2
1976	8.4	8.1	6.1	4.8
1981	8.0	7.3	6.0	4.2

Source: 1921-1971 figures from Table 1, Vital Statistics, General Mortality 1950-1972, Statistics Canada, Ottawa, 1976.
1976-1981 figures from 1976 and 1981 Vital Statistics, volume III, Statistics Canada, Ottawa, 1978, 1983.

Field (1980) explains that the employment of a young age structure in direct standardization can effect in a much higher decline over time than otherwise, the rationale being that the trend of death rates has been growingly in favour of the young but against the old. When combined with a young population, it produces lower age-adjusted rates.

In the Canadian situation, the 1956 base-population inflates the trend of decline (Field 1980). That is, the negative changes in standardized death rates would be less impressive if an older age structure is adopted. A way to avoid such a problem is to examine the trend in life expectancy. From Table 2.2, gains in life expectancy began to slow down from the mid-1950s onwards.

Among the males, intercensal increase reached a minimum in 1956-1966 and reboundced in the 1970s. Such a pattern is similar to that from standardized rates. For females, the trend differs slightly from that with adjusted rates. Although less prominent than in the case of the males, gains in female life expectancy did reach a minimum in 1961-1966. And, the size of recovery since then was smaller than for the male counterparts.

Compared with other developed countries, Canada seems to fit fairy well into the general pattern in which four stages of change can be identified. There were increases in life expectancy until the mid-1950's (for Canada, the 1960's), followed by a trough that lasted until the beginning of the 1970's. Recoveries were recorded since then

Table 2.2 Life Expectancy and Intercensal Changes by Sex, Canada, 1926-1981

Year	Male	Female
1926	56.9	58.9
1931	60.0	62.1
1941	63.0	66.3
1946	65.0	68.6
1951	66.3	70.8
1956	67.6	72.9
1961	68.4	74.2
1966	68.8	75.2
1971	69.3	76.4
1976	70.2	77.5
1981	71.9	78.9
1926-31	3.1	3.2
1931-41	3.3	4.5
1941-46	1.7	2.3
1946-51	1.3	2.2
1951-56	1.3	2.1
1956-61	0.8	1.3
1961-66	0.4	1.0
1966-71	0.5	1.2
1971-76	0.9	1.1
1976-81	1.7	1.4

Source: 1926-1976 figures from Table 1.2, Health Status in Canada, occassional paper no. 13, Institute for Research on Public Policy, Quebec, 1980.

until deceleration was again reported in the 1980s.

2.3 Mortality Deceleration and Development

Deceleration in mortality improvement in the developed and developing regions during the mid-1950's and 1970's might appear to have some resemblance, but their explanations share little similarities. The fact that the less developed nations have their death rates stalled at levels below the maximum attained by their industrialized counterparts suggest differences in the constituents involved.

Nonetheless, the incipient trends in both regions can be regarded as shaped by factors that brought mortality to the present low level, and in two inter-related ways. Firstly, factors which have greatly reduced death rates seem to become less and less efficient. Secondly, the paths through which these factors have worked and the extent to which they serve their purposes have in one way or another altered the mortality structure. Such change, better known as the epidemiologic transition (Omran 1971), requires new compromises and is posing a threat to future mortality decline. Understanding how the mortality structure has changed is essential to obtaining means for further mortality reduction.

Two groups of factors have been concluded as being indispensable in transforming mortality from a high to a low level, namely, socioeconomic and medical advancements. In

general, the former refers to aspects like improved food supply, better nutrition, and higher standards of living and housing; whereas the latter relates to medical innovations, better sanitation and public health programs (U.N. 1973).

To determine which one of these two groups or which elements therein are more powerful than the rest in reducing mortality is difficult, and results have often led to protracted debates. Similarly, attempts to determine the point in time at which one of these two groups becomes relatively more important than the other have also been debated.

2.3.1 Developing Countries

Studies done on the developing countries generally agree that mortality decline took place prior to any substantial improvements in social and economic order. Particularly, the progress since 1950 has been regarded largely as a consequence of diffusion or importation of disease control technologies and public health measures from the developed nations (Stolnitz 1955, 1956, 1965; Petersen 1975; U.N. 1962; Palloni 1981; Preston 1977, 1979).

Based on their observations on Latin America, Arriga and Davis (1969) explain that, while economic developments in less developed countries had reduced mortality, they were influential only before 1925. Since then, medical and public health measures were the prime factors. Also, it was this quick technology importation that mortality in the

developing regions could be reduced at such a rapid rate. Preston (1975) estimates about 80 per cent of the growth in life expectancy within the last three decades was due to technological changes.

The effectiveness of public health measures such as DDT, antibiotics, vaccines, and safe water supplies has been reported by various studies. The measures are essential for developing regions, as in Africa and for countries like Ceylon, Egypt, and India to reduce their mortality from infectious diseases (Gupta 1970; Stockwell 1956b; Chandrasekhar 1965; U.N 1973). The benefits of these "imports" were most significant for children and young adults who were the primary victims of many communicable diseases (U.N 1973).

However, the emphasis on medical technology and public health programs as the prime life saver is not without serious challenges. Studies on selected developing countries like Ceylon, Pakistan, and India suggest that there were improvements in life expectancy long before public health measures were introduced into the communities. Higher per capita income and improved nutrition from socioeconomic development were identified as the main stimulants for the observed mortality decline (Krishnan 1974; Heer 1975).

Recent path analysis by Yang and Pendleton (1979) on some 94 developing nations detectes the negative effect of health facilities on mortality and similar impact from economic development. The influence of the latter is

indirect through variables like living standard and nutrition. The authors thus conclude that simultaneous socioeconomic are necessary to depress mortality. An earlier study by Robinson (1967) also suggests interactive effects from economic development and medical services.

Much stronger objections to the significance of medical technology to mortality decline come from Taylor and Hall (1967) and Frederiksen (1960, 1961, 1966). The former investigators dismiss medical influences on mortality decline and attribute the progress to economic and nutritional improvements. The authors contended that a better economy allowed more food supply and education, which are essential in the fight against infectious diseases.

Fredriksen aims specifically at the widely acclaimed effect of DDT on malaria. His analysis of Ceylon indicates that death rates declined in both DDT-sprayed and non-sprayed areas, even before the chemical was applied. The progress in mortality decline is thus attributed to the increase of food imports. Meegama (1967) arrives at the same conclusion on the effect of DDT spraying in Ceylon but, at the same time, he credits the contributions made by medical facilities.

2.3.2 Developed Countries

With reference to mortality decline in the industrialized countries, the debate of socioeconomic versus medical development is by no means less vigorous. On the

contrary, availability of historical data permits more intense controversies on what initiated mortality decline. Anderson (U.N 1973:149, c.f) suggests that medical innovations have contributed relatively little to mortality progress in the Western European communities studied by him.

Based on the situations which prevailed in the 18th and 19th centuries in England and Wales, McKeown and associates (1955, 1962, 1972, 1978) argue that public health programs and medical technology did not start mortality decline. Instead, upgraded living standards and nutrition, in particular, were the keys to resisting infectious diseases and thus initiating subsequent mortality decline. McKeown (1979) recently traced the historical pattern of causes of human death and medical inventions and stated that the impact of medical technology remained insignificant until the introduction of sulpohonamides to combat tuberculosis in 1935.

The proposition by McKeown (1979) is to some extent parallel to what Arriaga and Davis (1969) have noted for the European countries. That is, medical measures became the prime life saver from 1930 onwards. Before this time, mortality decline depended on economic development, thereby explaining the comparatively long period required by the developed countries to reduce their death rate.

Razzell (1974) and Preston (1975, 1977), however, reject the leading role of socipeconomic development. They argue that studies by McKeown and colleagues underestimate

the impact of innoculation and vaccination upon death rates in England and Wales. A more recent study by Mercer (1985) on European countries also downplays the role of nutrition in the fight against epidemics like smallpox and strongly favors the effect of immunization.

Stockwell and Groat (1984) have taken a more neutral stand. They summarize that better food intake initiates mortality decline, but they also emphasize the decisive, yet later, role of medicine and public health measures. At this point, there is apparently more concensus on the significant impact of the medical sciences on mortality level since the turn of the 20th century. However, for the earlier period, both groups of factors had been equally and persuasively supported.

2.4 The Epidemiologic Transition

2.4.1 "Rock Erosion" and Epidemiologic Transition

Despite the heated debates, few studies would disagree that these two groups of factors, either by themselves or in collaboration, have drastically reduced death rate in most parts of the world. The accomplishments were particularly outstanding in the control of many communicable diseases, which are close to eradiction within the developed countries. Table 2.3 and Fig. 2.1 illustrate reductions in infectious diseases in selected Western nations and Ceylon, respectively.

Table 2.3 Reduction Of Infectious Diseases In Selected Developed Countries

(Average annual death rate per 100,000 population. Average of the rates computed by five-year age groups)

Country	All causes 5-34 years			All causes excluding violence 5-34 years			Group I (Infectious and parasitic diseases, influenza and pneumonia, and bronchitis below the age of five years)											
							All ages			5-34 years			45-64 years			65-74 years		
	1936-1938	1950-1953	1954-1956	1936-1938	1950-1953	1954-1956	1936-1938	1950-1953	1954-1956	1936-1938	1950-1953	1954-1956	1936-1938	1950-1953	1954-1956	1936-1938	1950-1953	1954-1956
							Males											
Denmark	176	103	91	131	53	38	183	69	37	62	13	5	165	59	37	409	181	109
Netherlands	162	98	82	128	61	46	156	63	44	61	13	6	175	68	47	459	224	156
United States	280	152	138	181	66	54	217	74	54	83	16	8	337	114	76	539	209	190
New Zealand	196	124	119	128	60	50	144	65	55	61	14	8	230	85	56	328	201	185
Canada	236	143	131	176	69	53	200	81	58	85	21	9	222	84	57	473	210	165
Australia	205	144	131	126	62	53	151	76	61	68	15	8	246	117	82	442	315	240
Norway	272	119	103	210	62	48	238	89	64	149	22	9	276	73	63	543	207	165
Scotland	248	125	107	196	84	65	269	108	75	118	31	14	314	157	107	476	304	252
Switzerland	240	132	133	170	57	56	207	88	76	86	22	14	281	138	91	567	291	266
England and Wales	235	108	89	186	70	51	224	116	85	99	23	10	315	162	106	418	393	318
Italy	310	153	127	263	109	77	413	150	100	154	44	24	399	177	127	858	345	250
France	344	141	127	279	87	66	266	164	110	139	35	14	337	204	143	583	427	319
Japan	663	279	205	586	210	124	519	202	130	365	100	54	445	223	229	771	540	455
							Females											
Denmark	150	66	53	139	50	38	172	62	34	64	11	4	138	58	24	425	170	93
United States	232	87	71	204	66	51	161	48	35	80	15	7	173	60	27	390	113	88
Netherlands	142	65	51	132	53	42	147	59	38	64	13	6	129	45	25	456	202	130
New Zealand	167	69	58	153	46	42	98	47	41	62	15	6	96	38	27	195	91	87
Australia	178	80	66	160	65	51	111	53	42	56	15	7	107	42	29	281	126	107
Canada	232	88	68	216	72	50	188	65	44	99	23	9	144	60	27	423	129	87
Scotland	223	116	74	209	106	64	205	87	57	115	47	17	151	61	40	358	175	129
Norway	212	62	50	201	54	42	222	86	62	132	18	7	208	39	23	541	147	119
Switzerland	199	74	61	180	56	45	194	77	65	93	19	11	176	57	43	502	211	182
England and Wales	209	79	58	199	71	49	161	86	65	99	25	10	129	65	40	281	234	153
Italy	294	106	87	283	96	77	362	120	75	170	44	20	262	75	46	772	261	146
France	289	100	74	273	87	59	182	132	82	132	34	12	141	72	44	304	252	165
Japan	705	253	163	675	229	134	484	172	104	398	102	53	271	151	101	474	393	237

Note: The calendar-year periods shown in the column headings could not always be observed for all the countries. The following are the exceptions:

1936-1938 should read 1935-1936 for Japan and 1937-1938 for Switzerland.

1950-1953 should read 1951 for Italy, 1951-1953 for Norway and 1952-1953 for Switzerland.

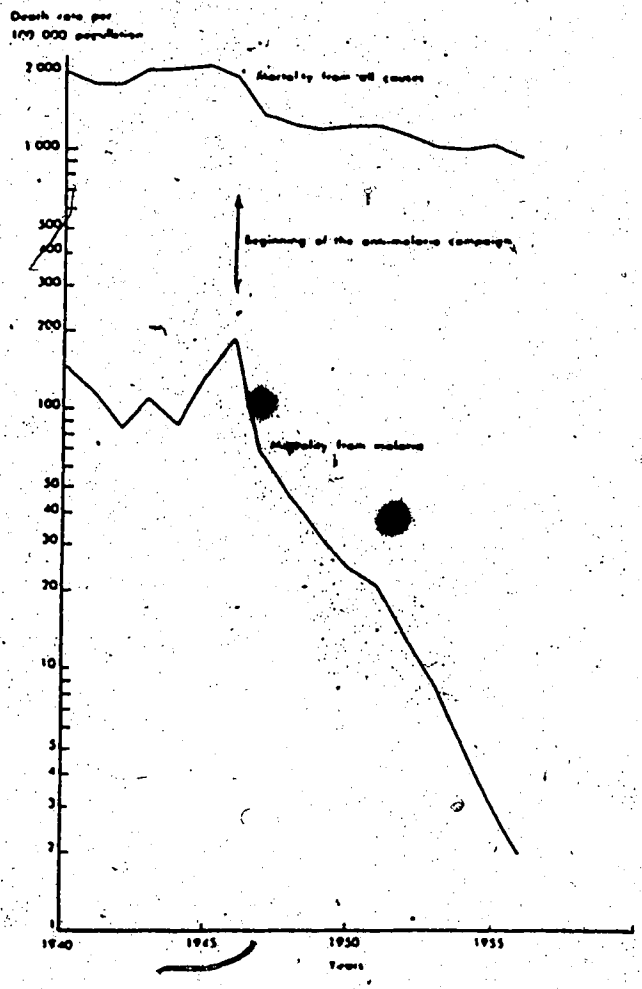
1954-1956 should read 1954 for Italy.

e Estimated rates.

Source: Population Bulletin of the United Nations No. 6-1962, Table V, 8, P. 80. N.Y., 1963.

Fig. 2.1 Changes in Malaria Mortality Rates in Ceylon, 1940-1957

CEYLON, 1940-1957. MORTALITY FROM ALL CAUSES AND FROM MALARIA (CRUDE DEATH RATE PER 100,000 POPULATION, LOGARITHMIC SCALE)



Source: Population Bulletin of the United Nations. No. 6-1962. Figure V.2, P. 82. N.Y., 1963.

The outcomes of this success are twofold. Firstly, as infectious diseases attack primarily children and young adults, their reduction means greater chances to survive to older ages. This has materialized in the substantial increase in life expectancies reviewed in earlier section. Secondly, the disappearance of infectious diseases, especially in the developed countries, generates a new mortality structure comprised of causes of death that are rather inelastic to present medical knowledge. The recent deceleration in mortality is partly the result of this new structure.

Mortality structure has been described as analogous to rock erosions (U.N. 1963). The outer softer rock resembles a layer of infectious and parasitic diseases and could be "weatherized" with the help of medical and health measures. The second layer however, consists of diseases that require not only technology but, more importantly, fundamental socioeconomic developments to support and to maximize the usages of available technology. Lastly, the inner hard rock layer is composed of diseases difficult to track or to cure. Its erosion demands medical technology beyonds the present limits (U.N. 1963).

The modification of the composition of causes of death can also be depicted through the notion of epidemiologic transition. According to Omran (1971), the transition is in many ways similar to the demographic and technological transition in the now developed countries. Three basic

determinants associated with the various stages of transition can be identified.

Ecobiologic and socioeconomic factors are the key elements behind the transition of the initial age of epilesence and famine, to the second stage of receding pandemics. Such transition is signified by increasing control of epidemics and rising life expectancy from about 30 to 50 years at birth. Around the beginning of the 20th century, medical and public health determinants became the predominant force to combat diseases.

This resulted in significant reduction or even disappearance of epidemics, but marked the beginning of the age of degenerative diseases. At this final stage, life expectancy is at the 70 years plus range, and heart disease and cancer are two of the major killers.

In order to visualize the relationship between life expectancy and changes in causes of death, the United Nations has constructed two model tables with different assumptions on the level of fertility. The first table is based on the estimated 1960 world population and high fertility. The age structure is, therefore, relatively young, and results describe the developing regions.

The second table is based on a stable population with a gross reproduction rate of 1.5, and it depicts changes in the developed countries (U.N., 1963). Although the present world population age structure differs from that employed to construct the tables, the estimations make the intricate

association between mortality level and causes of death almost self-explanatory. The two tables are reproduced in Table 2.4.

2.4.2 The Technological Misfit in Developing Nations

These model tables and the two perspectives on mortality structure aid in understanding the recent slow-down in death rate reduction. With current life expectancy averaged at 58 years and with a range of 50-64 years (World Population Data Sheet 1981), most developing countries are in the medium soft rock layer, or in the stage of receding pandemics (U.N. 1963, Omran 1971).

At this level of life expectancy, fewer of the leading causes of death are of the infectious type and are thus less inclined to respond to medical measures. For example, in the six Latin American countries with life expectancy around 60 years--Chile, Costa Rica, Mexico, Panama, Paraguay, and Venezuela; the six primary causes of death are influenza and pneumonia, enteritis and other diarrhoeal diseases, heart disease, cancer, cerebrovascular diseases, and accident (U.N. 1980a).

Of these six leading causes of death, the resistance of heart disease and cancer to medical technology is not unfamiliar. In fact, their increase over time has long been considered to have a stabilizing effect on the general death rate (Behm and Gutierrez 1967). Although the first two leading causes of death are of the communicable type, their

Table 2.4 Patterns of Mortality Structure with Young and Old Age Structure

PATTERN OF MORTALITY TRENDS IN A POPULATION WITH A "YOUNG" AGE STRUCTURE (BOTH SEXES), BY CAUSE-OF-DEATH GROUPS FOR EXPECTATIONS OF LIFE AT BIRTH RANGING FROM 40 TO 76 YEARS

Expectation of life at birth for both sexes (in years)	Standardized mortality rates per 100,000 population						Percentage distribution of rates					
	All causes	Group I	Group II	Group III	Group IV	Group V	All causes	Group I	Group II	Group III	Group IV	Group V
40	2,430	1,061	91	360	84	834	100.0	43.7	3.7	14.8	3.5	34.3
42	2,240	931	91	349	81	788	100.0	41.6	4.1	15.6	3.6	35.2
44	2,069	819	91	338	78	743	100.0	40.0	4.4	16.3	3.8	35.9
46	1,909	719	91	327	76	696	100.0	37.7	4.8	17.1	4.0	36.5
48	1,764	633	91	316	73	651	100.0	35.9	5.2	17.9	4.1	36.9
50	1,629	556	91	304	70	608	100.0	34.1	5.6	18.7	4.3	37.3
52	1,504	486	91	293	67	567	100.0	32.3	6.1	19.5	4.5	37.7
54	1,387	420	91	282	64	530	100.0	30.3	6.6	20.3	4.6	38.2
56	1,278	360	91	271	62	494	100.0	28.2	7.1	21.2	4.9	38.7
58	1,174	304	91	260	59	460	100.0	25.9	7.8	22.1	5.0	39.2
60	1,072	254	91	249	56	422	100.0	23.7	8.5	23.2	5.2	39.4
62	972	208	91	238	53	382	100.0	21.4	9.4	24.5	5.5	39.3
64	876	166	91	227	50	342	100.0	18.9	10.4	25.9	5.7	39.0
66	782	128	91	215	47	301	100.0	16.4	11.6	27.5	6.0	38.5
68	690	95	91	204	44	256	100.0	13.8	13.2	29.6	6.4	37.1
70	600	65	91	193	41	210	100.0	10.8	15.2	32.2	6.8	35.0
72	512	40	91	182	39	160	100.0	7.8	17.8	35.5	7.6	31.2
74	426	20	91	171	36	108	100.0	4.7	21.4	40.1	8.5	25.4
76	342	5	91	160	33	43	100.0	1.5	26.6	46.8	9.6	12.6

PATTERN OF MORTALITY TRENDS IN A POPULATION WITH AN "OLD" AGE STRUCTURE (BOTH SEXES), BY CAUSE-OF-DEATH GROUPS FOR EXPECTATIONS OF LIFE AT BIRTH RANGING FROM 40 TO 76 YEARS

Expectation of life at birth for both sexes (in years)	Standardized mortality rates per 100,000 population						Percentage distribution of rates					
	All causes	Group I	Group II	Group III	Group IV	Group V	All causes	Group I	Group II	Group III	Group IV	Group V
40	2,675	950	152	550	93	930	100.0	35.5	5.7	20.5	3.5	34.8
42	2,510	850	152	540	90	878	100.0	33.9	6.0	21.5	3.6	35.0
44	2,355	760	152	530	87	826	100.0	32.3	6.4	22.5	3.7	35.1
46	2,205	675	152	520	84	774	100.0	30.6	6.9	23.6	3.8	35.1
48	2,067	600	152	512	81	722	100.0	29.0	7.4	24.8	3.9	34.9
50	1,935	530	152	504	78	671	100.0	27.4	7.9	26.0	4.0	34.7
52	1,811	470	152	496	75	618	100.0	26.0	8.4	27.4	4.1	34.1
54	1,690	410	152	488	72	568	100.0	24.2	9.0	28.9	4.3	33.6
56	1,581	360	152	480	69	520	100.0	22.8	9.6	30.3	4.4	32.9
58	1,475	310	152	472	66	475	100.0	21.0	10.3	32.0	4.5	32.2
60	1,371	260	152	464	63	432	100.0	19.0	11.1	33.8	4.6	31.5
62	1,271	210	152	457	60	392	100.0	16.5	12.0	36.0	4.7	30.8
64	1,182	170	152	450	57	353	100.0	14.4	12.8	38.1	4.8	29.9
66	1,094	130	152	444	54	314	100.0	11.9	13.9	40.6	4.9	28.7
68	1,006	90	152	438	51	275	100.0	9.0	15.1	43.5	5.1	27.3
70	928	60	152	432	48	236	100.0	6.5	16.4	46.5	5.2	25.4
72	857	39	152	426	45	195	100.0	4.6	17.7	49.7	5.2	22.8
74	788	20	152	420	42	154	100.0	2.5	19.3	53.3	5.3	19.6
76	729	10	152	416	39	112	100.0	1.4	20.8	57.1	5.3	15.4

Note: Group I= infectious and parasitic diseases, II= Cancer, III= cardiovascular diseases, IV= violence, V= others.

Source: Population Bulletin of the United Nations, No. 6-1962, Table V.33, V.35., PP. 111-112, N.Y., 1963.

dominance over others causes like malaria and smallpox posts a new threshold.

Preston and Nelson (1974) observe that, as gastrointestinal and respiratory infections become more controlled, these two diseases increase in relative importance. Gwatkin (1980) adds malnutrition to this diarrhea-pneumonia complex of diseases and warns against their inelasticity to traditional health programs devised for the developing regions.

Such disagreement about causes and remedies created a "technologic misfit," which is partly responsible for the recent slowdown in mortality decline. To confront these new issues, Gwatkin (1980) emphasizes improvements in basic living standard and economic conditions as the prime solutions. In this regard, Gwatkin cites findings by John Byrant (1969) as part of his evidence of support:

> ...Diarrhea and pneumonia are often not affected by antibiotics and the frequent presence of malnutrition makes even supportive therapy difficult or futile... Their lives [of the children] are saturated with the causes--poverty, crowding, ignorance, poor ventilation, filth, flies...(Gwatkin 1980:75)

However, performance of either the social or economic sector in most less developed countries within the past years was far from impressive. Whether the concerns are per capita income, food consumption or education, the progress was just slightly noticeable (Gwatkin 1981). The consequence, perhaps best described by Palloni (1981) who stated that:

> ...a rather perverse consequence of isolated technological innovations unaccompanied by harmonic socioeconomic developemnt is a possible worsening of morbidity and mortality level...(P. 827)

Indeed, the emergence of new causes of death which resist current medical measures, compounded by sluggish socioeconomic development could explain the small mortality decline in most developing regions.

2.4.3 Technological Constraints and Increase of Cancer in Developed Nations

With regard to the developed nations, the reasons for the deceleration in mortality are different. Obviously, unlike the developing countries, the absence of socioeconomic development is not a factor in this part of the world. On the contrary, advanced social and economic establishments contribute to many factors in the transition from acute to predominantly degenerative diseases (U.N. 1984).

Through higher standards of living, more advanced medical technology, and more and better health care facilities, socioeconomic development has eradicated most infectious diseases (McKeown 1979; Walsh and Warren 1979; Spiegelman 1956). At the same time, behavioral factors associated with affluent living conditions and life-style, and environmental risk brought about by modernization enhance the risk of degenerative diseases (Weatherby et al. 1983; Keyfitz 1977).

Consequently, degenerative diseases, notably heart disease and cancer have become the leading causes of death (U.N. 1963; 1973). A study of 23 developed nations between 1950-1954 and 1970-1974 revealed that, while infectious diseases account for only 1 per cent of total deaths at the end of the period, heart disease and cancer had increased, respectively, from 44 to 48 and from 16 to 19 per cent (U.N. 1982).

Increasingly aging populations in the developed regions, coupled with the selectivity of degenerative diseases of persons middle-aged and older, have intensified the situation. The long decline of fertility, supplemented by lower death rate, has expanded the population of the age groups of 65 and over, and keeps degenerative diseases well supplied with high risk populations (U.N. 1963; W.H.O. 1984; Fries 1980). To illustrate the multiplicative effect of an old age structure, proportions of total deaths due to heart disease and cancer given in Table 2-4 can be compared at the same level of life expectancy.

In all cases, percentages are higher in the model assuming an older population. This age structure effect demands no less attention than the diseases themselves. It has been cautioned that since 70 per cent of all deaths took place after age 65, any major gains in life expectancy must come from the reduction of chronic diseases. If not, the aging population will push crude death rate aloft the present level (U.N. 1963, 1980b).

Following the rationale of the foregoing discussion, the setbacks since the 1950's and the higher death rates projected for upcoming years are not unexpected. Though the situation might appear pessimisitic, some mortality decline has been registered in some developed countries latley (U.N. 1982). Not only has the death rate started to fall again, but reasonable gains in life expectancy were also recorded.

This revitalization is quite startling in a period of general deceleration. Moreover, it stems from improvements in degenerative diseases whose reluctance to respond to curative medicine is believed to have imposed a technological limit on life expectancy (U.N. 1979b; 1980b). Also, many behavioral and societal factors have been identified as hazardous to health in terms of degenerative diseases, thereby making it even harder to extend the length of life (Manton 1982a; Lalonde 1974; Nightingale 1981). Under these restraints, the renewed [illegible] more than welcome as it suggests that the ceiling of human life expectancy is still distant (U.N. 1980a).

However, not all degenerative diseases are amenable to improvements. In countries experiencing positive mortality changes, only declines in heart disease death rate have been named the main contributor (U.N. 1980b; Crimins 1981; Manton 1982b). Also, it is believed that medical innovations have limited contributions to such a downtrend (Wunsch and Lambert 1981). Most of the new technology is still only capable of postponing fatality rather than offering a

complete cure (Keyfitz 1977; U.N. 1963). Major credit is accorded to modifications of disease-prone behaviors such as reduction in consumption of cigarettes and animal fat, and increase in physical exercise (Nightingale 1981; Crimmins 1981; Wunsch 1981).

Contrary to progress in controlling heart diseases, cancer, as the second major degenerative cause of death, remains in a less-than-favourable position. The upward trend of cancer death rate in the pre- and post-war years not only lingers on in most developed countries, but has actually steepened in many since then. According to the United Nations (1982), more than half of the 27 developed countries being reviewed had an increased in cancer mortality rate of at least 5 per cent between 1960-1976.

Increases exceeding 20 per cent were not uncommon in the age groups of 65 and above. Although some countries had either staiblized or reduced deaths from specific cancer sites or the younger age groups, such progress did not help to lower the proportion of deaths due to cancer. These achievements were usually waived by simultaneous increase in cancer of the lung, breast, intestines, and bladder and among the older age groups in general (Wunsch 1981; Logan 1975b; Benjamin 1977; U.N. 1982).

A recent study estimated that about 25 per cent of all deaths in the industrialized countries in the early 1980's were due to cancer. This statistic represents an increase from 18 per cent registered in 1976 (U.N. 1982; 1984). The

overall deterioration could be due to the absence of real improvement, since progress and recession are equally split among the developed countries (Lopez and Hanada 1982).

At the same time, the aging population also helps to increase the proportion of cancer deaths even though the death rate can remain unchanged (U.N. 1963). Equally true is that decline in heart disease mortality as a result of delaying death can expose more elderlies to the risk of cancer. It is rather obvious that the seriousness of cancer mortality draws on not only its own mortal forces, but also on the aging population and, perhaps to a lesser extent, the improvements in the survivals from heart disease.

2.5 Cancer and Societal Constraints

As with heart disease, preventive measures have been emphasized as the best cure for cancer. Based on the abundance of evidence linking cancer to occupational hazards and to deleterious health behaviours such as smoking, excessive drinking and improper diet, increasing numbers of studies have stressed the need for personal re-adjustments in terms of these factors in order to prevent the onset of cancer (Lopez and Hanada 1982; Keyfitz 1977; Nightingale 1981; Lalonde 1974).

The importance of such modifications is further demonstrated by analyses of various religious groups. Lower cancer deaths among the Mormons and the Seventh Day Adventists, for example, are being linked to their

refraining from a high-risk life-style (eg smoking) (Jarvis 1978; Philips et al. 1980). Higginson (1969) and Boyland (1967) have each attributed about 80 and 90 per cent, respectively, of all cancer deaths to environmental factors instead of genetic inheritance. The disease was thus promoted as preventable through behavioral and environmental modifications.

These emphases on life styles and behavior patterns fit well into the notion of societal constraints operating on life expectancy. As Manton (1982a) summarizes from earlier studies, a part of these societal constraints like hygiene, nutrition and sanitation has already accounted for the historical mortality decline through the control of infectious diseases.

Yet, the residuals like life style and environmental factors have deteriorated and create more problems in dealing with chronic or degenerative diseases. These societal constraints operate prior to the arrival of a biological limit to life expectancy and are deterministic in times when medical technology is having little impact on life expansion (Manton 1982a).

Since medical innovations can delay heart disease deaths until older ages but not in cancer deaths, societal restrictions are thus ever more critical in their preventive value. Also, because of their roots in the social structure, such behavioral or environmental factors must be weighted within different social settings.

In other words, the social determinants of cancer should be studied in relation to various social settings which themselves could define the type of constraints to be involved (Behm, 1980). Results of such researche could prove to be valuable in formulating policies that could have far-reaching effects across all population subgroups.

2.6 Canadian Cancer Mortality Trend

Canadian cancer mortality trend has been analyzed in various studies. A series of reports by Statistics Canada (1965) discuss the general cancer mortality between 1950-1963, and highlight on the situations in earlier decades. It is indicated that cancer crude death rate for both sexes combined increased by more than 50 per cent in the period 1926-1963; cardiovascular-renal diseases, on the other hand increased by only 40 per cent, while infectious diseases were reduced drastically by about 67 per cent.

Although the absence of historical data on causes of death has prevented the documentation of the timing of epidemiologic transition in Canada, it has been suggested elsewhere that most industrialized countries completed their transition sometime during the mid 1950's (Lopez and Hanada 1982). From the percentage distribution of changes in the major causes of death, it seems that Canada would not deviate from this pattern.

For the period 1950-1963, standardized death rate based on the 1956 base population showed that cancer deaths of

both sexes increased by slightly more than four per cent; up from 126.2 per 100,000 population to 131.6 by the end of the period. For males, the change was from 128.2 to 145.5, and for females, from 124.1 to 117.4. Crude death rate from all causes of death together dropped from 9.1 to 7.6 per 1000 population, or by 16 per cent (D.B.S. 1965).

By 1972, cancer mortality of both sexes has climbed by another 5.4 per cent to 138.7 per 100,000 population. Male death rate rose to 162.5, while the female rate decreased to the level of 114.3. Meanwhile, overall death rate continued to decline to 6.9 per 1000 population, which was an eight per cent improvement. (Statistics Canada 1976). An upward trend in cancer mortality is noticeable for males and for both sexes combined, and minor improvements are recorded for females.

Noteworthy in such trend analysis is the influence of the base population. The inflationary effect of the 1956 age structure on mortality decline has been reported earlier. The same effect could be anticipated to become more exaggerate, since cancer mortality attacks mostly the middle aged and the elderly. The farther away the year of the standardized rate is from the base population, the more likely that the adjusted rate is lower than what it should have been. In other words, the real trend of cancer movement can be masked by an inappropriate base population.

Not unrelated to the issue of population age structure is the observation of cohort effects in the proportion of

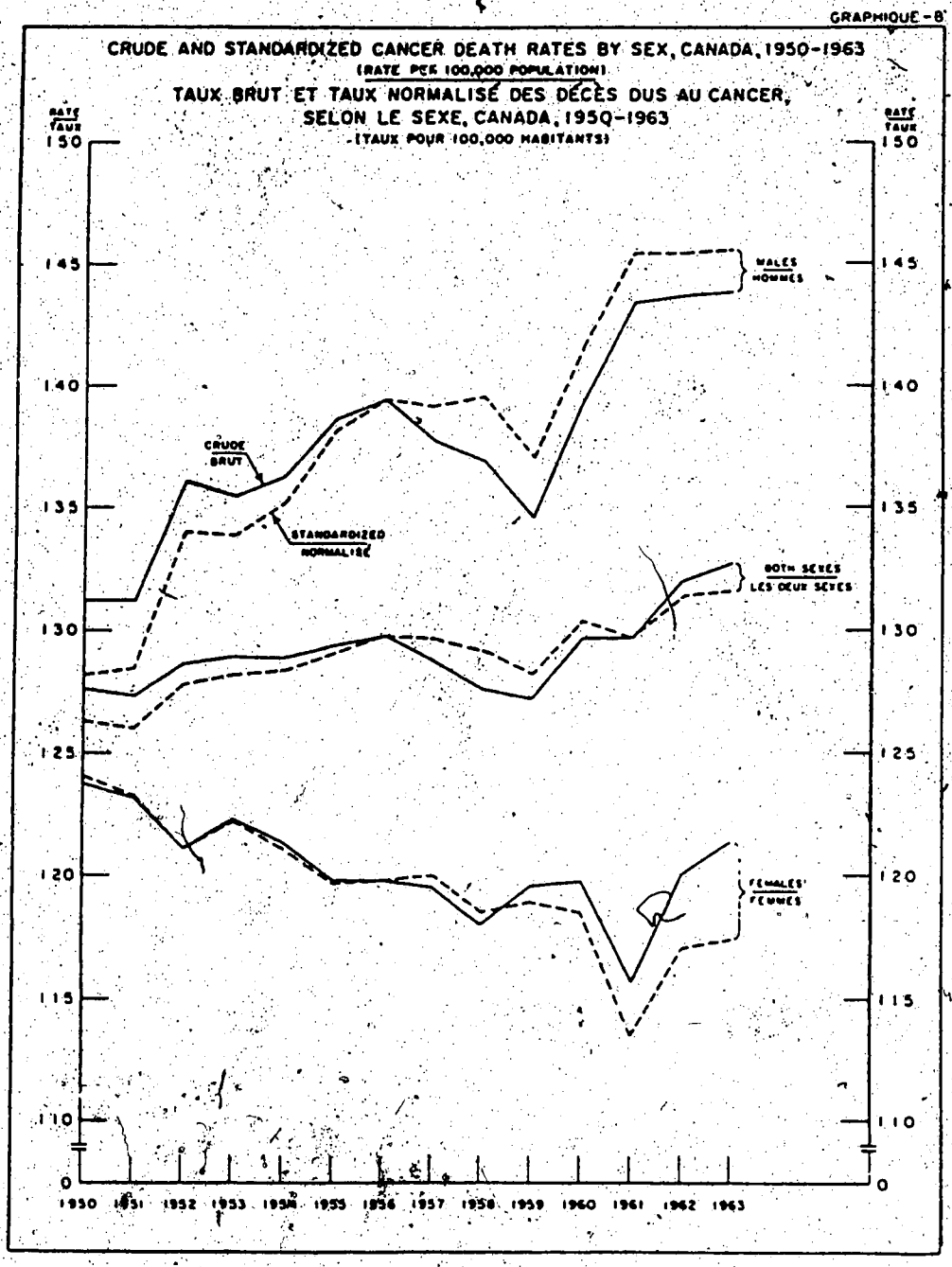
elderlies. When crude and standardized cancer death rates were compared for the period 1950-1963, it was found that, prior to 1956, male crude death rates were well above the adjusted rates, but were rising more slowly than the latter.

In the following three years, however, the crude rates were dropping much faster than the standardized cancer rates (D.B.S. 1965). Such changes are depicted in Fig. 2.2. Among the females, similar occurrances were not observed, and both the crude and adjusted death rates were moving closely with each other.

The reason given for the exceptional pattern among males is the lessening proportion of elderly males between 1950 and 1959 (D.B.S 1965). Factors behind this reduction may be from three sources: the baby boom in 1946-60, the baby bust in late 60's and 70's, and immigration in the subsequent period. In fact, the sharp decline in 1959 for males and in 1961 for females (Fig. 2.2) could plausibly be due to the influx of migrants between 1951-1961, which peaked at around 1958. Total immigration within this decade was close to 9.6 per cent of the then average population (Kalbach and McVey 1971:Table 2:1). The reason why the female cancer death rate fell sharply three years later, and not before, could be due to the selective nature of migration.

In the above discussions, the importance of age structure to cancer mortality analysis has been briefly reviewed. Of particular relevance to policy implications is

Fig. 2.2 Crude and Standardized Death Rates by Sex, Canada, 1950-1963



Source: General Cancer Mortality 1950-1963, P. 39, Chart B. Dominion Bureau of Statistics, Ottawa, 1965.

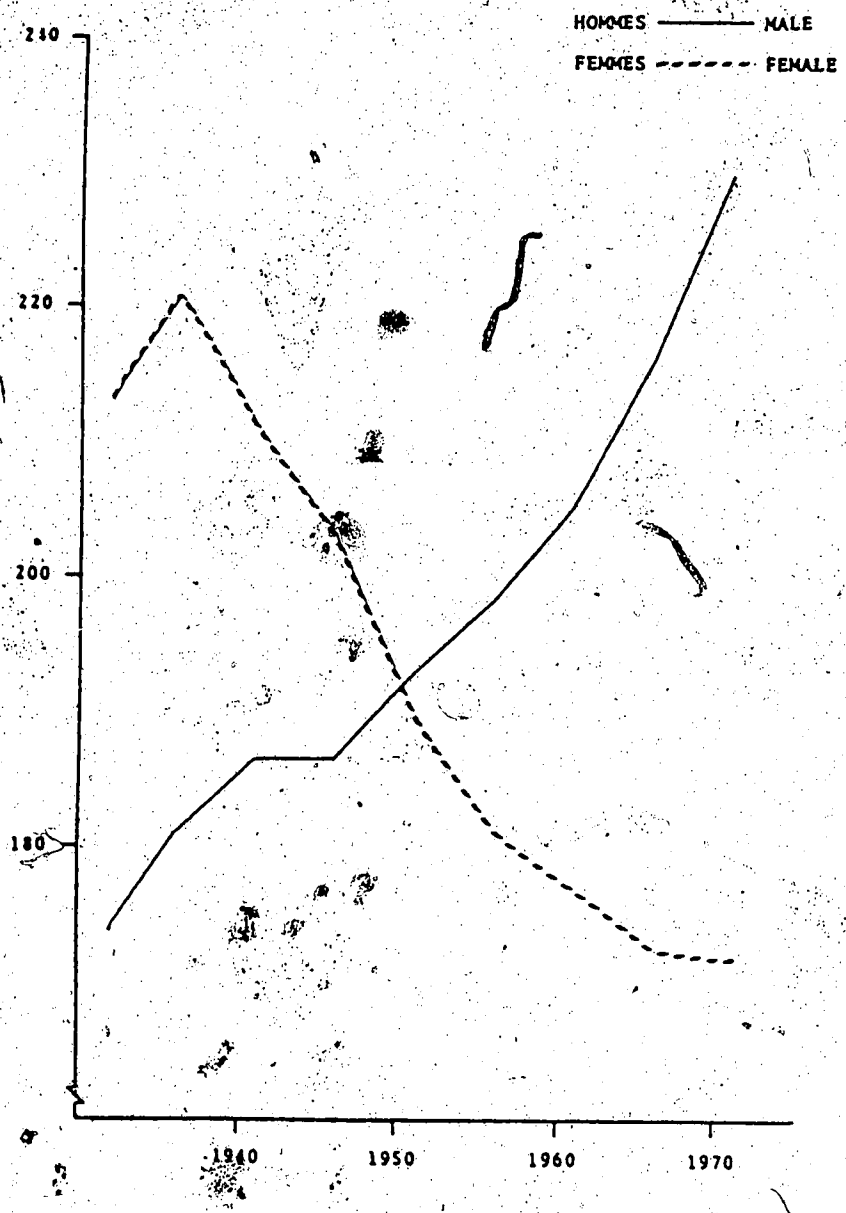
the possibility of variations in the supply of risk population (middle aged and above) by different birth cohorts. It is generally desired to estimate the effect of aging on cancer mortality so that appropriate health care facilities can be planned. If the different contributions by various cohorts to the overall risk population are neglected, then the planning can be easily off target.

A trend analysis with comparatively longer time series has been conducted by National Health and Welfare of Canada (1976) for the period 1931-1974. This analysis differed from that discussed earlier in the selection of the base population. A standard population approximating the 1971 Canadian age structure was adopted. The results could reflect a more realistic picture of the changes.

Yet, the standard population selected still consisted of fewer elderlies and the estimates were mainly for the 25-74 years age groups. The ignoring of deaths from other age brackets, especially those 75 years and above, could produce a more drastic decline in cancer mortality, as seen from the graphs in Fig. 2.3.

In Fig. 2.3, there is a clear crossover between male and female death rates. Prior to 1950, females had a higher mortality than males; but this has reversed since then. In fact, male cancer death rates had been on an upward trend all the time. Analysis of the 1964-1974 data indicated significant [redacted]ive trend for males but no direction was observed for [redacted]es.

Fig. 2.3 Age Standardized General Cancer Death Rates by Sex, 25-74 years of Age, Canada, 1933-1973



Source: Cancer Pattern In Canada: 1931-1974, P. 78, Figure 36. National Health and Welfare, Ottawa, 1976.

When the cancer death rate of all ages, instead of the 25-74 age groups, is plotted (Fig. 2.2), the crossover as well as the sharp declines for both sexes in 1959-1961 are not evident. The impact of cancer is measured better through life expectancy gains after cancer is hypothetically eliminated. The estimates from the National Health and Welfare report are given in Table 2.5 along with earlier results by Cheung et al. (1984) and, more recently, by Ng and Krishnan (1985).

We see that gains in life expectancy after removing cancer have increased across the selected age groups and for both males and females throughout the period. Such gains indicate the growing seriousness of cancer as a cause of death. While the positive changes among males are not unexpected from their increased cancer death rates, it is less so for the females. The situation for the females appears to be at odds with their cancer improvements in 1950-1974. One of the reasons could be the decline in other causes of death, which therefore maintains or even boosts the mortality forces of cancer among the females.

Besides the focus on overall cancer mortality, the trends of individual sites have generated considerable interest among researchers. For example, Cudmore and Zayachkowski (1967) studied the trends of most cancer sites in Canada between 1950 and 1963. Elwood and Lee (1974) looked into the movements of primary skin cancer in 1951-1970.

Table 2.5 Life Expectancy Gains After Cancer Elimination at Selected Ages by Sex, Canada, 1970-1983

Year		Age 1	20	40	65
1970-72	M	2.78	2.71	2.66	1.87
	F	2.92	2.87	2.71	1.48
1975-77	M	2.83	2.82	2.78	1.93
	F	3.15	3.13	3.00	1.79
1978-80	M	3.05	3.06	3.02	2.11
	F	3.26	3.25	3.13	1.89
1981-83	M	3.22	3.22	3.19	2.21
	F	3.40	3.39	3.26	1.99

Source: 1970-72 figures from Table 5, Cancer Patterns in Canada 1931-1974, National Health and Welfare, 1978.
1975-1977 figures derived from Cheung et al., Life Tables by Causes of Death, Canada 1975-1977, Discussion Paper no. 36, Department of Sociology, University of Alberta, 1984.
1978-1983 figures derived from Ng and Krishnan, Gains in Life Expectancy on Elimination of Certain Causes of Death, 1978-1983, unpublished manuscript.

Furthermore, Hill and associates (1981) and McPhee and Hill (1981) reported on the incidence and mortality of breast and bladder cancer, respectively, in Alberta, Canada. Cancer of the large bowel during 1950-1973 was examined by Stavraky and Lindsay (1979). Philips (1966) and Miller (1977), to name just a few, analyzed the trends of lung cancer between 1936-1964 and 1935-1973 respectively.

More comprehensive review on national and provincial cancer mortality by sites is also available in the two reports by the Dominion Bureau of Statistics of Canada (1966). These reports consider the period of 1950-1963 and 1960-1973. Trend analysis on cancer sites has also been carried out by National Health and Welfare of Canada (1976). Their short term study of 1964-1974 reveals a significant decline in stomach cancer for both sexes and in cancer of the cervix and uterus for females.

Unfortunately, they had also observed positive trends in lung cancer for both males and females, as well as intestinal cancer (minus rectum) among the former. The top ten cancer sites in Canada during 1975-1979 have recently been determined by Statistics Canada (1984). The rankings are given in Table 2.6.

In most trend analyses, the variations among the provinces are not left unnoticed. Provincial variations in breast and lung cancer are illustrated in Tables 2.7 and 2.8 as examples. Recently, variations in cancer by sites in 1971-1976 were captured in a series of mortality maps

Table 2.6 The Top Ten Cancer Sites in Canada by Sex, 1975-1979

	Male		Female	
Rank	Site	Percent	Site	Percent
1	Lung	30.4	Breast	20.5
2	Prostate	9.5	Colon	11.7
3	Colon	8.4	Lung	9.6
4	Stomach	7.4	Ovary	6.2
5	Pancrease	5.5	Pancrease	5.5
6	Rectum	3.9	Stomach	5.2
7	Bladder	3.3	Rectum	3.6
8	Unspecified	2.7	Unspecified	3.1
9	Brain	2.3	Cervix Uteri	3.1
10	Kidney	2.3	Brain	2.3

Source: From Cancer Incidence and Mortality At Different Ages, Canada 1975-19709, Statistics Canada, Ottawa, 1984.

Table 2.7 Provincial Differentials In Female Breast Cancer Death Rates, Canada, 1960-1973

Province	10. Breast – Sein				
	Always higher – Toujours supérieur	Usually higher – Généralement supérieur	No consistent difference – Pas de différence soutenue	Usually lower – Généralement inférieur	Always lower – Toujours inférieur
	Female – Féminin				
Newfoundland – Terre-Neuve				x	
Prince Edward Island – Île-du-Prince-Édouard				x	
Nova Scotia – Nouvelle-Écosse			x		
New Brunswick – Nouveau-Brunswick			x		
Québec	x				
Ontario			x		
Manitoba				x	
Saskatchewan				x	
Alberta					x
British Columbia – Colombie-Britannique			x		

Source: Vital Statistics, Cancer Mortality by Site: 1960-1973, P. 29. Statistics Canada, 1977.

Table 2.8 Provincial Differentials In Lung Cancer Death Rates, Males and Females, Canada, 1960-1973

Province	Always higher – Toujours supérieur	Usually higher – Généralement supérieur	No consistent difference – Pas de différence soutenue	Usually lower – Généralement inférieur	Always lower – Toujours inférieur
Male – Masculin					
Newfoundland – Terre-Neuve					x
Prince Edward Island – Île-du-Prince-Édouard				x	
Nova Scotia – Nouvelle-Écosse				x	
New Brunswick – Nouveau-Brunswick					x
Québec			x		
Ontario					
Manitoba		x			
Saskatchewan					x
Alberta					x
British Columbia – Colombie-Britannique		x			
Female – Féminin					
Newfoundland – Terre-Neuve					
Prince Edward Island – Île-du-Prince-Édouard		x			
Nova Scotia – Nouvelle-Écosse			x		
New Brunswick – Nouveau-Brunswick				x	
Québec		x			
Ontario			x		
Manitoba			x		
Saskatchewan				x	
Alberta					
British Columbia – Colombie-Britannique		x			

Source: Vital Statistics, Cancer Mortality by Site: 1960-1973, P. 27. Statistics Canada, 1977.

(National Health and Welfare 1980). Stomach cancer, for instance, was consistently higher in the eastern Census Divisions of Quebec, but lower in Saskatchewan and Ontario.

Cancer of the bladder, which was one of the top ten sites for males and females alike, was higher in Ontario and the Montreal region of Quebec. Similar regional differentials in terms of general cancer mortality and of the lung and breast were revealed by Field (1980) in his temporal and spatial analysis of mortality in Canada.

The author suggested that regional variations appeared not as much a result of the spatial sex distribution than as a result of the population age structure. The age group of 65 years and above was particularly influential. Also, geographic variations in general mortality and in selected causes of death, including cancer, showed only minor discrepancies. Field (1980) then pointed out that the regional mortality variations could be due to a complex of socioeconomic and life style factors, as well as environmental and man-induced elements.

3. Risk Factors And Differentials In Cancer Mortality

3.1 The Nature Of Cancer

Prior to the investigation of cancer related factors, it is necessary first to give a brief account of the disease. In simplistic terms, cancer is a tumor due to abnormal cell divisions. These wild cells differ from normal ones in their size and shape. More importantly, they do not respond to normal biological control, and will spread and destroy the functions of vital organs (Canadian Cancer Society 1981). The end result is then death to the host. Pyle (1971:46) drawing from a report by the American Cancer Society, summarizes the characteristics of cancer as follows:

1. A higher rate of cell growth and multiplication than normal cells.
2. Complete lack of tissue and organ boundary maintenance.
3. A microsceptic appearance which suggests immature rather than mature cells.
4. The spread in late stages to parts of the body far from the place of origin.

3.2 Genetic Versus Environmental Factors

Factors found causally related to cancer have been classified as either host or environmental elements. The host factors embrace genetic malfunctions like chromosomal and single-gene disorders (Mulvihill 1975), or genetic inheritance such as family susceptibility. Persons born in

families with past records of cancer for example, have higher risks of getting the disease themselves (Anderson 1975). Other genetically related cancer also includes those due to immune deficiency (Kersey and Spector 1975).

Environmental factors, comparatively, involve a much wider scope of elements each correlating with different cancer sites (Higginson 1969; Boyland 1967). Wynder and Gori (1977) broadly define environmental factors as carcinogens external to the host's body. Statistics Canada (1976) suggests that there are overlaps between life styles and environmental factors. The main difference between the two lie in the circumstances under which individuals come into contact with these elements. Life style factors are voluntary, but not so the environmental factors.

MacMahon (1975) however, distinguishes three specific dimensions of environmental factors. Firstly, there are physical agents that include primarily radiation. Secondly, diet and life style elements like smoking, alcohol consumption, food contaminants, and drugs as well as industrial carcinogenics are chemical agents. Lastly, the biological agents include sexual behavior and pregnancy.

Noteworthy is the effect of environmental factors through physiological development, which determines the rate and pattern of cancer growth (MacMahon 1975). Of the three types of environmental factors, chemical and biological agents are of special interest to the present study because of their close relationship with social constraints.

3.2.1 Smoking And Alcohol

Of all chemical agents, the association between cigarette smoking and lung cancer is perhaps best documented. Cutler (1955) in his early statistical review, concludes a concomitant increase between lung cancer and tobacco smoking. Comparison of smokers and non-smokers revealed substantially higher risk among the former than the latter. However, Cutler (1955) cautions that the connection itself does not provide sufficient ground to justify that smoking per se causes lung cancer. There are findings on other causal factors as well. The author notes the increase in female smoking does suggest, however, that any increase in future lung cancer death rates would help to set up the causal relationship.

In fact, evidence on rising lung cancer deaths for both sexes and the connection with tobacco smoking are available in the literature. Studies by Dorn (1955), Doll and Hill (1964), Hammond (1966; 1975), Preston (1970), Retherford (1973), W.H.O (1979), and Wynder and Gori (1977) have all related lung cancer in various populations or subgroups with tobacco smoking.

Veechia (1985) for example, detected that Italian male lung cancer deaths closely followed the peaks in cigarette sales in the 1920's, the 1950's and the 1970's. In Canada, the study by Best et al. (1967) and Miller (1977) have unamiously identified increased female lung cancer with cigarette smoking. Miller (1977) emphasizes the need of

antismoking programs, especially directed at young females.

Benjamin (1977) reports on countries with life expectancy of 55 years or more and indicates that female lung cancer deaths between 1960-1964 and 1970-1972 had increased more rapidly than for males. Within the United States, for example, the average female lung cancer death rate of 60-69 year olds jumped by 94 per cent during this period. In contrast, it was only 33 per cent for males.

In Canada, the figures, although lower, were equally alarming. Female death rate climbed by 65 per cent, and male death rate by 44 per cent. For such dramatic ascent among the females, the influence of genetic or biological factors was considered not significant. Rather, the deterioration was due to a growing number of females engaging in "...regular, as against occasional social indulgence in cigarette smoking, at work as well as at play..." (Benjamin 1977:122).

Tobacco smoking does not confine its effect on lung cancer alone. In their case-control study of cancer patients in Alberta, Canada, between 1971-1973, Wigle and others (1980) showed that cancer risk escalates in the tongue, mouth, pharynx, esophagus and bladder. Similar observations were reported by Hammond (1975) and Wynder and associates (1963). Hoover and Cole (1971) indicated the high risk of bladder cancer.

Alcohol is the next life style related chemical agent that has received wide attention in its relation with

certain cancer sites. When assessing the effect of alcohol, however, Rothman (1975) stated that few studies have succeeded in isolating its impact, owing to the confounding effects of other cancerous agents.

Indeed, a positive connection between alcohol and cigarette consumption has been reported by Conway and Ward (1981). Breslow and Enstrom (1974) also detect high correlation between three types of alcohol and smoking in 41 States of the United States. Despite problems of collinearity, which hamper the evaluation of individual effects, alcohol and tobacco are found significant in most cancer sites under their scrutiny.

Rothman (1975) cites studies by Wynder and colleagues (1957), Keller and Terris (1965), and Martinez (1969) which have successfully controlled the tobacco effect. Their findings relate alcohol consumption with cancer of the oral cavity, pharynx, and esophagus. Rothman (1975) added cancer of the stomach, liver, and rectum to the list.

3.2.3 The Diet Factor

Similar to alcohol, but even more difficult to single out, are dietary patterns. Berg (1975) clarifies that the relationship depends strongly on the combination of noxious agents, cofactors, and natural or acquired metabolic peculiarities of the host (1975:202). Moreover, despite the abundance of carcinogens in our diets, cancer is linked with dietary patterns but not with specific agents (Berg 1975).

Such a connection indirectly suggests that the cultural aspect of diet in relation to cancer should not be overlooked.

For example, there is a high incidence of stomach cancer in Japan for both sexes, and at the same time, findings of high salt content in their pickled vegetables and fish (Berg 1975). Kagawa (1978) associates the substantial increase of Japanese colon cancer between 1950 and 1975 with the adoption of a Western diet-- more meat and fat consumption but fewer traditional staple items like rice. Fat consumption has also been related to cancer of the breast and uterus (Armstrong and Doll 1975).

In the Western nations, cancer of the intestines has also been linked to components of diet. Logan (1975) reports from previous analysis that excess intake of animal fat, like beef, and too little fibre among others foods, are high risk factors. Coffee drinking, as another example, was held accountable for 24 per cent of male and 49 per cent of female bladder cancer (Staszewski 1980).

3.2.3 Occupation Hazards

While the list of food borne carcinogens can be extended, arguments exist on the effect of air-borne carcinogens upon the respiratory system. For example, considerable concern has been expressed regarding the impact on health of polycyclic aromatic hydrocarbons, which are released into the atmosphere through motor vehicle

emissions, heat and power generation, and refuse burning (Greenberg 1983). On the whole, environmental pollution including that from work areas, has led to heated debates over the relationship with the urban-rural differentials in cancer mortality.

Greenberg (1983) has traced the controversies elaborately and has recounted one school of thought which stressed the importance of air pollution and industrial exposures on the differentials. The counter-arguments, however, heavily stress on the tobacco smoking gradient as the prime factor. Retrospective analysis by Vena (1982), for example, refutes the notion that air pollution by itself could promote high lung risk. Instead, an interactive effect of smoking and long term exposure to air pollution is advocated.

Many specific occupations and industries have been reported as hazardous in relation to cancer development. Most evidence comes from case-control studies which involve comparison of death rates of workers at risk with those of the general public. Perhaps the best known and earliest study was conducted in London in 1775 by Pott, who linked the large number of cancers of the scrotum among chimney sweepers to their profession (Cole and Goldman, 1975). A recent study by Pyle (1971) indicated that cancer in the United States is largely concentrated in the Manufacturing Belt where death rates are higher in the North Central to the East coast districts.

Currently, the impact of asbestos has become a major concern because of its wide applications in daily products and its presence in work areas (Greenberg, 1983). Blot and associates (1979) attribute high cancer mortality in American shipyard workers to their frequent contact with asbestos and also point out similar situations in the European communities.

Delzell and Monson (1981) trace mortality of rubber workers in the United States between 1940 and 1974. Excess deaths due to cancer of the bladder, cervix and leukemia were noticed. It was speculated that, exposures to hepatotoxic chemicals that were commonly employed by the rubber industry could be a major cause.

In Canada, higher repiratory cancer deaths were found among workers employed in plants that produced vinyl chloride monomer and PVC (Theriault and Allard 1981). A study of Ontario miners revealed that those receiving compensation for silicosis exposure in 1940 had lung cancer deaths two to three times higher than the general Ontario population (Finkelstein et al. 1982).

Escalated cancer risk was also noted for Ontario uranium miners (Chovil 1981). However, no significant excess deaths were found for these industries in a later nation-wide analysis. Howe and Lindsay (1983) link death records in 1965-1973 to occupational information retrieved from a ten-percent sample of the Canadian labour force from 1965 to 1969. No excess cancer deaths were unveiled for the

mining, chemical, or even the manufacturing industries. In terms of occupations, workers in the service sector and professionals had a high risk of cancer in the bucal cavity and the pharynx, and of overall cancer, respectively.

Howe and Lindsay (1983) explain that the absence of previously-confirmed high cancer deaths in some industries or occupations might reflect data limitations, especially the minimal latency period permitted by the data. In the latter regard, Cole and Goldman (1975) suggest an incubation period of about 20 years for most occupational carcinogens.

In addition, the authors warn against confounding effects derived from the close connection between social class and occupation. Correlation between occupation and risk of cancer can therefore be spurious. An empirical example is the finding of reduced stomach cancer risk among Swedish soft-coal miners after control for social class.

3.2.4 Fertility and Female Breast Cancer

Unlike cancer of many other sites, cancer of female sex organs has been related to sexual behaviors as in cervix cancer, and pregnancy in breast cancer (Henderson et al. 1975). Considering the latter, MacMahon et al. (1973) indicate in their study that fecundity and age at first pregnancy have protective effects against breast cancer. Yet, lactation is not an important element. Henderson and colleagues (1975) classified five groups of interrelated and interdependent risk factors in breast cancer. The four

factors which have more reliable evidence pertain to demographic, menstural, reproductive, and hormonal aspects.

Burns and others (1981) have tested some of these factors within the Canadian context. Their case-control study employed data on women 30 to 79 years of age, who had registered in 1971-1975 for breast cancer diagnostics at a clinic in Edmonton, Alberta. Major findings from this analysis include an association between high breast cancer risk and age at first birth.

The risk doubled for females who had their first child at the age of 30 and older. The association remained valid in both groups of pre- and postmenopausal women. Regarding the interval between menarche and first child birth, the pattern was almost identical. Risk rose with the interval length. Lastly, contrary to common observation was the absence of a relationship between parity and risk of breast cancer.

In an earlier study of Quebec and other provinces in Canada, an inverse relationship between breast cancer and fertility was reported. Specifically, high death rates for the 55-64 years-of-age cohort appeared to correspond to their low fertility in 1934-1938, when they were around 15-19 years old. Also, low death rate for females 40-54 years old corresponded to the peak of fertility in 1945 to 1963 (Fabia et al. 1977). Burns and associates (1981) explain that the absence of a parity effect in their analysis could be due to the independence of parity and age

at first birth. Such a phenomenon has been documented for the city of Edmonton (see Morah, 1977).

A recent study by Wigle (1977a) also highlights the relationship between fertility and breast cancer. Canadian breast cancer death rate for age groups between 30 and 69 years was found correlated with the percentage of females first married at age 15-19, which was used as an indicator of early pregnancy. Mortality rates for women 40-44, 45-49, 50-54, and 55-59 were observed to correlate with their cohort fertility rate at the age of 20-24.

The three older age groups had a significant correlation coefficient of -0.60, -0.86 and -0.80 indicating an inverse gradient with fertility. However, scattergrams provided in the report reveal distinct heteroscedasticity and non-linearity between fertility and breast cancer. Thus, the real association and strength are incorrectly measured and might not be the same as indicated. Nevertheless, the study makes obvious the need for a further probe along this route.

3.3 Cancer Mortality Differentials

3.3.1 Age Differentials

Chances of death are not homogeneous across all ages. Empirical evidence shows that the risk subsides following the initial peak during infancy and then remains more or less stationary till the second peak in early adulthood.

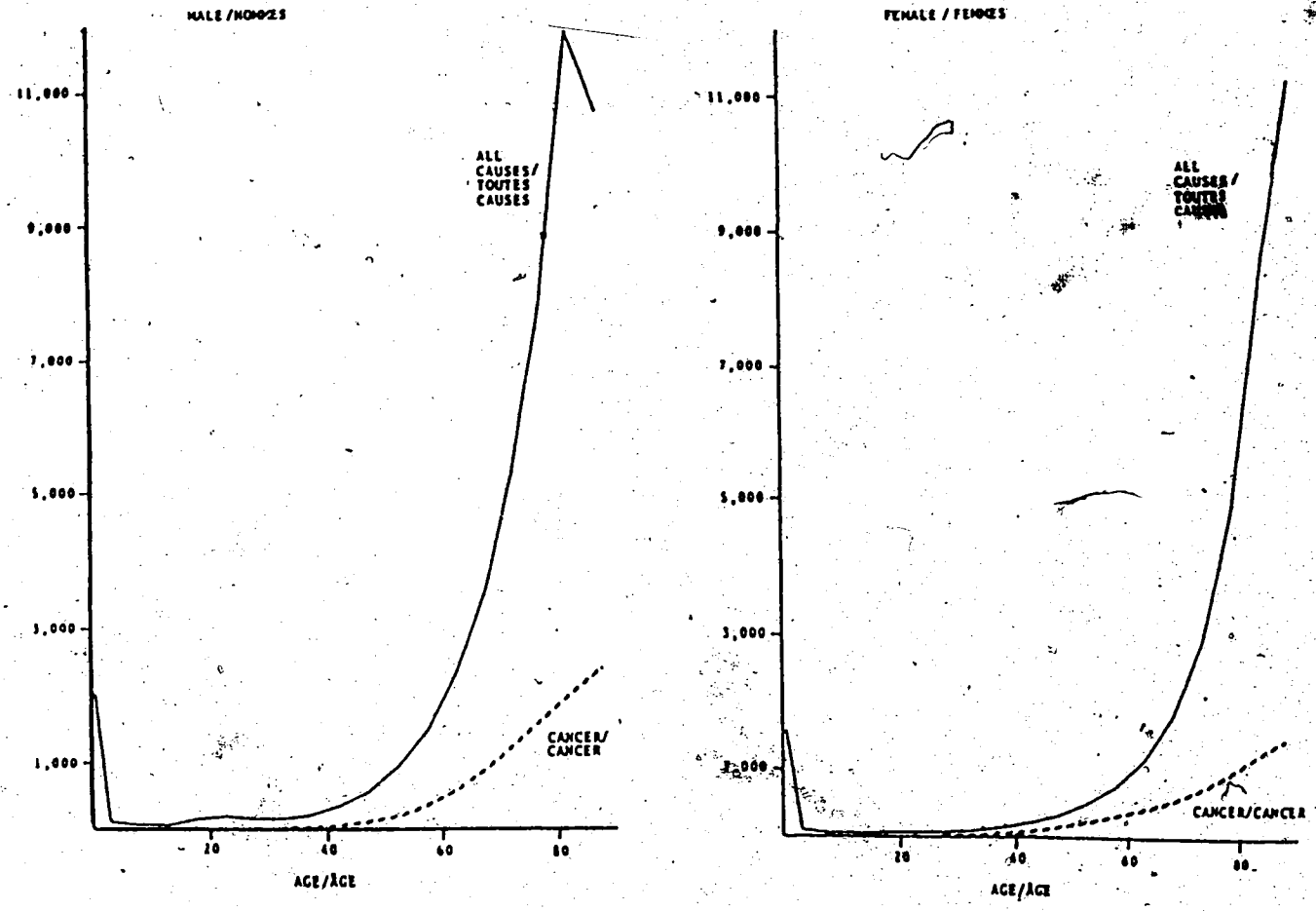
From mid-life onwards, however, probability of death escalates progressively with age (Petersen 1975). Though this age pattern is fundamental to all levels of mortality, it is largely shaped by the prevailing major causes of death.

When the level of death rate moves from high to low, the U-shaped age pattern of mortality becomes J-shaped. This reflects, on one hand, the replacement of infectious diseases by chronic diseases as the predominant causes of death (U.N. 1963; Preston 1976). On the other hand, it signifies a shift in the risk of mortality from the young to the old.

The age pattern of cancer mortality approximates this J-shaped curve with the exception of a less "hooked" curvature at young ages and with a more steep ascent on reaching middle ages. These pecularities cast back the seriousness of cancer after as a cause of death at middle ages and above. The trajectories of the overall and cancer mortality in Fig. 3.1 show diversities.

The fact that cancer increases with age can draw two explanations. Firstly, the chronological aging of an individual is accompanied by his biological aging, which results in physiological functional changes and celular changes of senescence (Sacher 1980; Manton 1982a). Though such progress does not directly translate into cancer, the decline of the host's immunological defence could produce higher risk of the disease (Health and Welfare Canada

Fig. 3.1 Age-Sex Specific Overall and Cancer Death Rates, Canada, 1931-1974



Source: Cancer Patterns In Canada: 1931-1974, P. 189, Fig. A-1. National Health and Welfare, Ottawa, 1976.

1976a).

Secondly, the long latency period between carcinogenic exposures and manifestation of the consequences implies that cancer primarily show itself in late adulthood. A latency period of 20 years or more has been suggested for most cancers (Cole and Goldman 1975).

3.3.2 Cancer: Old versus Young

Despite the apparent advantages of the young, it should not be assumed that child cancer mortality is negligible. A recent report by West (1984) on the developed countries in 1955-1974, reveals the prevalence of cancer of the nervous system, and bone as well as leukemia among children. Notwithstanding the overall downward trend, cancer in some nations accounted for an alarming 20 per cent of all childhood deaths.

The United Nations (1982) reports stablized death rates from cancer for age groups between 5 and 34 years but indicates a substantial increase for older groups. The largest increment of 22 per cent is noted for the 60-74 years group. Such deterioration is common in most of the 37 countries included in the study, and the continued worsening of lung cancer is considered the prime promoter.

In their study of international mortality trend among the elderly, Lopez and Hanada (1982) came up with the same conclusion regarding the increase of cancer among males 65-74 years old. Other cancer sites that could have helped

to reduce progress in male cancer mortality include the intestine and the bladder. Death rates related to both of these sites increased between the 1950s and the 1970s (Logan 1975b; Staszewski 1980).

For females, the mortality conditions are a bit better. Death rates declined in all groups except in the 55-64 category. Of the age groups with improvements, the most impressive is the 25-44 group (U.N. 1982). There is a tendency to associate such progress with breast cancer, the major site for females at child bearing ages. Unfortunately, analysis showed deterioration (Logan 1975a).

In fact, breast cancer has underlined the increase in overall cancer mortality among older women (Lopez and Hanada 1982). Lung cancer which is the second leading cancer site among females, has increased also and did not show any improvements. Since there is little evidence connecting the overall decline with these cancer sites, it appears that the females have benefited from reduced death rates related to other sites. For example, cancer of the intestines and esophagus had decreased among the females but not among the males (Logan 1975b).

3.3.3 Age Differentials in Canada

Regarding the age patterns in Canada, various reports have indicated that changes over time generally resemble those of other developed nations like the United States and Australia (D.B.S. 1965) and of the world as a whole (U.N.

1982). Reports indicate that overall cancer mortality for Canadian males 25-74 years of age would have declined if not for the increase in lung cancer deaths. For females, the level would have lowered even more (Health and Welfare Canada 1976).

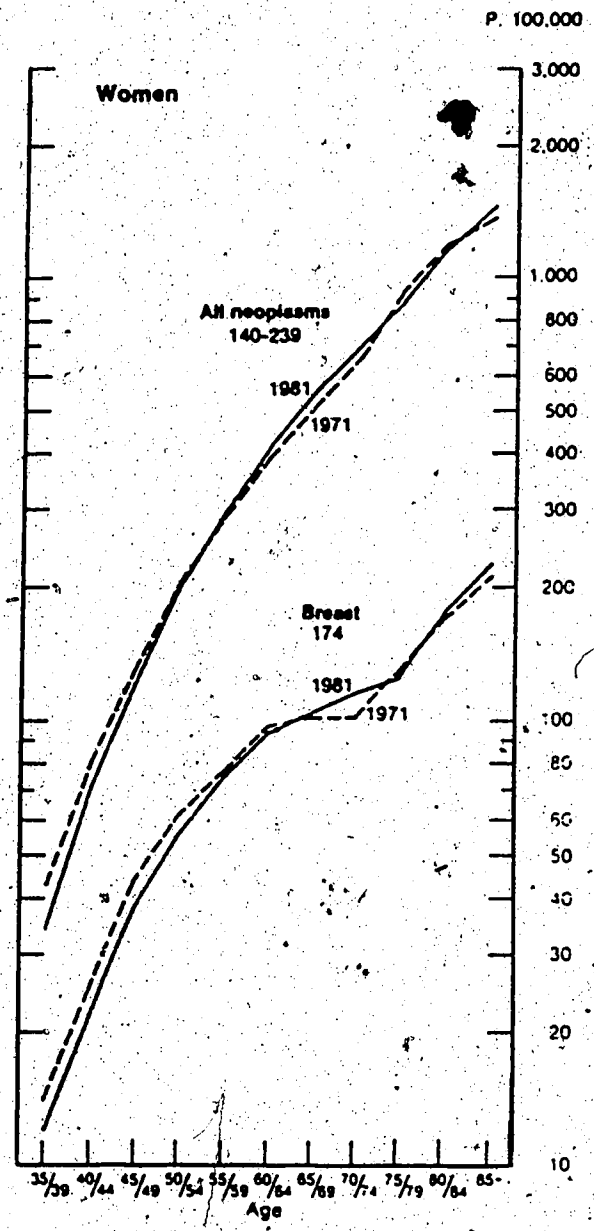
Nevertheless, there are some variations in individual sites. Perhaps female breast cancer has deviated the most in this respect. Age-specific breast cancer death rate among Canadian women had declined by 10.2 and 8.3 per cent in the age groups of 35-44 and 45-54 years, respectively, between 1965-1969 and 1971 (Fabia et al. 1977).

Further improvements, illustrated in Fig. 3.2, were reported for the following decade. Evidence tends to suggest that, within Canada, improvements in female cancer mortality could be related to progress in breast cancer. Yet, annual breast cancer death rates between 1931-1972 reveal no impressive declines (Health and Welfare Canada 1976). Age differences in cancer death rates, and their changes over time are illustrated with Canadian age specific cancer mortality rates by sex in Fig. 3.3.

3.3.4 Sex Differentials in Cancer Mortality

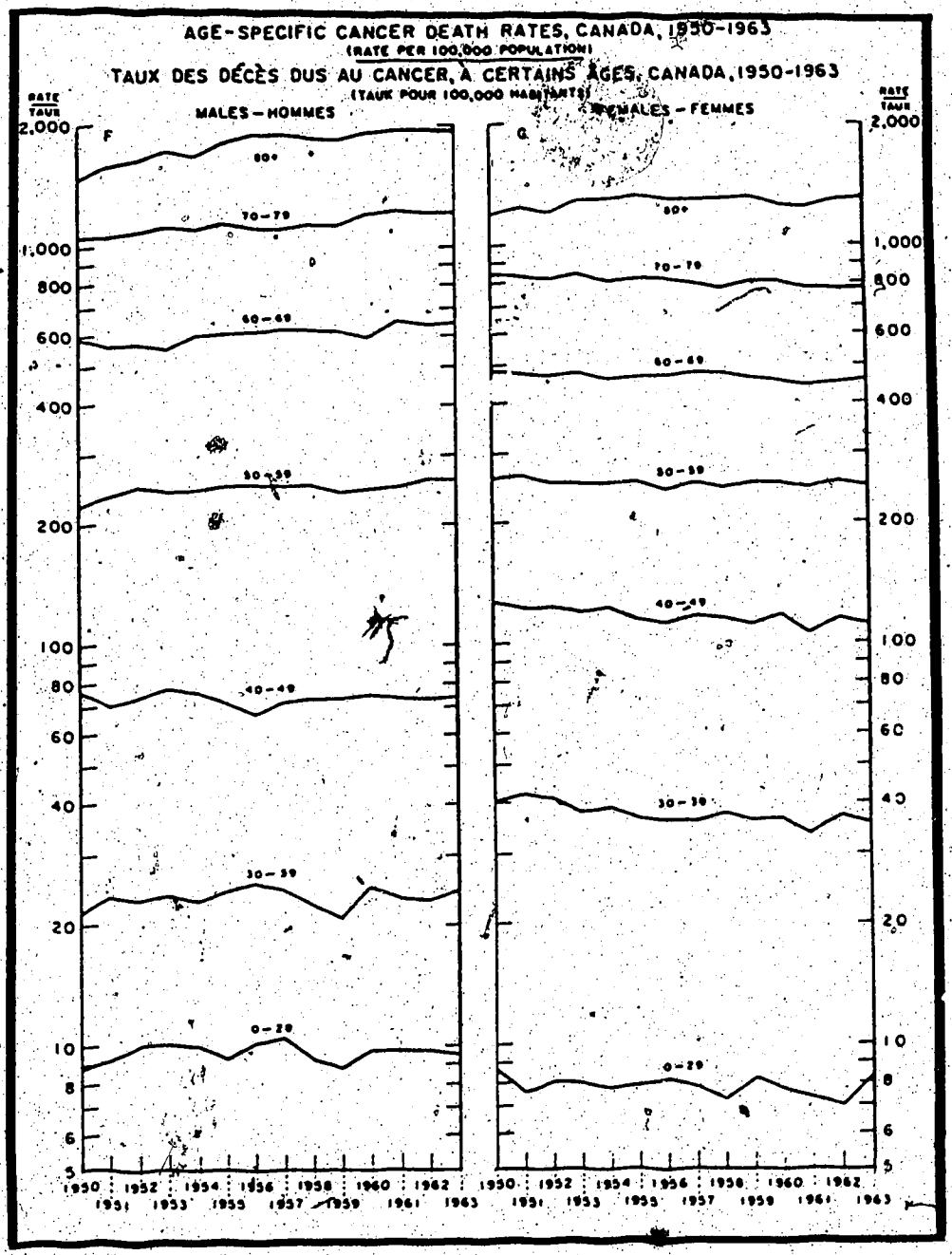
From Fig. 3.3, it is obvious that male cancer mortality exceeds that of females in all age groups except between 30 and 50 years of age. These crossovers become more apparent when both male and female age-specific death rates are plotted within the same frame as in Fig. 3.4. Females have

Fig. 3.2 Age Specific Female Breast Cancer Death Rate, Canada, 1971-1981



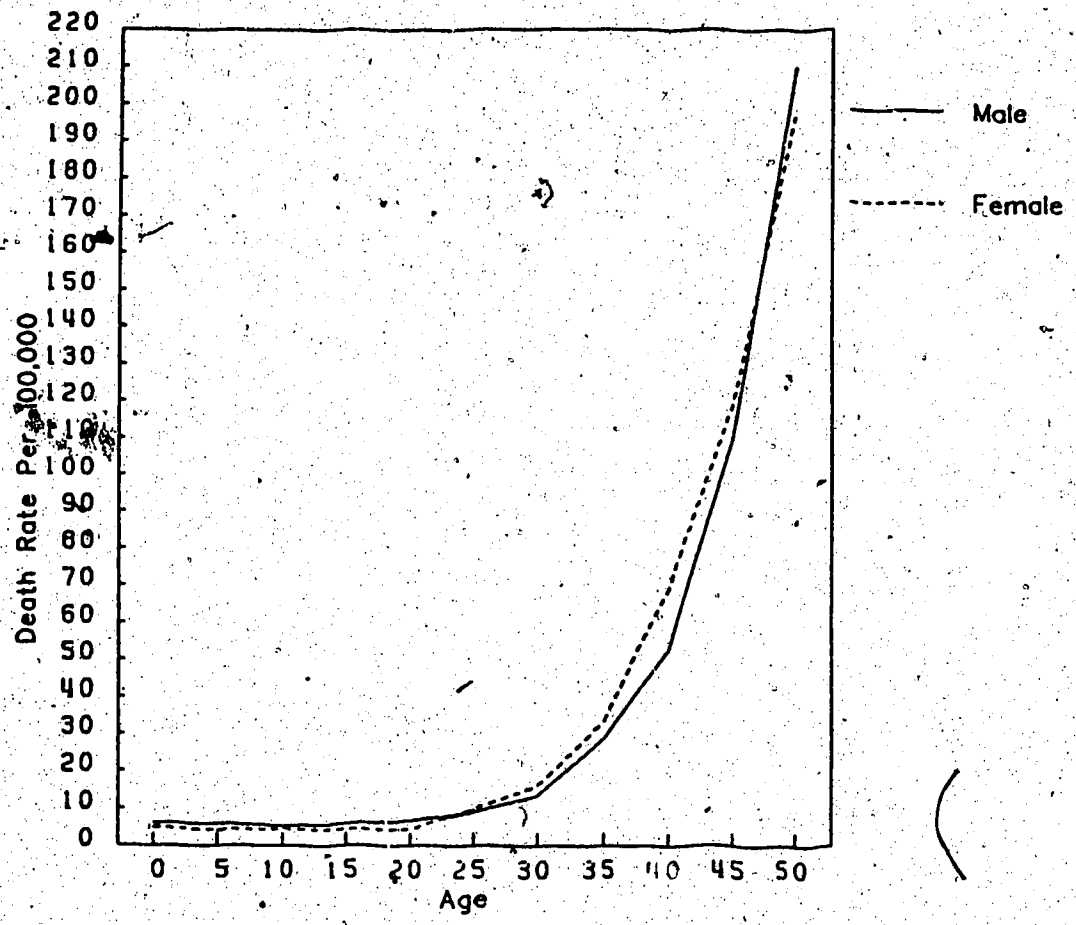
Source: Extracted from Current Demographic Analysis. P. 87. Chart VIII. Statistics Canada, Ottawa. 1985.

Fig. 3.3 Age-Sex Specific Cancer Death Rates, Canada, 1950-1963



Source: General Cancer Mortality: 1950-1963, P. 42, Chart F-G. Dominion Bureau of Statistics, Ottawa, 1965.

Fig. 3.4 Age-Sex Specific Cancer Death Rates, Canada, 1981



Source: Data from Vital Statistics, 1981, Vol. III, Causes of Death. Statistics Canada, 1983.

higher cancer death rates than males between the ages of 23 and 50, but the patterns are reversed at younger and older ages. Similar differentials emerge when life expectancy gains after cancer elimination are considered.

From Table 2.2, we could see that males have smaller life expectancy gains than females prior to age 65. Such a pattern arises from the fact that there are more female than male cancer deaths in early ages, largely from cancer of the breast and cervix (Statistics Canada 1984). Also, fewer female than male deaths due to other causes in the age bracket 20-64 years increase the impact of cancer.

Recent analysis by Veevers and Gee (1983) on Canadian sex differentials, revealed excess male deaths from accidents in the age group of 25-34 and from circulatory diseases other than cancer, in the 65-74 age group. Together with a large decline in maternal deaths, these three groups of causes of death contributed significantly to the widening sex differential in Canada between 1971 and 1981. The observed sex mortality differentials are not unique to cancer, nor are they limited to Canada.

Studies of the United States, England and Wales, and countries with reliable information on age-sex specific causes of death have found results similar to the Canadian observations (Verbruge 1976; Preston 1976; Birkin 1981; Retherford 1975; Enterline 1961; Gee and Veevers 1984; Kitagawa and Hauser 1973; U.N. 1979b; 1982). Although these studies have different focuses, temporally and

geographically, three major generalizations can be drawn from them.

First, the gap between males and females has widened to the point where differences between the two sexes within a country is larger than the differences for either sex between nations. Secondly, infectious diseases have become negative contributors to sex mortality differential while chronic diseases like cancer and accidents are positive promotors.

Lastly, substantial improvement in female mortality especially in deaths related to pregnancy and child birth, has played a crucial role in making the male death rate excessive. Though rises in female lung cancer deaths work in reducing the male-female gap, this reduction is compensated for by the continued worsening of male lung cancer.

These findings are reinforced by Lopez (1983) in perhaps the most detailed analysis of international and national trends in sex mortality differentials from 1900 to 1978. He classified Canada as part of the Anglo-Saxon group consisting of countries like the United States and Australia, which is characterized by "...little change in sex mortality differentials... prior to 1930 but a dramatic widening occurring since..." (Lopez 1983:88).

Noted for this Anglo-Saxon group and for the Western and Northern European countries is its bimodal distribution of sex differentials in contrast to the absence of a second peak for countries from Eastern Europe. Such distribution is

due to a large number of motor vehicle and violent deaths in early adulthood and to heart diseases and cancer at old ages. Lopez (1983) estimates that cancer alone accounts for about one fifth of the total sex differential. Of this discrepency, 70 per cent is by male lung cancer.

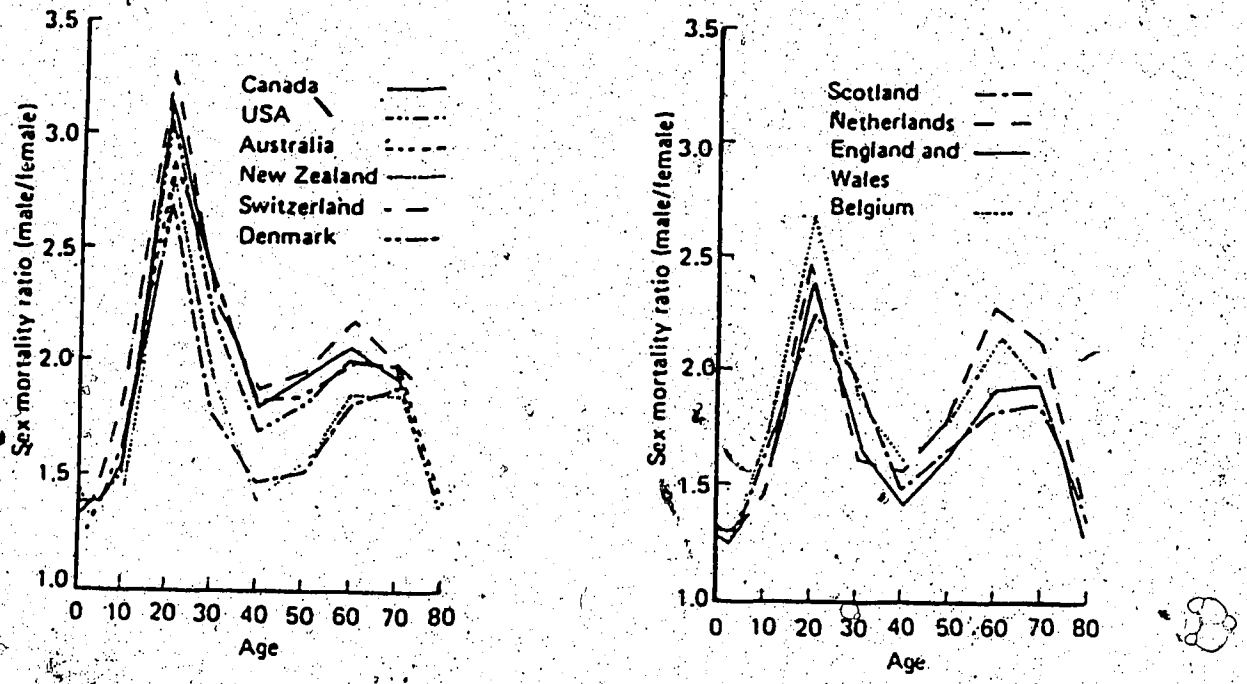
Breast cancer tends to reduce the differencies by about 6.6 per cent. Yet, the contribution of 4 per cent of excess male stomach cancer levels off most of the effect. The distribution of age-sex differential reported by Lopez (1983) is shown in Fig. 3.5, and changes in the contributions of various causes of death to the sex differential are summarized in Table 3.1.

On the basis of the above evidence, it appears that the sex mortality inequality in favor of females is universal and an established fact. Exceptions to this rule are observed over a number of decades in a few developing countries notably India, Pakistan, Bangladesh and Ceylon. (U.N. 1964; D'Souza and Chen 1983). Even among developed nations, excess female deaths were not uncommon before the 1930's (Stolnitz 1956b). In some European communities, the reversal came as late as the end of World War II (Wunsch 1980).

3.3.5 Factors Behind Sex Differential

Although explanations for the unusual sex differential in the above-mentioned developing countries do not rule out under-registration of female deaths, high childhood and

Fig. 3.5 Sex Differentials by Age, in Selected Developed Countries, 1975



Source: Sex Differentials In Mortality, A.D. Lopez and L.T. Ruzicka, edit., P. 71. Canberra, 1983.

Table 3.1 Contributions of Selected Leading Causes of Death to Sex Differentials, for High Life Expectancy Countries, 1975

Nations with HIGHEST Male-Female Difference								
Cause of death	Titles in the A-list of the ICD (8th Rev.)	Average contribution of cause: %	Average contribution of cause: per 100,000	Country	Observed difference per 100,000	Deviation from overall average	Component due to mortality of: Males	Component due to mortality of: Females
Malignant neoplasms	A45-A60	19.6	139.9	France	237.4	97.5	67.2	30.3
				Czechoslovakia	235.8	95.9	111.6	-20.7
				Belgium	210.5	70.6	79.8	-9.2
				Finland	201.6	61.7	30.2	31.5
				Netherlands	196.6	56.7	58.3	-1.6
Malignant neoplasms of lung	A54	14.3	102.5	Netherlands	180.6	38.1	70.1	-8.0
				Belgium	168.5	66.0	60.1	5.9
				UK: Scotland	107.7	60.2	90.8	-30.6
				Finland	156.9	56.4	48.7	7.7
				Czechoslovakia	156.7	55.7	49.2	6.5
Cardiovascular diseases	A80-A88	48.8	348.1	Finland	627.7	281.8	314.1	-32.5
				UK: N. Ireland	512.3	166.2	277.1	-110.9
				UK: Scotland	484.3	138.7	257.6	-119.4
				UK: England & Wales	441.4	95.3	97.4	-2.1
Ischaemic heart disease	A83	34.8	249.5	Finland	525.8	270.3	320.2	-53.9
				UK: N. Ireland	439.4	189.9	298.3	-108.4
				UK: Scotland	414.6	165.1	284.5	-119.4
				UK: England & Wales	368.5	119.0	162.7	-43.7
Cerebro-vascular disease	A85	5.7	41.0	Japan	133.5	72.5	125.3	50.8
				Portugal	106.2	65.2	182.0	116.8
				Czechoslovakia	71.8	30.8	88.0	57.2
				Hungary	66.8	27.8	65.9	38.1
Respiratory diseases	A89-A96	10.1	72.2	UK: Scotland	157	87.0	75.3	28.3
				Czechoslovakia	114.2	41.5	54.3	12.8
				German Dem. Rep.	113.4	40.7	38.7	2.0
				UK: England & Wales	107.4	34.7	69.3	-7.6
Bronchitis, emphysema, asthma	A91	6.3	45.0	German Dem. Rep.	97.7	47.7	49.1	-1.4
				Czechoslovakia	84.3	38.9	47.9	-8.0
				UK: England & Wales	67.5	22.5	26.9	-3.4
				UK: N. Ireland	66.3	21.3	29.0	-8.0
				Australia	62.5	17.5	19.9	-2.0
				UK: Scotland	62.3	17.3	26.5	-9.2
Violence	AE138-AE150	10.0	77.0	Finland	144.4	67.4	78.1	-10.7
				Hungary	120.3	43.3	61.0	-17.8
				Poland	117.1	40.1	45.2	-5.1
				Portugal	120.7	43.7	61.3	-17.6
				Austria	108.1	31.1	43.7	-12.6

Source: Sex Differentials In Mortality, A.D. Lopez and L.T. Ruzicka, edit., P. 78, Canberra, 1983.

maternal deaths are concluded as the major reasons (El-Badry 1969). A recent study on Ceylon, however, reveals that a reversal has taken place since the beginning of the 1960s, causing an ever increasing male-female death ratio (Nadarajah 1983). Not surprisingly, the change is in accordance with the familiar switch from high female maternal deaths to high male deaths from degenerative diseases.

The parallel which exists between Ceylon and the developed countries is the increase in sex mortality differential when predominant causes of death in either sex changed. This parallel prompts the question of whether females are biologically more resistant to non-pregnancy related diseases, or whether females have fewer contacts with exogenous factors that cause high male deaths? This issue of nature vs nurture frequently addressed in general mortality is no less important when considering cancer.

Decomposing the sex differential according to genetically inherited factors and those from one's social milieu is so complicated that it has not been very successful (U.N. 1984). Difficulty occur not only in timing when one of these two groups of factors overtakes the other, but more so in assigning weights when interactions are suspected.

Genetically, the extra X chromosome in females has been found to provide them with greater immunity against infectious diseases, and fewer recessive disorders than for

their male counterpart. Also, endogenous female sex hormones produced by the ovary may decrease the risk of certain heart diseases (Waldron 1976, 1983a, b; Graney 1979).

The classical test of the hypothesis that males are biologically the weaker sex was performed by Vance and Madigan (1956) on a sample of Brothers and Sisters of the Roman Catholic Church between 1900 and 1954. Findings showed that sex mortality differential of the test population approximated those in the general population, and females still outlived males. Since life styles of both sexes in the test population were assumed to be alike, Madigan concluded that females were biologically less vulnerable, particularly to chronic diseases than males. However, the conclusion is not well accepted by others.

Waldron (1976) points out that Madigan's research design had neglected past smoking and drinking habits of the Brothers and Sisters, which could have been discrete factors in producing the differential. The researcher stresses that genetic effect on sex mortality differential varied depending on environmental or life style factors. In accidents, heart diseases and lung cancer, life style elements are strongly deterministic.

Graney (1979), who shares relatively the same argument, analyzed the issue from a sociological approach, wherein the evolution of genetic sex differential is viewed as a social process. By tracing the leading causes of death according to their order of appearance over the life course, Graney

(1979) concludes that "...biological disadvantages of the male are a decreasing function of chronological age, and that after adulthood...social factors became the primary explanations..." (Graney 1979:18).

Hetzel (1983) reviews the trends of lung, gastro-intestinal, and endocrine cancer in the United Kingdom, Australia, France, and the United States. He points out that the differences in animal fat, tobacco smoking and alcohol consumption are associated with differentials in the above cancer sites. The results prompted him to state that even biological factors are crucial in sex mortality differential, and that their impact could not hold after age 65.

Retherford (1975) on the other hand, examined increased sex mortality differentials in the United States, New Zealand, and England and Wales between 1910-1965. The rapid increase in deaths due to accidents, heart disease, and cancer led him to conclude that genetic codes could not have changed fast enough to allow for the observed differentials. Environmental factors and sex behavioral differences are primarily responsible.

In defining the relative significance of the genetic-biological and the environmental-socioeconomic influences, Ruzicka and Lopez (1983) perceive sex differential at any given level of mortality as a product of the interactions of these influences. When mortality level declines, the responsible improvements in non-biological

factors like better health care and nutrition work to increase females' chances of survival. At the same time, the genetic-biological factors help to advance female longevity. However, once female life expectancy exceeds that of the male, life styles become "...the essential determinant of one sex's excess mortality..." (Ruzicka and Lopez 1983:478).

The effects of genetic factors on cancer sex differentials are not conclusive, and it is not easy to establish or to rule out their possible influences (Waldron 1983b). Certain types of cancer for example, polyposis of the colon and some forms of retino-blastoma, are related to inherited genetic factors. Still, we cannot discount environmental factors in the developments of cancer by sites in general (Health and Welfare Canada 1976).

3.4 Ethnic Differentials in Cancer Mortality

Similar to age and gender differentials, members of different ethnic groups show differential by causes of death. However, understanding the factors behind these differentials has been confined to inherited single-gene disorders which are an extremely rare occurrence. There has been less success in isolating factors in deaths with respect to multiple causation. Yet, this group of diseases has dominated the causes of death structure in developed countries (Damon 1969).

Studies have increasingly paid attention to inequalities in socioeconomic status, life style,

occupational hazards, and access to health services as the prime factors in ethnic mortality differentials (Krueger and Moriyama 1957; U.N. 1973; Damon 1969). In cancer, perhaps with the exception of skin cancer, origins of most ethnic differentials have been traced to social status or sub-cultural traits (Pyle 1971). Studies on American whites and blacks, for example, relate excess cancer deaths among the latter with smoking, industrial hazards, dietary habits, and alcohol consumption (Burbank and Fraumeni 1972; Henschke et al. 1973).

A more recent work by Michielutte and Diseker (1982) set out to compare the level of cancer knowledge between the United States blacks and whites. The report reveals that the blacks receive comparatively brief and simple information through television and radio. The whites, on the other hand, obtain more detailed information from newspapers and pamphlets. Generally, the blacks are less knowledgeable than the whites about cancer. Such a pattern remains even after education and income inequalities are statistically controlled.

The findings are in agreement with those revealed earlier by the American Cancer Society (Michielutte and Diesker 1982, c.f). However, the Michielutte-Diseker study found higher income blacks more informed about cancer than those in low income brackets. The differences in knowledge over the income groups highlight the interaction between ethnicity and other socioeconomic factors in the formation

of differentials.

In this regard, the crossover effect is not unrelated (Manton et al. 1979). American blacks, for example, have higher mortality than whites in most age groups, but this is reversed at advanced ages. Manton (1980) attributes this phenomenon to the selection process, which left the most robust persons within the disadvantaged group to survive untill old age, thereby contributing to lower death rates.

For cancer, the crossover between the whites and the blacks occurred at the age of 60 and above in 1950, but was around 80 in the 1970's. Increasing socioeconomic equalities and better access to health services were regarded as the factors delaying the crossover (Markides 1983). Apparently, the issue of ethnic differentials in cancer has to be treated simultaneously with other socioeconomic or environmental factors.

Interest in the relation between environmental exposures, behavioral patterns and ethnic differentials in cancer has prompted numerous studies to focus on the foreign- and native-born populations. Others have selected migrants and the host population for comparison. Studies on the Chinese and Japanese population in the United States have provided valuable information (Smith 1956; King and Haenszel 1973; Haenszel and Kurihara 1968; King et al. 1985).

It is generally observed that, for both ethnic groups, there are excess deaths in some specific cancer sites but

few deaths in others. For example, the Chinese have excess deaths in nasopharyngeal and liver cancer and the Japanese in esophagus and stomach cancer. Yet, both these groups have lower death rates in prostatic and breast cancer than the American-white population.

The effects of environmental and behavioral changes are suggested when the descendents of these two ethnic groups are found to have lower death rates in cancer sites common to their ancestors, and high death rates in those cancer sites frequent among the host population.

3.4.1 Ethnic Differentials in Cancer Mortality in Canada

Within Canada, information on cancer differentials among ethnic groups is relatively limited. However, available studies do point to some differentials. Schaefer et al. (1975) studied the changes in the patterns of cancer sites among the Canadian Eskimos. The authors noted, on one hand, that some of the cancer sites common to this ethnic group are likely to be related to their own life styles and habits. The relation between the salivary gland tumours and the practices of chewing leather.

On the other hand, they also suggested that increases in lung cancer, among others, perhaps can be linked to adaptations of "external" life styles. Similar findings on the influences of life styles have also been reported by Choi (1968). He studied ethnic distribution of cancer of the gastrointestinal tract in Manitoba in 1956-1965. Death

ratios revealed excess stomach cancer deaths among the native- rather than the foreign-born.

Detailed breakdown of the death ratios by place of birth showed immigrants from Poland, Scandinavia, and the Soviet Union (Ukrainians) were particularly associated with high risk. Moreover, the risk declined in the descendents of these immigrants and was lowest among the native-born Manitobans.

Drawing on Dungal and Sigurjonsson (Choi 1968, c.f) regarding the popularity in Iceland of smoked and singed food, which had been found carcinogenic, Choi (1968) suggests that diet factors and host habits are likely responsible for the high stomach cancer deaths in these ethnic groups. Changes in the risk of colonic and rectal cancer among the Eastern European immigrants, from a low among the first generation immigrants to a high among the Canadian born descendents, also provides support for the greater importance of environmental changes over genetic factors.

Cook and others (1972) strongly recommend the analysis of cancer mortality in relation to country of birth. Their study on cancer morbidity in 1966 Ontario utilized hospital discharge records, which distinguished four ethnic groups according to their surnames. Distinct cancer distribution by sites were observed among the Italians, the Polish, the French, and a residual group of other origins. For example, the Italians were found to have excess incidence of cancer

of the digestive system but low in incidence of prostate cancer.

A more detailed study of the Ontario ethnic groups was recently conducted by Newman and Spengler (1984). Cancer mortality of six ethnic groups, including the Italians, the British, the Germans, the Dutch, the Polish, and the Soviets were compared with cancer mortality among the Canadian-born and with death rates in the country of birth. The results shown in Table 3.2 indicate the high and the low risk of each ethnic group in relation to various cancer site, and to each group's country of birth.

It is suggested that the differentials observed could be partially explained by the selective nature of migration. Immigrants could be healthier than those who remained behind. Also, the degree of integration could have contributed indirectly. Greater integration or assimilation into Canadian society can bring more dietary and other life style changes that can be either detrimental or beneficial in terms of cancer.

So far, the discussions on cancer differentials between ethnic groups have emphasized variations in environmental exposures and behavioral patterns. In a recent analysis of the mortality inequalities between the Canadian indigenous population of French and British origin, and the aboriginal population in 1950-1952 and 1970-1972, another important aspect of racial differential was revealed (Trovato 1985). The observed mortality inequalities are interpreted as a

Table 3.2 Cancer Differentials in Canada, for Selected Ethnic Groups, 1969-1973

	Immigrant groups			
	Compared with Ontario residents born in Canada		Compared with population of country of birth	
Cancer site	Excess of deaths	Deficit of deaths	Excess of deaths	Deficit of deaths
Stomach	British Italians Dutch Poles Soviets			British Italians Germans Poles
Colon/ rectum		Italians Germans (W) Dutch Poles (W) Soviets	Poles	Italians (W) Germans (W)
Lung	British Poles (M)	Italians Poles (W)	Poles (M)	British Italians (M) Dutch (M)
Prostate		Italians Poles	British	
Breast	British	Italians Germans Poles Soviets	Poles	British

*An SMR was considered significant, showing an excess or a deficit of deaths, when the 95% confidence interval did not include 100.0. W = women only. M = men only.

Source: A.M. Newman and R.F. Spengler, Cancer Mortality Among Immigrant Population In Ontario, 1969 Through 1973. Canadian Medical Association Journal, Vol. 130: 403, 1984.

consequence of minority status attached to the socially and economically disadvantaged group. In this case, the native population of Canada.

This study, though focused on the Canadian native population, highlights the possibility that exposure to some of the cancer risk factors like occupational hazards are not voluntary. Yet, the extent to which minority status affects cancer differentials, either as a whole or by individual sites, in comparison to other known risk factors awaits to be answered.

Perhaps in Canada more than elsewhere, the government's encouraging ethnic groups to preserve their cultures adds further plausibility to differentials in cancer mortality. Despite the process of integration or assimilation, many culturally related habits and behavioral patterns, either detrimental or beneficial in terms of cancer risk, would be retained. Consequently, cultural preservation could indirectly affect mortality from cancer for the various ethnic groups. In view of such possibilities, the analysis of differentials and the factors underlying them are informative and useful to the health practitioners.

3.4.2 Testing of Ethnic Effects

To test ethnicity as a risk factor in cancer mortality, Wellington and colleagues (1979) adopt a more direct approach. Instead of the usual comparison between observed and expected mortality of selected ethnic groups, the effect

of ethnicity was assessed as an independent variable along with others, using direct estimation techniques. The French, the British, the Scandinavians and other Europeans in the United States are the objects of analysis.

Cancer inducing factors being studied include air pollution, temperature, income, and alcohol and cigarette consumption. Methods like principal component, and discriminant and regression analysis, which allow direct and simultaneous assessment of all variables, were used. Total cancer site specific mortality were examined, and findings from the analysis are informative and detailed. A number of observations regarding the ethnic effect are noteworthy.

First, the ethnic groups that have maintained either a high or a low risk in relation to various cancer sites in their homeland remain so within the United States. This is attributed to either genetic or cultural factors. Secondly, non-ethnic variables respond differently to various cancer sites. Thirdly, these non-ethnic variables changed their significance and strength as ethnic factors were entered into the equation.

The last two points indicate that ethnic groups tend to have some individual impact on cancer. Also, the changes reflect the existence of interactions between ethnical, environmental and behavioral variables. Unfortunately, such interactions have not been examined fully. Besides providing information on ethnicity and cancer, Wellington and associates (1979) demonstrate the practicality of combining

an ecological approach with direct estimation techniques in cancer research.

In his study on race and geographic distribution of cancer in Los Angeles in the United States, Steiner (1954) summarizes the intricate relationship between racial, hereditary, cultural, and environmental factors as follows:

> ...racial differences in cancer tend on critical analysis to indicate environmental rather than hereditary factors in etiology. The geographical differences tend to prove to be environmental rather than racial per se. The hereditary differences tend to be explained by cultural rather than genetical factors. The genetical factors is difficult to recognize.[but they are] important in determining whether environmental factors are at sub- or super-threshold carcinogenic levels... (P. 328)

3.5 Socioeconomic Differentials in Cancer mortality

In previous discussions, inequalities in cancer mortality pertaining to age, sex and race were addressed. Yet, differentials exist with respect to other characteristics as well. Vallin (1980) emphasized that in a framework of biological constraints set within a given geographic context, the major mortality differentials are of cultural and socioeconomic origins. In the latter regard, the existence of an inverse relationship between socioeconomic status, or social class, and mortality has been well documented.

Regardless of the multipicity of the indexes used to measure the concept of socioeconomic status, Stockwell and Groat (1981) note that there would always be a lowest status

group identified with the highest death rate. Such observation holds true for both developed and developing countries and for many causes of death, including cancer. It should be emphasized, though, that socioeconomic status seldom acts upon mortality differentials directly.

Rather, it is through inequalities in levels of living and the associated differences in life style, diet, occupational hazards, and knowledge, access and utilization of health care services that socioeconomic status materializes its effects (U.N. 1982). It has been warned, therefore, that socioeconomic status or classes can be surrogates for other cancer inducing factors and/or can interact with them to produce the observed differentials (U.N., 1981). It is necessary to keep this in mind as we review the findings on socioeconomic differentials.

3.5.1 The Theoretical Framework

Based on the success of mortality control through social and medical improvements, especially in relation to environment-sensitive infectious diseases, Petersen (1975) like many others, suggests that social class differentials are becoming smaller or even disappearing. On the contrary, not only have differentials remained relatively unchanged in the last 50 years or so, but evidence recently collected for France and the United Kingdom shows their further widening (Vallin 1980; U.N. 1980).

St[redacted] report that shrinking class differentials in Chicago back in the United States between 1930 and 1960, had reversed since the mid point of the period to a significant negative relationship. In his comprehensive literature review on social class and life expectancy, Antonovsky (1967) emphasizes that a downward unilinear relationship between class and health over time is an inaccurate image and an over-simplification.

When confronted with the predominance of chronic illnesses, as experienced presently by developed countries, preventive medical actions and health knowledge become increasingly essential in combating the diseases (Antonovsky 1967). Disadvantages of the lower class in these areas can actually reverse the shrinking class differentials caused by the control of infectious diseases. Therefore, the relationship between class and health over time appears curvilinear (Antonovsky 1967; 1980), and the recent increase in socioeconomic differentials is not unexplainable.

Antonovsky (1967) also contends that class differentials would be small in times when mortality rates are extremely high or low. However, when there is only moderate success in fighting death, class inequalities are inevitable, the reason being preventable deaths, which at any given level of knowledge, technique, and social organization tend to exaggerate class differentials. Although the proposed differential scheme is not aimed specificly at cancer mortality, it provides a theoretical

framework for the analysis of socioeconomic differentials in cancer mortality.

Firstly, the emphasis on health-related behaviors in chronic diseases apparantly parallels the importance of life styles and other individual actions hazardous to health in cancer. The issue of personal responsibility in the prevention of cancer is thus affirmed. Secondly, the notion that health related behaviors are class differentiated suggests that the same could takes place in cancer inducing life styles.

Inequalities in social and economic well being can precipitate disparities in level of living and, therefore, different styles of living and acquired habits that correlate with the high risk of cancer. For example, it has been shown that there is higher prevalence of cigarette smoking among lower social classes, whether grouped by education or income (Preston 1969; Siemiatycki 1974).

The less-than-modest progress in the prevention and treatment of cancer fits well into Antonovsky's description of the prerequisites for social differentials. Thus, the appearance of socioeconomic differentials in cancer is not without theoretical bases. Lastly, the emphasis on social actions as a means to remove differentials in chronic diseases (Antonovsky 1967) coincides with the advocation that social constraints limiting success in the fight against such diseases (Manton 1982). These concerns together provide a strong rationale for studying the impact of

socioeconomic status on cancer mortality.

Nevertheless, this theorectical framework for the study of cancer is not without problems. Antonovsky (1967, 1980) states that class differentials incline to disappear after age 65 but peak in the age group of 30-44, or at 25-34. Such an age trajectory casts doubt over the possibility of class differentials in cancer mortality in general or mortality from chronic diseases. In fact, not all studies report an inverse gradient between socioeconomic status and cancer mortality or attribute socioeconomic discrepancies as the sole contributor to the differentials.

3.5.2 Empirical Findings

Using data on males aged 25-64 in the United States, Preston (1969) questions the effects of social class on mortality differentials in the presence of cigarette smoking. Death ratios of smokers versus non-smokers in two education categories for fifteen causes of death were found clustered into two distinct groups. The results enable Preston (1969) to conclude an education effect among non-smoking related deaths, for example, cancer of the stomach, intestines and rectum. However, different smoking habits among various social strata are responsible for class differentials in cigarette related diseases like lung cancer.

The report by Graham and others (1960) on Buffalo, New York in 1948-1953 however, indicate opposite results. In

their study, the social areal approach was adopted. Census tracts were grouped into social classes based on median rental income and divided into quartiles approximately equal to population size. Incidence ratios of the low to the highest rental group, showed an inverse relationship in male lung cancer but not for females. To determine the effect of smoking, an age standardized percentage of smokers by sex and social status, computed from a 1956 random sample of adults, was analyzed.

Among males, an inverse gradient was noticeable between class and cigarette consumption. Yet, it was not as strong as between smoking and lung cancer. The authors suggested that the gradient differences reflect socioeconomic status as a causal factor of cancer differentials which was working independently with tobacco. For females, however, class status and smoking were found positively associated. Consequently, the negative socioeconomic effect might be counterbalanced and thus explain the absence of a negative relationship between class and female lung cancer.

It is necessary to point out, however, that these two studies are not strictly comparable. The most apparent difference is in the unit of analysis; death rates versus incidence rates, and individuals versus census tracts. Aside from the above, differences in socioeconomic indexes could have raised unequal response. Nevertheless, studies employing the same measures and approach do not warrant the same findings.

A study by Lerner and Stutz (1977) on the United States during 1959-1961 and 1969-1971 report a direct relationship between social class and cancer mortality. Average per capita income was used as the single indicator of socioeconomic status. Results show that, although cancer increased by 6.4 per cent in the low-income States in contrast to a slight decline among the affluent ones, death rates were actually much higher in the latter throughout the entire period.

Studies reviewed so far offered indications of an inverse relationship between socioeconomic status and cancer mortaltiy. Yet, the findings are far from conclusive. With income as the only social status indicator, the observed associations are diversified or just barely agree with the expected relationship. This could be due partly to the fact that income by itself is not a reliable socioeconomic status indicator (Kitagawa and Hauser 1973). Such a shortcoming can be compounded by the long latency period involved in cancer, which makes any negative income effect hard to sustain over time. Less time sensitive indexes are needed for the analysis of cancer differentials.

Many studies have chosen occupation as a surrogate of social class. William Farr pioneered researchs in this area by compiling death rates for different occupations in England and Wales beginning in 1851 (Kass 1977). Statistics of similar professions were then ranked into high and low social classes and their mortality experiences compared.

Logan (1954) analyzed the changes in class differentials in England and Wales between 1921 and 1950. His reports include five specific cancer sites, as well as female differentials based on their husbands' occupations. Mortality was found to improve with class status. With the exception of breast cancer, which was positively correlated with social class, the inverse relationship was generally more intense among males than females. Higher fertility among lower social class women was regarded as giving a protective effect against the threat of breast cancer.

A more detailed report on the inverse relationship between social class and cancer come from a recent summary by the United Nations (1982). Studies conducted in England and Wales, Scotland, Finland and the United States for various time periods in 1950-1973 are reviewed. The results pertaining to cancer are extracted and reproduced in Table 3.3.

From the given figures, a negative relationship between class and cancer mortality is readily observed. The literature review cautions that positive gradients observed for some individual cancer sites could be suppressed when they are aggregated. In the case of females, the overall inverse pattern could be diluted because of the positive relationship between certain cancer sites and social class.

One of the major criticisms of using occupation to differentiate social class is the possibility of a recursive relationship between the two. Although low occupation groups

Table 3.3 Occupation Class Differentials in Cancer Mortality, Selected Developed Countries, 1950-1972

Class	England and Wales (1970-72)	Scotland (1959-63)	Finland (1969-72)	United States (1950)
	<Standardized Mortality Ratio>			
I (High)	75	77	53	89
II	80	82	89	92
III	91	100	124	106
IV	126	99	153	119
V (Low)	197	143	82	129

Note: Figures for Finland pertained to lung cancer only.
Class III Manual in England and Wales is not listed here.
Class VI of agriculture in the U.S. is not listed here.

Source : Extracted from Level and Trends of Mortality Since 1950, United Nations, New York, 1982, Tables II.26, 27, 29, 30.

are linked with high death rates, it is also likely that poor health prior to death can lead to a downgrading in occupation. Consequently, it would not be easy to pinpoint the direction of causation.

A recent longitudinal analysis on England and Wales between 1976 and 1981 by Fox and associates (1985) nevertheless provides strong support for the negative effect of low status occupation on mortality. Although total mortality was the object of analysis, the findings are relevant to a socioeconomic framework of cancer mortality as well.

By relating their findings to those reported earlier for 1970-1972, Fox and colleagues (1985) notice that negative social class gradients based on occupation have persisted among those 75 years of age and above. The authors stress that such observations are not due to changes in social class because of ill health since the deceased had retired more than 10 years before death. In other words, the recursive forces from health to occupational based social class were not in effect.

It is emphasized instead, that the differentials are because of socioeconomic inequalities and life styles in the period before death. Fox and colleagues (1985) contend that even though poor health can precipitate downward occupational mobility, the former is actually led by health related behaviors. Their effect must persist into old ages. Consequently, the differentials reflect and reinforce health

behaviors in childhood or early adulthood. In relation to cancer mortality, the above arguments of prolonged health behavior effect from young to old ages parallel the impact of many cancer risk factors and thus provide a strong rationale for studying socioeconomic differentials in cancer mortality.

In their classical census-death record linkage study, Kitagawa and Hauser (1973) employed education as an indicator of socioeconomic status. Results provide evidence of its inverse relationship with cancer mortality. The authors report that in the United States in 1960, males between 25-64 with less than 8 years of schooling had a cancer death rate 30 per cent more than those with a college background. At ages of 65 and above, however, the pattern was reversed by as much as 11 per cent.

Mortality for less educated females in both age groups, were in excess of 23 and 12 per cent of those best educated, respectively. To account for the sex variation, the authors noted three specific cancer sites acting in opposite directions. Firstly, cancer of the prostate was positively related to education at all ages. Secondly, male lung cancer began to invert its negative relationship at the age of 65. Their suggestion that other factors might have led to this reverse seems to be confounded by Preston's conclusion of insignificant class effects in smoking related diseases (1969).

Lastly, female breast cancer also maintained a positive association with education. Death rate of the least educated younger women was about 22 per cent below that of the best educated. Less childbearing and breast feeding by women in high socioeconomic group are the prime suspects for such discrepencies. The findings reported here serve not merely to document the inverse gradient between social class and cancer mortality. In conjunction with earlier reports they illustrate two essential points when studying cancer differentials.

Perhaps the choice of socioeconomic index and the methodology could, indeed, affect the result. The hypothesized inverse relationship between social status and cancer is not stable. In fact, positive association has been reported. Extra attention is required when only one indicator of socioeconomic status indicator is used. Although any exceptions to the inverse gradient could be due to population characteristics, a possibility remains that a long latency period in cancer mortality allows the disease to respond unequally to various socioeconomic factors.

Not unrelated to the above is that individual cancer sites differ in their reactions to socioeconomic conditions. The chance of obtaining a spurious overall correlation rises if individual sites are not weighted with care. Similarily, class differentials observed in areal studies could be distorted easily if there are distinct variations in the geographic distribution of cancer sites.

The reliability of employing a single social indicator is a major concern, which has led to other approaches. Comstock and Tonascia (1977) examined education and mortality with data from Washington county in the United States in 1963. The cause-specific death rates were adjusted for seven socioeconomic and demographic variables including age, sex, race, years of schooling, and smoking habits through multiple binary regression. Annual mortality rates of 25 year-olds and above in 1963-1971 were traced and compared across education groups.

The study did not find a significant time trend between cancer risk and educational level, despite the fact that they were negatively distributed. Even so, this analysis indicates the need to take into consideration other variables in socioeconomic differential research. As the authors summarize, the adjustment of relevant social and demographic factors can increase the specificity of the relationship.

Ellis (1957) chose to analyze socioeconomic differentials in chronic diseases in Houston, Texas, in the years 1949-1951. The ecological approach was adopted, and education, occupation, and rental value were utilized to form a composite index. The census tracts were then grouped into five classes, with I to V representing the lowest to the highest social class. Comparison of the standardized death rates across the classes indicated an inverse gradient in male cancer mortality but with higher death rates in

class IV than V.

Regarding females, the negative gradient was maintained throughout all five social classes. For the particular male pattern, Ellis (1957) concludes that it might because of the greater eligibility of the most disadvantaged group to receive free medical services. For those in class IV, however, conditions were not "unfortunate" enough to allow them to benefit from most social and medical assistance. Yet, observations on a later time period are quite different.

Roberts and associates (1970) replicate the same ecological study for the period of 1959-1961. A similar composite index was developed, except that income was used in place of rental value. The index was then correlated with the cause specific death rates. The authors found that the negative correlations between degenerative diseases, including cancer, and social class were statistically insignificant, whether ethnicity (Blacks and Spanish) was controlled or not.

Significant relationships were found only in infectious diseases and social causes like suicide. The authors acknowledge that their findings could be due to their unconventional methodology, that is, using correlations rather than direct comparison of the death rates among the social ranks. Studies with the latter approach have yielded more expected results.

Nagi and Stockwell (1973), for example, construct a social rank score for each census tract in Hartford, Connecticut, in 1960 using income, education, and occupation. The ratio of observed to expected deaths revealed an inverse gradient between cancer and the areal social ranks and increased markedly as social rank dropped.

Yeracaris and Kim (1976) set up a social index for census tracts in three metropolitan areas in the United States in 1960 and 1970, respectively. The areal sociceconomic index was developed using per cent of professionals, family income, years of schooling, and monthly rent. As expected, there was an inverse relationship which had generally increased during the decade. More importantly, the analysis found that when overall mortality declines, socioeconomic differentials from heart disease and cancer increased.

Together with earlier reviews, it is obvious that socioeconomic status does exert its effects on mortality differentials directly and in conjunction with factors like age, sex, and ethnicity. In male lung cancer, for example, the inverse relationship with social status was higher among United States-born non-Jews than among the foreign-born. Female breast cancer, on the other hand, was higher among U.S. born Jews than among the foreign born, especially those with high social status (Seidman 1971).

3.5.3 Socioeconomic Differentials in Canadian Cancer Mortality

In his case-control study of two Canadian cities in Ontario in 1969-1973, Wigle (1977) ranked the census tracts therein into five income groups according to the median household income. The observed lung cancer deaths were then compared with expected mortality based on the average age-sex specific lung cancer death rate for Ontario. An inverse association between male lung cancer and level of income was observed.

National Health and Welfare of Canada (1980) conducted a similar analysis but covered 21 census metropolitan areas which represented 54% of the 1971 Canadian population. The census tracts were again ranked by median household income. Comparative ratios of age standardized death rates in low income groups to those of the highest income level were used to track any class differentials. The results appeared supportive of the earlier findings, on the one hand, yet rather inconsistent within themselves, on the other.

For all cancer deaths combined, a moderate negative gradient between death ratios and income level was observed among males. This association remained notable in the age group of 1-14 and 35-64 but was subdued otherwise. In the broad middle-age group, strong connections were apparent for cancer of the larynx, and the cervix and corpus of the uterus, but only moderate for stomach and lung cancer.

Surprisingly, there was little differential in female cancer deaths by income status. Two explanations are deemed plausible. It has been suggested already that absence of female cancer differentials could due to the strong positive relationship between breast cancer and social class (U.N. 1982). In other words, there is a good chance for the negative gradient to be diluted.

Recall the positive connection between class and smoking among females (Graham et al. 1960), a connection which could undermine the effect of socioeconomic factors on female lung cancer. These two factors together might have diminished any negative income effect on female cancer mortality.

However, the evidence does not seem to support such reasonings. While lung cancer was stated as having "...no role in differentials among females...," no relationship in either direction was reported for breast cancer (National Health and Welfare 1980:18). Also noteworthy in this study is the finding of the largest income differential in the young male age group. Such an observation is inconsistent with the notion that child cancer deaths are less subject to exogenous factors than are adults cancer deaths.

Other than income, information on socioeconomic differentials in Canadian cancer mortality is comparatively limited. In his report on the health status of Canadians, Wilkins (1980) noted similarities between the occupation-class mortality differentials in Quebec reported

by Billette and Hill (Wilkins 1980, c.f) and those revealed in Britain and the United States.

Wilkins then advocated that, in the absence of Canadian information, association between socioeconomic status and mortality for this country can be borrowed from similarly developed countries. His suggestion seems to receive some support from a recent study by Thouez (1984) on the metropolitan region of Montreal. A strong inverse relationship was found between cancer mortality and socioeconomic status, indexed by income, education and occupation.

In a simple analysis on distribution of causes of death, Siemiatycki (1974) observed higher cancer death rate in the poorer, southern region of Montreal in 1970. Massam and Nisen (1980) studied the spatial arrangements of causes of death and socioeconomic variables in the same region and time period. Spatial mapping indicated that only male heart disease and female breast cancer had some form of socioeconomic clustering within the region.

Household income, percentage of French speaking population, number of persons per room, and residenc occupancy were then used to correlate with the diseases. Yet, the variance being explained in each case was very low. It was therefore suggested that there was no spatial clustering of causes of death according to areal socioeconomic indicators. It would not be too surprising if the choice of indicators for socioeconomic condition had

affected the results. A few of them actually reflect either the social or the economic milieu of the census tracts.

Nevertheless, analysis by Thouex (1984) of the metropolitan region of Montreal in 1971 has revealed the expected relationship. Measurements of median family income, education, and occupation were used for a composite index. The results show a strong negative association between social status and standarized cancer death rates.

Obviously, the present review does not exhaust all the research on Canadian mortality differentials. Most Canadian studies on socioeconomic differentials in cancer mortality have focused on the Quebec-Montreal regions rather than on the remaining areas. Due to the concentration of French speaking population in these areas, it is possible that different patterns exist elsewhere.

Moreover, most researchers have chosen to analyse a single dimension of the complex social and economic milieu. These might reflect the interest of the analysts, or limitations of the data required. Yet, socioeconomic indicators besides income and occupation, by themselves and as composite scores, should be analyzed for more meaningful findings. Last, but not the least, few socio-demographic factors like ethnicity have been included in most studies on Canadian cancer mortality differentials.

3.6 Summary

Socioeconomic and medical developments have been identified as the fundamental forces behind the increased human life expectancy. Along with this progress, the same factors have altered the causes of death structure. Such changes, in turn, affect on future mortality decline to various degrees. The developed countries, with their epidemiologic transition completed, have their new mortality structure dominated by degenerative and chronic diseases.

The causes of death, primarily heart disease and cancer, are inelastic to the current level of curative medical technology. This is especially true in the case of cancer. While there is increasing success in delaying death from heart disease until more advanced ages, most cancers remain incurable.

Degenerative diseases in general and cancer in particular, therefore, tend to impose a limit upon technology's effectiveness in improving human life expectancy any further. Although the developing countries are still some steps away from such a stage, the same situation is foreseeable when they complete their epidemiologic transition. Findings on degenerative diseases have linked the causes more to human actions than to genetic factors.

Regarding cancer, detrimental life styles and other behavorial patterns, plus environmental exposures including those from work areas have been found causative. The fact

that such risk factors are socially rooted prompted many investigators to advocate that societal constraints actually operate on human life expectancy before the exhaustion of medical alternatives.

Individual and social readjustments, or modifications, in terms of these constraints are emphasized to prevent the onset of degenerative diseases. In view of the incurable nature of cancer, the need to identify and eliminate these societal constraints is crucial.

4. Data and Methodology

4.1 Introduction

In our review, cancer mortality trends in Canada up to 1974 have been briefly accounted for. Steady increases are found not only in overall cancer death rate, but also in many cancer sites. It is speculated that death rates will continue to rise in the near future. The review has reported on the various kinds of cancer inducing factors, ranging from biological elements to personal hazardous behaviours. There are controversies over socioeconomic differentials in low mortality countries, particularly over their prevalence in cancer mortality. Although the evidence may not be conclusive, it does indicate an unequal cancer risk among social classes. For Canada, our knowledge of this area comparatively fall short of the minimum we should know.

Canada is chosen as the country of analysis, and three objectives are developed: to reinterpret the Canadian cancer mortality trend, to estimate future cancer death rates, and to assess the socioeconomic-demographic influences on cancer mortality. Since new approaches will be taken, only total cancer and cancer of the lung, bladder, stomach and intestine in males and females separately, and breast cancer in females, are selected for the analysis. These cancer sites are chosen for their importance as being among the top ten cancer sites in Canada or for their known variations with socioeconomic conditions. All analyses are carried out

on Canada as a whole.

In the subsequent discussions, the data sources from which variables required for analysis are extracted or derived will be reported. The characteristics and limitations of the data or the variables will also be accounted for. The methods of analysis selected for each of the three objectives are to be followed. If applicable, working hypothese between the dependent and the explanatory variables are developed.

The discussions are divided into three sections, each pertaining to one of the targets set up for this study. For the convenience of description, general cancer mortality is being treated as an individual cancer site and is used interchangeably with total or overall cancer mortality.

4.2 Trend Analysis

Our trend analysis focuses on the decades between 1951 and 1981. Three types of cancer site-sex specific death rates are used for this study. Annual crude and age specific death rates for the entire 31 years and the standardized (age adjusted) death rates up to 1978 are extracted from the annual vital statistics reports prepared by Statistics Canada.

Prior to 1979, standardized death rates are based on the 1956 population age structure, but are replaced by the 1971 age structure since 1979. To make the data comparable, age adjusted rates after 1978 are recomputed here using

direct standardization (Shryock and Siegel 1976) with the 1956 age structure.

Instead of just describing the fluctuations in crude and standardized death rates during the designated period, efforts are made to decompose cancer mortality into meaningful components. Changes in cancer mortality will then be examined in terms of the movements of these components. In earlier discussions, we noted that cancer mortality can be affected by changes in the population age structure. A simple example would be the increase in cancer death rate as the proportion of elderlies in a population expands.

Cancer mortality changes when cancer inducing factors improve or deteriorate. A typical case would be the increases in females smoking cigarettes. By making use of the intertwined relationship between the crude and the standardized death rates, and with the 1956 crude cancer death rate as the base line, we can single out these two components. In terms of a mathematical equation, the first component of age effect can be defined as follows:

(4-1)

$$\text{Age Effect} = \text{Crude Cancer Death Rate} - \text{Standardized Cancer Death Rate (1956 Age Structure)}$$

On the right-hand side of equation (4-1), the age structure for the standardized death rate is fixed at 1956, and only factors other than age affecting cancer are allowed to vary with time. For the crude death rate, however, none of the factors is being kept constant. The only common varying element in these two death rates is the effect of cancerous factors. Once this element is removed, the difference is the death rate attributable to changes in the age structure.

Since the age structure in Canada is changing over time, positive differences from the years after 1956 indicate that the aging population has acted to increase cancer death rates. Negative differences show just the reverse. For the years before 1956, the interpretation is more complex, the reason being that age structures in 1951-1955 are likely to be younger than the base population. As a result, explanations for this period are the reverse of those for later years. To reduce confusion, it is to our advantage to keep this period as short as possible. This age effect component summarizes influences contributed by all age groups.

The other component, cancer force, collects influences exerted on cancer mortality by all cancer inducing factors. One cannot single out the biological factors from life styles or occupational hazards. Cancer force can be

expressed by the following equation:

(4-2)

Cancer Force = Standardized Cancer Death Rate (1956 Age Structure) − 1956 Cancer Death Rate

Unlike the age effect equation, we will now fix the age structure. On the right hand side of the equation, both the annual standardized death rate and the 1956 base rate have the same 1956 age structure. However, they differ from each other, because the effects of cancer inducing factors alter with time. The numerical difference between the two is then the death rate due to fluctuations in cancer force. A positive value indicates deterioration in cancer force, and a negative value stands for otherwise.

Unfortunately, there is a major shortcoming in the above decomposition. The 1956 age structure used to standardize the cancer death rates has been criticized for having a deflationary effect on the resulting rates (Field 1980). Detailed explanations for such an effect have been provided in Chapter 2.3. For our present approach, a smaller standardized death rate means that the age effect could be overestimated. As far as cancer force is concerned, the effect would be minimized since the same age structure is employed throughout its derivation (see Eqn. 4-2).

An age structure of later years (e.g. 1971) could have been adopted if not because of the difficulties in interpretation. With the base population age structure set at 1956, it will always be younger than those at later time points. The trends of the two components then easily reflect their progress over time. If a later population is selected, this advantage is partially lost.

Besides adding confusion, if the population age structures are equally split between young and old before and after the base year, then there will be greater instability in our component trends. For these reasons, the 1956 population age structure remains to be the standard. In order to compensate for the effect of the age structure, an age bias-free indicator of mortality is desired. Schoen (1973) suggests that geometric means of age specific death rates have such a property.

In addition, geometric means permit direct comparisons among themselves if equal numbers of age groups are involved. This feature proves to be helpful when contrasting male and female mortality experiences. For our selected cancer sites, except total cancer, the geometric means do not involve all of the usual nineteen 5-year age groups. Many young age groups have registered no cancer deaths, or they have extremely small death rates.

These age groups are excluded because they would either render the geometric mean zero or greatly lower the results. The age groups included for stomach cancer are from 30

onwards, 25 and above for intestinal and lung cancer, and 40 or older for bladder cancer. In short, the present trend analysis utilizes two decompositions of cancer mortality and three types of cancer death rates.

4.3 Cancer Mortality Rate Estimation

To estimate future cancer death rates, two equations are developed using the age effect and cancer force derived earlier. The age effects, whether positive or negative, are due to changes in the population age composition. To be specific, they can be explained by the sizes of the different age groups, which ultimately determine the supply of population at-risk.

Since cancer occurs primarily from young adulthood onwards, the age groups of 25-44, 45-64 and 65+ are selected to account for the age effects. We will hypothesize that the rates of their changes directly influence the age effect. The faster they grow, the higher will be the age effect, and vice versa.

In order to measure the speed of change, annual percentage of growth in the proportion of these three age groups is employed. Proportions of these three broad age groups are computed using annual population estimates published by Statistics Canada. Once the proportions are determined, the percentage of growth for each age group is

defined as follows:

(4-3)

$$\text{Percentage of Growth} = \left(\frac{A - B}{B} \right) \times 100$$

where,

A = percentage of annual growth of year h+1

B = percentage of annual growth of year h

Once the proportions of the three age groups are derived, stepwise regression (Gunst and Mason 1980) is carried on the equation below:

(4-4)

$$\text{Age Effect} = A\,(\text{P.G of } 25\text{-}45) + B\,(\text{P.G. of } 45\text{-}64) + C\,(\text{P.G. of } 65+) + \text{Error}$$

where P.G. stands for percentage of annual growth.

At this stage, all independent variables are entered into the equation linearly. Quadratic or even higher power

forms of the variables will be added to the equation if deemed necessary. Since there are no prior reasons to exclude collinearities among these variables, remedial methods must be prepared if this indeed occurs. For our purpose of estimating future cancer death rates, this is especially crucial. Collinearities can result in, to mention just two, unstable regression coefficients and enlarged coefficient variances, which render the estimates unreliable (Gunst and Mason 1980).

In view of these possible adverse effects of collinearities, the technique of ridge regression developed by Hoerl and Kennard (1962; 1970) is selected. It will be applied, if conditions require it. This technique is preferred over other alternatives for the advantage of allowing variable selection and collinearity reduction at the same time. Detailed explanations and derivations of this method are available in Gunst and Mason (1980), Draper and Smith (1981), Daniel and Wood (1971), and Chatterjee and Price (1977). Also, Wellington, Macdonald and Wolf (1979) and McDonald and Schwing (1973) have demonstrated its applicability in mortality studies.

Comparatively, the equation for cancer force is simpler. Cancer force is expressed as a function of time and assumes an autonomous growth. The independent variable, time, is measured in terms of the number of years lapsed after (or before) 1956. This assumption is suitable for cancer force since it does not claim its victims

immediately, but after a fairly long period of time. Therefore, the intensity of cancer force should grow as time progresses. The equation for cancer force is as follows:

(4-5)

$$\text{Cancer Force} = A + B\,(\text{Time}) + \text{Error}$$

After all the regression coefficients are determined, future age effect and cancer force can be predicted. For the latter, the estimation procedure is straightforward. The input data depend only on the period of prediction. For age effect, however, the percentage of growth of the three age groups as the input data must be derived from reliable population projections.

For our purpose, the Canadian population by age and sex composition, projected for 1984-2006 under low fertility assumption are used. The projections are prepared by Statistics Canada (1985). Equation (4-3) is repeated on the projected age groups to derive their annual percentage of growth. Age effect and cancer force will be estimated for the years from 1983 to 2003, inclusive.

Once the two components are estimated, equations (4-1) and (4-2) can be rearranged to obtain the following equations to predict crude and standardized cancer death

rates for the same period of time:

(4-6)

Crude Death Rate = Age Effect + Cancer Force + 1956 Base Rate

(4-7)

Standardized Cancer Death Rate = Cancer Force + 1956 Base Rate

It is necessary to note that the estimated standardized death rates are based on the 1956 population age structure. Furthermore, we should not expect regression procedures to be smooth. As in most regression analysis, potential outlying points could appear and demand special identifying indexes and treatments (Belsley, Kuh and Welsch 1980).

All diagnostic procedures taken during our model fitting will be explained along with the regression results in later chapters. The stepwise regression procedure utilized for the present model building is from SPSSx (1983), and the ridge regression (if needed) is from the TROLL Statistical Package (1982).

4.4 Socioeconomic-Demographic Analysis

In this analysis, efforts are made to evaluate the impacts of various socioeconomic and demographic factors on Canadian cancer mortality rates. The dependent variables, male and female death rates from total cancer, and from cancer of the lung, bladder, stomach, intestine, and female breast cancer, are extracted from the Mortality Atlas of Canada prepared jointly by Health and Welfare Canada and Statistics Canada (1980).

The death rates are sex specific, standardized with the 1971 Canadian population, and pertain to the Census Divisions (CD). Exceptions are two CDs in Ontario and four in the Northwest Territories. They have been combined into one unit in each province. The Northwest Territories and the Yukon are not included in the present study due to their distinct demographic, social, and economic structures.

With the exception of three provinces, the death rates are based on the annual average of cancer deaths registered between 1966 and 1976. For British Columbia, the average is from 1972 onwards, and for Saskatchewan and Newfoundland, it begins in 1974. Such irregularities arise because in these provinces, death records prior to the stated years are not geocoded by CD boundaries. And, it is not possible to reconcile them with the 1976 CD boundaries on which the atlases are based (Health and Welfare and Statistics Canada 1980).

The independent variables are selected for the four groups of factors known to be associated with cancer mortality. They are age, ethnicity, socioeconomic status, and occupation. Since the dependent variables are based on the 1976 CD boundary, our explanatory factors have to comply with this regulation as much as possible. Therefore, 1976 Census data are the primary data source. Information from the 1971 Census is used if the required data are not available from the 1976 Census. The variables used in the analysis are discussed as follows:

A. Age

For each Census Division, proportions of males and females aged 25-44, 45-64 and 65+ are calculated using age-sex composition from the 1976 Census. We intend to find out what effects the concentrations of these age groups would have on cancer mortality within a CD.

The results should not be interpreted as individual risk, but rather the feature of a CD that contains the age groups in question. This characteristic of ecological approach applies to all variables used in this study. As far as the relationships between age groups and cancer death rates are concerned, we can anticipate that:

(1) Positive effects for males from middle age onwards.

(2) The youngest age group should have the largest

impact on female cancer, especially breast cancer.

B. Ethnicity

In the 1976 Census, the ethnicities or racial origins of the Canadian population are not reported. To distinguish the various ethnic groups, data on mother tongue have to be used as the surrogates. From the available data, the number of people in each CD identifying their mother tongue either as English, French, Chinese or Japanese, or Scandinavian are selected.

Their proportions to the total population in each CD are then computed. Since data are not provided for the Chinese and Japanese speaking populations separately, the two are combined to form the Asians group. Consequently, the the English, the French, the Asian and the Scandinavian ethnic groups will be studied.

Due to the fact that mother tongue has been used to identify these groups, some unreliabilities can be expected. Among the Asian and Scandinavian groups, Canadian-born descendents might identify either English or French as their mother tongue, rather than the language of their actual ethnic group. If this occurs on a large scale, then the two majority (English and French) and minority groups (Asian and Scandinavian) would be over- and under-represented, respectively. For these ethnic variables, we hypothesize that:

(3) Ethnicity does not have fixed impacts on cancer mortality, but varies according to its interaction with other variables.

C. Socioeconomic Status

In most areal studies of socioeconomic differentials of mortality, the Census Tracts are classified into high and low or even into more refined social classes. Level of income, years of schooling, or occupation status are the common criteria. The death rates are then compared across the defined strata to find out the class-mortality relationship. However, this approach cannot partial out confounding and interaction effects from other factors (McMichael 1981). Considering these limitations, it was decided to assess directly the effects of socioeconomic standing on cancer death rates, while at the same time eliminating undesirable influences.

To accomplish our objective, the first ideal variable would be the average years of schooling of the population in each CD. Unfortunately, this information is not available from the 1976 Census. In its place, we must rely on the levels of schooling of the labour force in each CD obtainable from the same Census. The size of the labour force is provided for five levels of schooling. Instead of using all levels, only the two categories, high school completed and has vocational training dipolma and some

university or above education, are being considered. Proportion of the total labour force with these levels of schooling are calculated.

The decision to use only higher levels of schooling is partly due to our interest in its relationship with cancer mortality after the confounding effects are removed. To estimate the contributions of each level to cancer death rates is not our concern here. The more important reason is that if a relationship between cancer and higher education really exist, then the reverse can be observed for a lower education level.

If we include all high and low levels of schooling, redundancy is introduced into our analysis. Also, the possible high correlations between the various levels of schooling and the occupation variables to be selected could be problematic. Based on these reasons, other levels of schooling are not included.

There is a major limitation of the data. Persons with various levels of schooling but not in the labor force for different reasons are excluded. The elimination of those retired affect our education index. This is becuase the level of education tends to be stabilized by the age of 25. As a result, the educational characteristics of a CD may not be truly represented. Due to such a shortcoming, the level of education should be interpreted as an index of the quality of the active work force. We expect that:

(4) A negative relationship between proportion of labour force with high education level and cancer death rate.

For income, the relevant data are not available from the 1976 Census. Since our dependent variables encompass the period 1966 to 1976, average family income in Canadian currency is extracted from the 1971 Census. To use these data, we have to assume that changes in family income between 1971 and 1976 are neligible. The major obstacle in adopting the 1971 figures is the problem of changes in CD boundaries.

It is found that 121 of the total 259 CDs do not have a valid value for family income. This problem of missing value is most serious in Manitoba as all of its CD's redefined boundaries in 1976. In terms of our statistical analysis, such a large number of cases without valid family income would eventually cut down the sample size, as well as the sample variation. To solve this problem, it was decided to estimate the missing family income using all of the remaining independent variables designed for our cancer analysis. These include education, ethnicity, age, and the industrial variables.

Since family income can vary among provinces or regions, the Census Divisions are grouped into the Atlantic Provinces, Ontario, Quebec, the Prairie Provinces, and British Columbia. Missing family incomes within these groups are then estimated using a special missing value treatment

program from BMDP (1981). The method of multiple regression is used by this program, and a maximum of 50% missing cases is allowed for each group or the entire sample. With this criterion, estimates cannot be provided for Manitoba.

To go around such a restriction, the estimation procedure is first carried out for all the provinces except Manitoba. The resulting estimates are then entered into the data file and treated as original values. The missing income values in Manitoba are then estimated without the earlier provincial grouppings. Using these steps, all missing cases are savaged. A high multiple correlation between family income and the independent variables, around 0.85, assures reliability in our estimates. For income, we hypothesize that:

(5) A negative relationship between family income and cancer death rates.

D. Industry

In socioeconomic differential analysis, studies have reported an inverse association between occupation status and cancer mortality. Yet, some studies attribute excess cancer deaths among workers of various occupations to their hazardous working environments. From our standpoint, we would agree with the latter approach. The effect of occupation should be directly through exposures in the work areas more

than indirectly through life styles or living conditions. If treated as such, then the risk of cancer in certain occupation can be uncovered much better and more correctly after the confounding income or education effects are removed.

It is, therefore, worthwhile to examine the influences of proportions of workers in selected occupations on cancer death rates. A positive effect should be displayed for proportions of workers engaged in cancer inducing occupations. To test this hypothesis, the required data have to be retrieved, once again, from the 1971 decennial Census.

Even with the occupational data at hand, it is difficult to pick those individuals most likely to associate with high cancer risk. Also, not all CD's will have workers in the same occupations. This in return could result in highly skewed distributions, or in a large number of missing values for some occupations.

Hence, the attention was switched to the proportion of the labour force in selected industries. In making such changes, we concentrated on industries that have positive impacts on cancer mortality and left more detailed analysis on the occupations therein to other researchers.

For our purposes, the percentages of the labour force in each CD engaged in occupations under the industrial divisions of farming, fishing, forestry, mining, manufacturing (except the food industry), construction, education, health and welfare, personal service, finance and

trade were considered. Of all the industries, we anticipate that

(6) Manufacturing and mining have positive impacts on cancer mortality.

E. Fertility

This last group of variables are applied only to female total cancer and breast cancer mortality. Ideally, crude birth rate, general fertility rate, completed family size, or similar measurements should be used in order to test the effect of fertility on breast cancer. However, the closest fertility index available from the 1976 Census at the CD level is the average number of children under 24 and staying at home. Obviously, this index is limited in a variety of ways.

First, it does not reflect the actual average number of births. Missing from the figures are children who already left their homes. The actual number of children per family is therefore underestimated, and so will be our fertility index. Since young adults tend to move away, the present index is more likely to reflect the average number of children for young women who are still of their childbearing age. However, women that past childbearing ages have had the largest number of births, a fact which undoubtedly again reduces the reliability of our index as a fertility

measurement.

The chosen index can further underestimate fertility of a CD if there is a large number of families of only elderly couples with no children staying with them. Due to these shortcomings, the ratio of children under the age of 4 to women 25-44 years of age is computed. This ratio will be used as another measurement of fertility.

Ratios of older age groups are not used because the denominator tends to shrink as females pass through the high death rate ages of 25-44. This, in turn, could produce a high child-woman ratio and inflate the impact of fertility on breast cancer. The two indexes discussed above are hypothesized that:

(7) They have positive influences on female breast cancer mortality.

4.5 Method of Analysis

The multiple regression technique is used for the statistical analysis of the data. Since these variables might affect cancer mortality individually and interactively with each other, the number of independent variables will eventually exceed the above. With such a large number of explanatory variables, it is necessary to locate the most parsimonious combination that gives gives the lowest error of estimate and the largest R^2.

To choose such a combination, the method of best possible subset regression is adopted with Mallow's Cp

statistics as the guildeline (Gunst and Mason 1980; Draper and Smith 1981; Chatterjee and Price 1977). The Cp statistics can be used to compare with the number of parameters (P), in a specific subset equation.

If Cp is greater than P, then the concerned equation has substantial bias. If the amount of bias is negligible, then Cp should close to P. When Cp is less than P, then the figure reflects the amount of variation in that specific equation (Daniel and Wood 1971). More detailed explanation of the derivation and usages of Mallow's Cp statistics are available in the above-cited references. The selection of our best possible equation is done by the subset regression program in the statistics package of BMDP (1981).

5. Canadian Cancer Mortality Trend: 1951-1981

5.1 Introduction

The present analysis looks at the trend in cancer mortality from 1951 to 1981. Besides total cancer mortality, specific sites selected for investigation include the lung, breast, stomach, bladder, and intestines. With the exception of breast cancer, all analyses are carried out for the two sexes separately.

Three types of death rates are utilized to explain the trend-crude and standardized death rates based on the 1956 age structure and the geometric mean of age specific death rates. In addition, the two components of cancer mortality derived from the crude and standardized death rates, namely, age effect and cancer force, are employed as well to account for the observed trend movements.

5.2 Total Cancer Mortality

From Table 5.1, we see that proportions of deaths due to cancer, for both sexes combined, rose from 14 per cent in 1951 to 24 per cent in just three decades. This change is clearly alarming. Equally shocking are the increases noted for males and females, from 13 to 23 per cent, and 16 to 24 per cent, respectively. Intercensal increase was highest between 1976 and 1981, showing 2.9 per cent for men, 2.5 per cent for women, and 2.7 per cent for both sexes jointly.

Table 5.1 Proportion of Cancer Deaths by Sex, Canada, 1951-1981

	Year	Male	Female	Both
Proportion (%)	1951	13.0	15.7	14.2
	1956	14.9	17.1	15.8
	1961	16.0	17.9	16.8
	1966	17.1	19.1	17.9
	1971	19.0	20.8	19.7
	1976	20.2	21.8	20.9
	1981	23.1	24.3	23.6
Intercensal Changes	1951-56	1.9	1.4	1.6
	1956-61	1.1	0.8	1.0
	1961-66	1.1	1.2	0.9
	1966-71	2.1	1.7	1.8
	1971-76	1.2	1.0	1.2
	1976-81	2.9	2.5	2.7

Source: 1951-1971 proportion figures from Vital Satistics, General Mortality, 1950-1972, Table 15, Statistics Canada, Ottawa, 1976.
1976-1981 figures derived from 1976 and 1981 Vital Statistics, Vol. III., Statistics Canada, Ottawa, 1978 and 1983.
Intercensal changes are from author's computation

Comparison of males and females reveals higher proportions, with declining differences over time, of cancer deaths among the latter. The differential impact is clearly demonstrated by higher female life expectancy gains after cancer as a cause of death was hypothetically eliminated (see Table 2.5). However, such a differential has been narrowed when male cancer mortality increases.

To explain the shrinking proportion in differentials, it would be far too simple to attribute it merely either to improvements among females or to their absence among males. Intercensal changes shown in Table 5.1 registered significant increase in proportion of cancer deaths for both sexes.

Nevertheless, it is necessary to point out, that using proportion to account for mortality trends or sex differentials demands extra precautions. A higher (or lower) proportion of cancer deaths might well be a result of decline (or increase) in fatalities from other causes, despite actual stability of the disease. Declines in cardiovascular-renal diseases, the leading cause of death, given in Table 5.2 show such a feasibility.

Due to the competition among causes of death for victims, the reductions could also mean more people are exposed to the risk of cancer. Consequently, there were real deteriorations in Canadian cancer mortality as reflected in positive intercensal changes in proportion of cancer deaths. While these interpretation remain possibilities, one must

Table 5.2 Proportion of Deaths Due to Cardiovascular Diseases by Sex, Canada, 1950-1981

Year	Male	Female	Both
	Proportion (%)		
1951	46.8	47.8	47.2
1956	48.7	49.8	49.2
1961	50.0	51.6	50.6
1966	50.0	52.2	50.9
1971	48.6	51.6	49.9
1976	47.8	51.2	48.7
1981	43.9	47.6	46.9
	Intercensal Changes		
1951-56	1.9	2.0	2.0
1956-61	1.3	1.8	1.4
1961-66	0.0	0.6	0.3
1966-71	-1.4	-1.4	-1.0
1971-76	-0.8	-0.4	-1.2
1976-81	-3.9	-3.6	-1.8

Source: Proportion figures of 1951-1971 from Vital Statistics, General Mortality, 1950-1972, Table 15, Statistics Canada, 1976.
1976-1981 figures from the 1976 and 1981 Vital Statistics, Vol. III, Statistics Canada, Ottawa, 1978 and 1983.
Figures of intercensal changes are from author's computation.

take note that figures in Table 5.2 are again subject to the same aforementioned limitations.

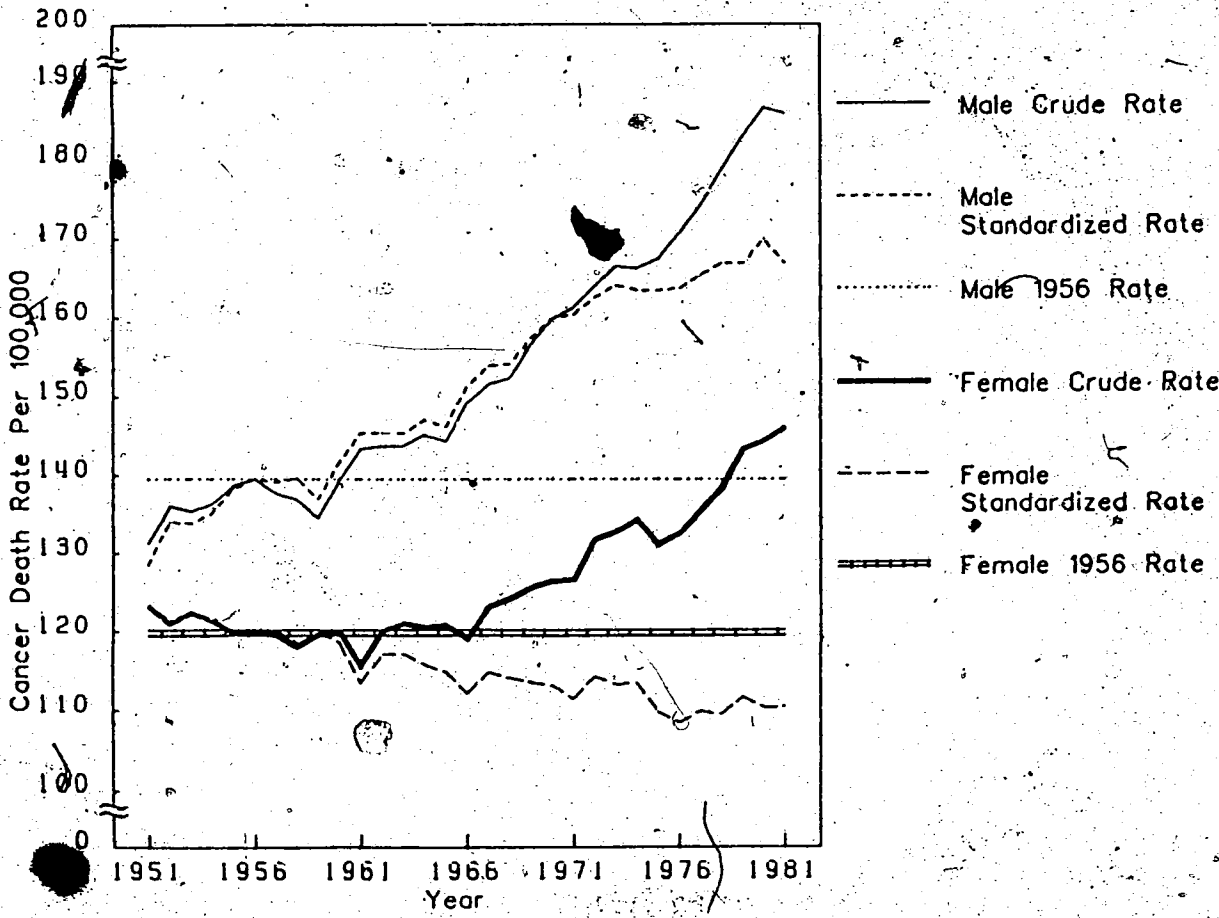
A more reliable and frequently adopted alternative for trend analysis is to utilize death rates. Male and female crude and standardized cancer death rates, as well as their 1956 base rates, are plotted in Fig. 5.1. From the diagram, the all too familar notion of higher male mortality is readily visible.

With the given information, it is probable that deaths from other causes affect cancer unequally among males and females. Even though more men died of cancer, the percentages were smaller because of greater number of deaths due to other causes. For females, it was just the opposite. Given these conditions, the impact of cancer on females could be more serious than was indicated by its relatively lower death rate, and should not be overlooked.

Aside from the above, there are other distinct differences between males and females with respect to their mortality trend. With regard to crude death rates, the two sexes had shared, for the most part, a common upward sloping trend. Major exceptions were in the years before 1966 when females experienced a much slower pace of increase. In fact, ignoring annual fluctuations, female pattern within this period was rather stable.

Since then, however, and particularly from the mid-1970's onward, female death rates were ascending at such a rapid speed that the curve greatly steepened, making it

Fig. 5.1 Crude and Standardized General Cancer Death Rates by Sex, Canada, 1951-1981



Source: Trend analysis.

almost identical to that of the males. In sum, the higher proportion of cancer deaths in the last decade or so is due to actual deterioration rather than to decline in deaths from other causes.

Taking standardized death rates into consideration, male and female cancer mortaltiy trend depart from each other to a greater extent. For males, careful examination found three distinct segments formed when their crude and adjusted rates cris-crossed each other. Between 1951 and 1956, crude death rates were higher than the age adjusted rates. A cohort effect that shortened the supply of elderlies as a risk population can be used to explain tnis (Statistics Canada 1976). Yet, an alternate explanation, derived from the deflationary effect of the 1956 base population, warrants our attention.

Since 1956 was at the height of the baby boom period, the population age structure at that time was younger than before. When age adjustments were carried out, standardized death rates now based on a population with large young age groups had to be lower. The age effects were then positive and reduced in size as age structures became closer to the base population. Such a pattern is clearly visible in the diagram.

If we focus on the cancer force during the same intercensal period 1951-56, we realize that it was negative, yet upward-sloping. This indicates that, although impacts on cancer from factors other than aging were less detertimental

in 1951-1955 than in 1956, they were progressing from low to high. Such an observation seem realistic as we expect the risk of cancer from environmental deterioration or hazardous behavior to increases over time. In the following period, continued growth in cancer force in Canada appeared in other forms.

Between 1957 and 1970, standardized death rates were higher than the corresponding crude rates. The age effects are, therefore, negative. Two main factors account for this phenomenon. First, large immigration could have produced lower crude death rates. It had been reported that absolute numbers of immigrants in the decades of 1951-1971 were close to the record volume achieved between 1901 and 1921 (Kalbach and McVey 1979:46). In addition, the majority of these immigrants was in the 20-35 age group which is only at the periphery of the high cancer risk age group. Undoubtely, the increase in the number of cancer deaths could not have been able to compensate for the huge population expansion.

Second, unexpected high standardized death rates are likely the results of increasing force of cancer mortality. Although the 1956 base population tended to deflate adjusted rates, non-age related factors might have deteriorated further to keep standardized rates from falling and to exceed crude death rates. Increase in cancer force was visible from the widening gap between the 1956 base rate and annual age adjusted rates.

Beginning in 1970, the male crude cancer mortality rates once again dominated their corresponding standardized rates. Contrary to the shrinking differences that prevailed between 1951 and 1956, the current age effects were increasing on a greater scale. When compared to negative age effect of the immediate past period, the changes were even more striking.

Combined with the moderate positive trend of standardized rates, we can infer that population aging had contributed significantly to high cancer death rates in the past decade. Even so, we should not neglect the impacts that arise from other factors. In fact, cancer force was also on the rise despite a short period of stabilization between 1971 and 1975.

All evidence suggest that males within the last decade w[illegible] under the adverse effects of both aging and cancerous fa[illegible]ors. Although we could not discount the experience of a similar situation in previous years, it had never manifested itself more clearly.

Turning our attention to female cancer mortality trend, we can easily observe that there are two distinct segments. Prior to 1956, crude and standardized death rates were both declining and were virtually of equal size. Such closeness could be due to the absence of significant changes in the female age structure within this period. To put it differently, the deflationary effect of the male 1956 age structure was not evident among females.

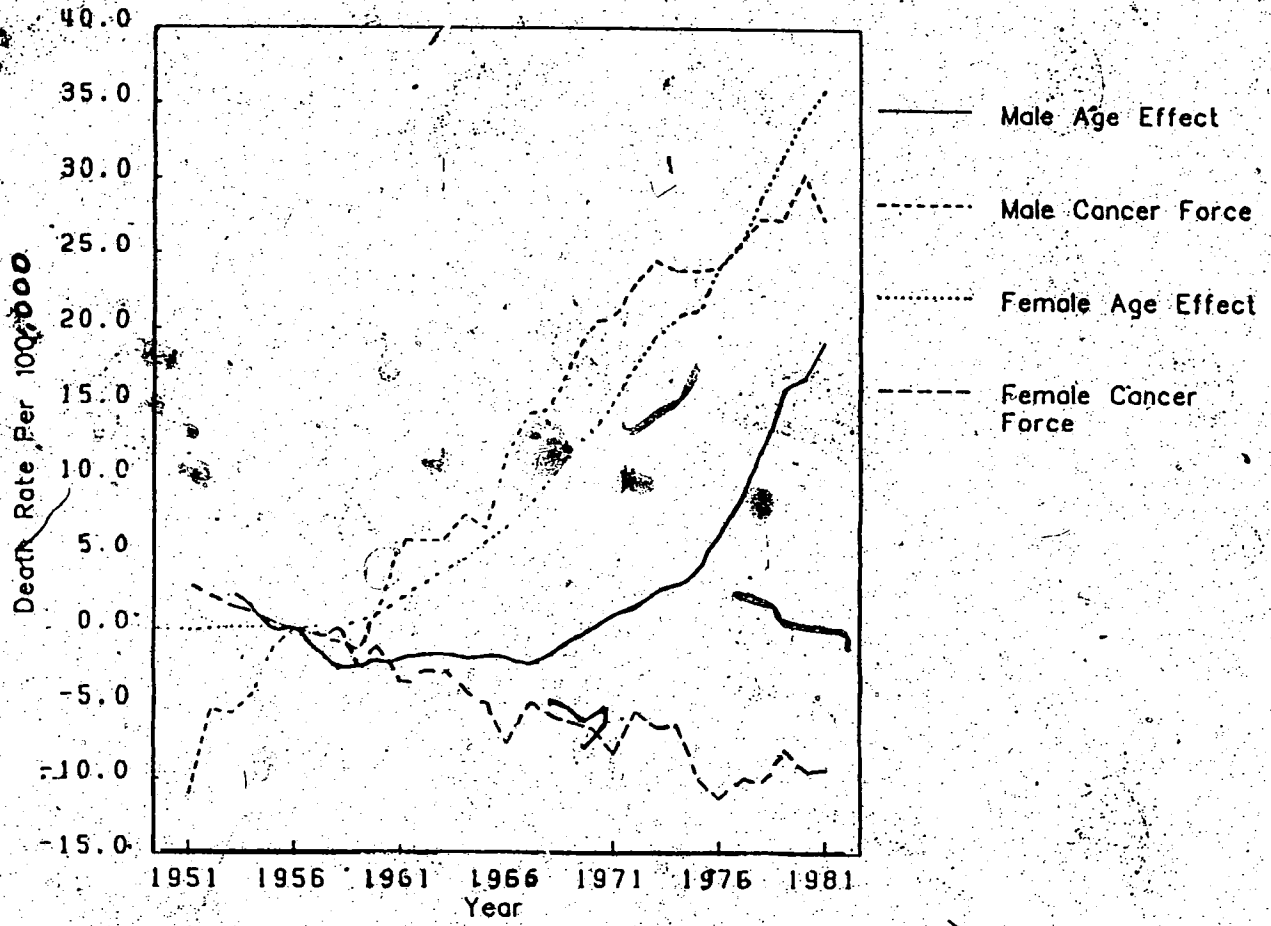
Another possible explanation is that strong cancer forces have prevented the young base population from lowering the age adjusted rates. From the diagram, we see that the effects are moderate, but the gap between standardized rates and the 1956 base rate is narrowing.

Starting from 1957, the age adjusted cancer mortality rate begins to fall sharply below its 1956 base rate, showing a reduction in cancer force. Only from 1976 onwards does it revitalize its deteriorating effect and settle down at relatively higher levels. In the mean time, crude death rates continue to register significant increases. As a result, there are increases in age effects.

These patterns of change signify that, while female cancer mortality was heavily influenced by population aging, there were minor improvements regarding other cancerous factors. Such opposing movement in crude and standardized death rates is the major factor that differentiates male from female cancer mortality in the past three decades.

An additional distinguishing feature of the female cancer trend is that the two types of death rates reveal a minimum for the census years of 1961, 1966, 1971, and 1976. Cohort effects, as well as lower age specific death rates are possible explanations. Male and female cancer mortality patterns reviewed up to this stage indicate drastic differences in age effect and the cancer force. Fig. 5.2 illustrates these differentials.

Fig. 5.2 Age Effect and Cancer Force of General Cancer Death Rates by Sex, Canada, 1951-1981



Source: Trend analysis.

From the diagram, it is evident that, while the female cancer force was on the decline, that of the males was on the increase throughout untill it was exceeded by the female age effect in 1976. These movements clearly reflect the absence and the presence of modifications in male and female cancer risk factors, respectively. The upturn in female cancer force since 1976 could be due to increased numbers of women engaging in health hazardous behaviours like cigarette smoking and entering traditionally male-dominated occupations that have high cancer risk.

Recall that female crude and standardized death rates arrive at a minimum in the census years between 1961 and 1976 in Fig. 5.1. A close pattern is also observed for female cancer force but not for age effect. This suggests that there are possibly cohort effects among the female cancer force. The factors that constitute these specific movements remain to be identified.

With respect to age effects, males display a U-shaped pattern, which is lower than that of the females and even more so than their own cancer force. From the distinct positions on these curves, some interesting obervations can be noted. By randomly fixing a horizontal line, at 10 per 100,000 population, we can easily realize that this was the amount of female age effect increase over the crude death rates in 1968.

Following this line, however, we observe that the same increment had been achieved by male cancer force in 1966 but

by [illegible] male age effect in 1977 only. This timing stayed relatively constant for most levels of increment above 5.0 per 100,000 population and halted when female age effect topped the remaining curves and distorted the relationship.

To inquire into why the male age effect lags behind female's by almost a decade for an equal amount of impact, a number of factors can be speculated. First, as females enjoy longer life expectancy, more of them will be exposed to the risk of cancer. This, in return, gives more intensive age effects. Second, high male cancer force could have minimized or delayed age effects by claiming most of its victims at younger ages. Third, the fact that male cancer mortality occurs largely after middle ages while that of females takes place mostly in child-bearing ages (20-45) could have resulted in such a time lag in the age effect. Fourth, differential timing could be due to disimilarities in rates of aging.

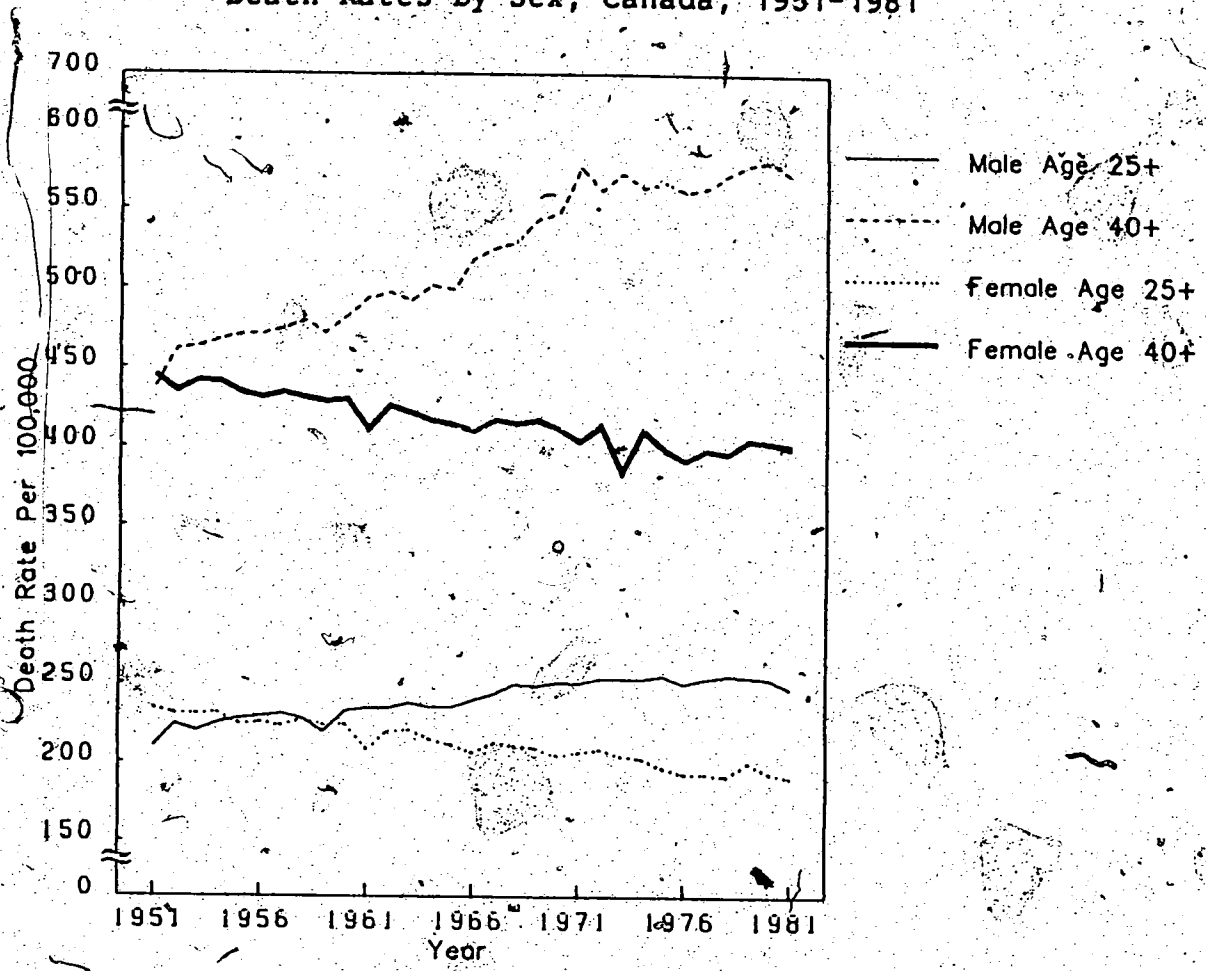
Between 1949 and 1981, annual rate of increase of population 24-44, 45-64, and 65 years of age or above were higher among females than males. Older age structure of female population combined with greater aging speed results in high age effects. Last, variations in the latency periods of male and female cancer sites could be responsible for observed timing differentials. That is, male cancer sites might involve comparatively longer latency periods and as a result, require a longer time to reach the same effect.

Previously, male and female cancer trends were discussed in terms of their age effects and cancer force. A few questions must answered such as what would be the actual sex differentials and the patterns of cancer mortality if age structure biases were discounted. Fig. 5.3 provids geometric means for males and females of 25 and 40 years of age and above. The diagram confirms clearly the earlier discussions of increasing male cancer mortality.

When males 25-39 are excluded, mean death rates rise by more than double. In addition, the gradient of increase is also much steeper. The differences reveal the deflationary effect on summary measures of mortality, if low-risk age groups are included. As far as females are concerned, their mean death rates are much lower than that of the males and are on the decline. Similar to males, however, the exclusion of the younger age groups still boosts the mean death rates.

These statistics show that the levels of Canadian cancer mortality as portrayed by usual crude or standardized death rates are rather deceptive. Actual mortality for high-risk age groups as represented by geometric means is well above that indicated by other death rates. Not evident in Fig. 5.3, however, are the minimums of female cancer mortality rates in 1961 and 1973. It seems that such phenomena are more likely artifacts of mortality rate calculations. On the contrar, nevertheless, influx of large immigration at these two time points could be the underlying cause.

Fig. 5.3 Geometric Means of Age Specific General Cancer Death Rates by Sex, Canada, 1951-1981



Source: Trend analysis.

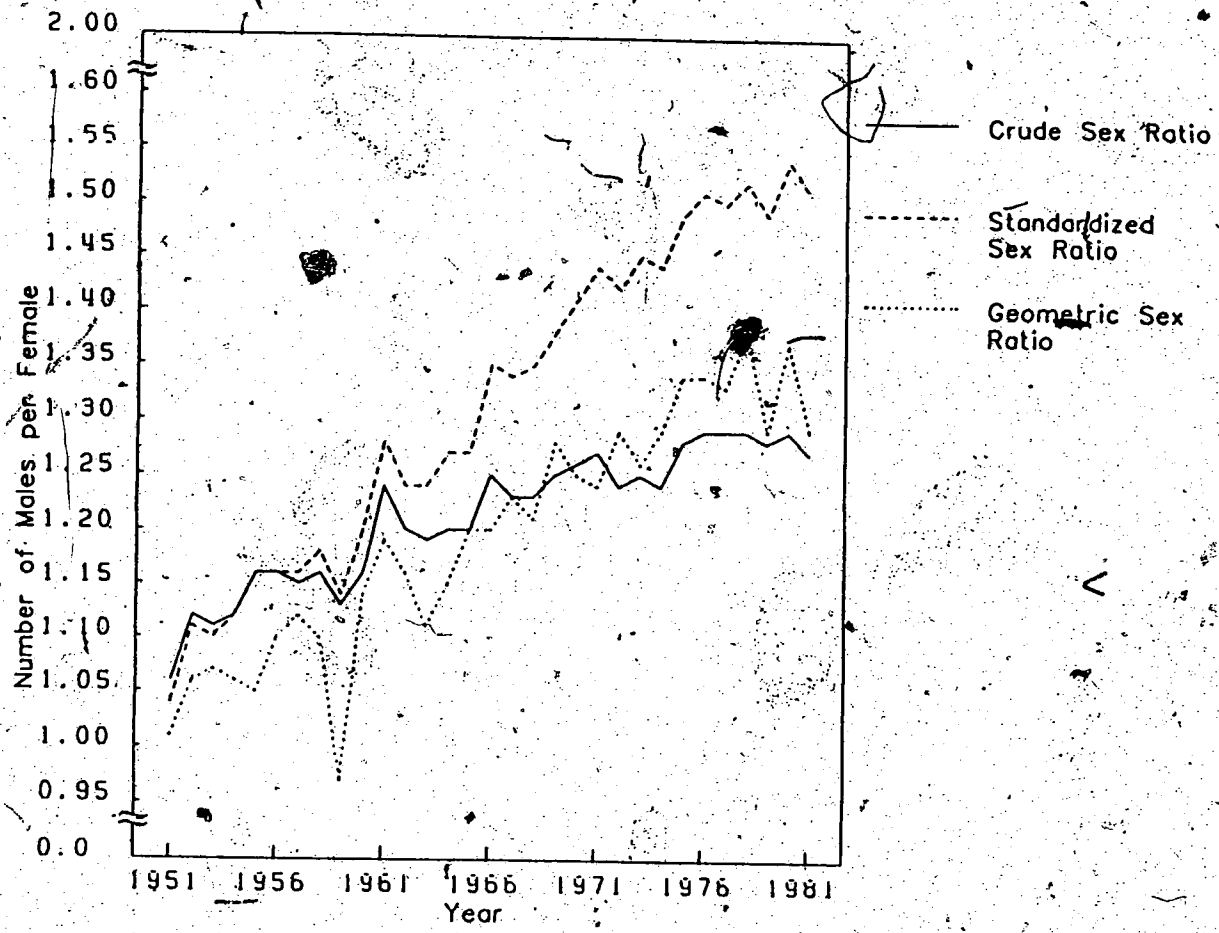
Regarding sex differentials, the three types of sex mortality ratios show in Fig. 5.4 all increases over time. In other words, excess male cancer deaths continue to increase. The level of sex differentials is generally lowest when the geometric means are used, the exception being from 1971 onwards. Since both crude and standardized rates suffer from the same shortcomings, that is, male and female population age structures are not strictly comparable, sex differentials from the geometric means are more representative. The level is increasing over time. We can conclude then that such an increasing trend in sex differential is due to continued deterioration of male cancer mortality, together with some improvements in female cancer mortality.

5.3 Lung Cancer

Lung cancer is the major cancer site among males, and is an increasingly prominent threat to females. Between 1951 and 1981, deaths from lung and trachea cancers grew from 12 to 32 per cent for males, and from 3 to 12 per cent for females. In terms of intercensal change, females experienced a 300 per cent increase. Yet, as shown in Fig. 5.5, female mortality rates throughout the entire period were still about four to five times lower than those of the males.

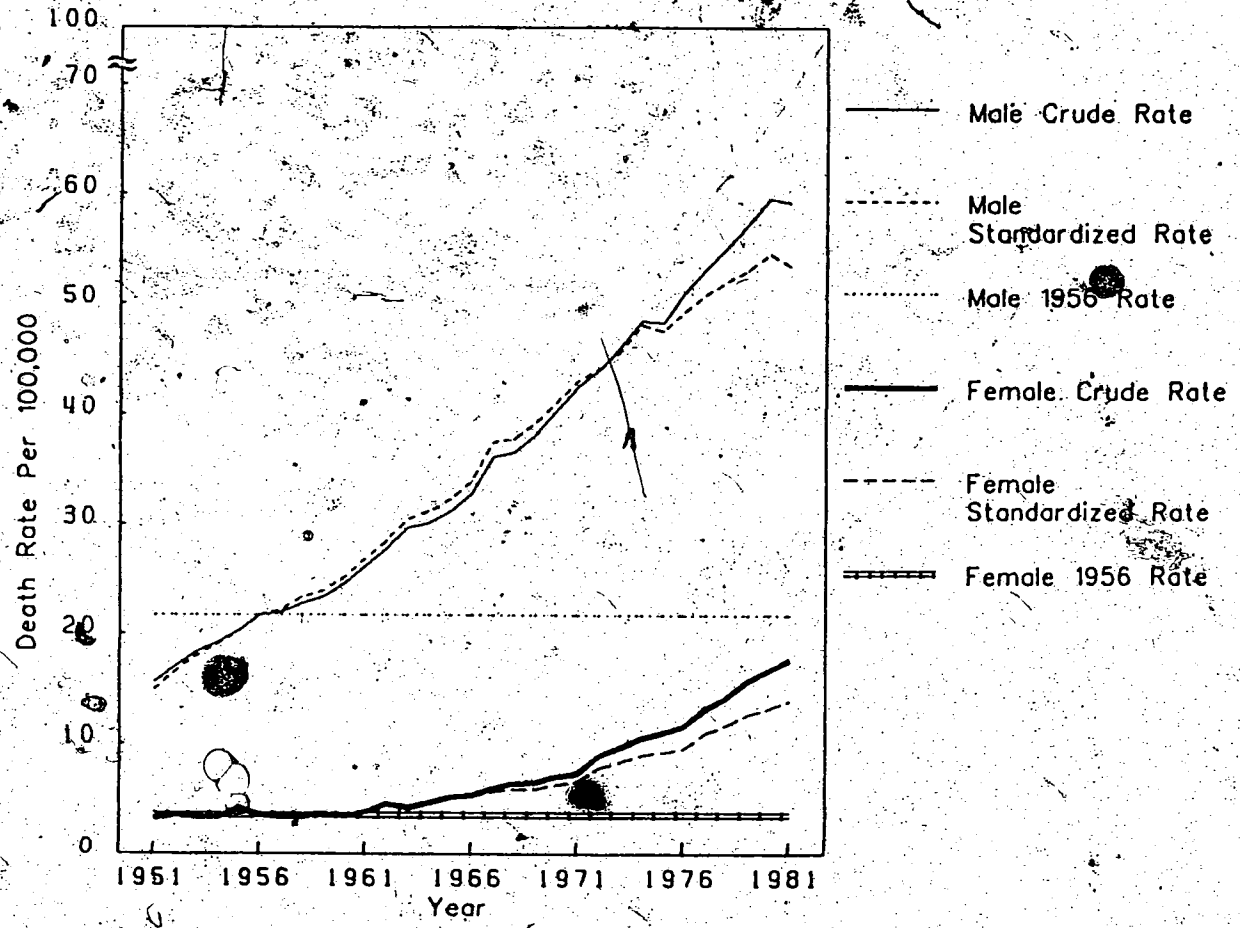
Geometric means in Fig. 5.6 show a smaller increase and do not become substantially higher even when low-risk age groups are excluded. This is quite different from the case

Fig. 5.4 Sex Differentials in General Cancer Mortality, Canada, 1951-1981



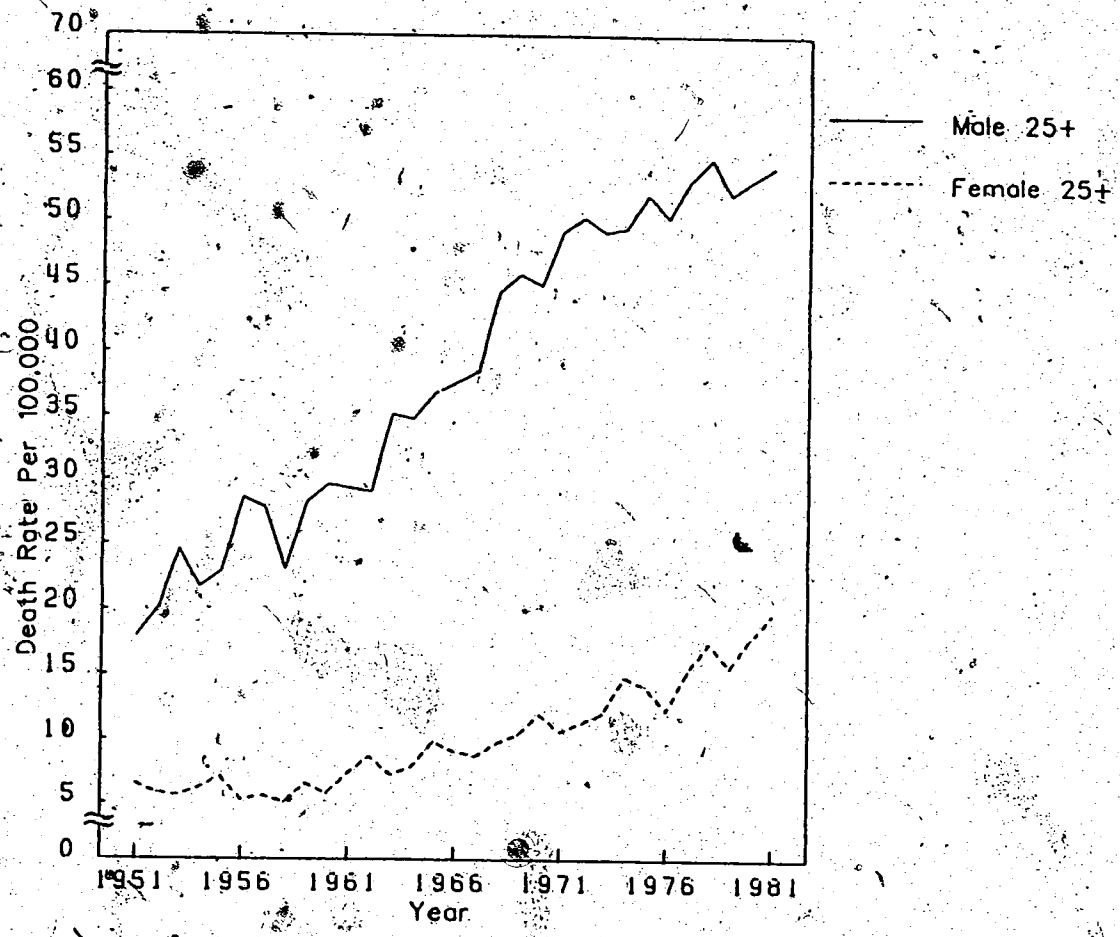
Source: Trend analysis.

Fig. 5.5 Crude and Standardized Lung Cancer Death Rates by Sex, Canada, 1951-1981



Source: Trend analysis.

Fig. 5.6 Geometric Means of Age Specific Lung Cancer Death Rates by Sex, Canada, 1951-1981



Source: Trend analysis.

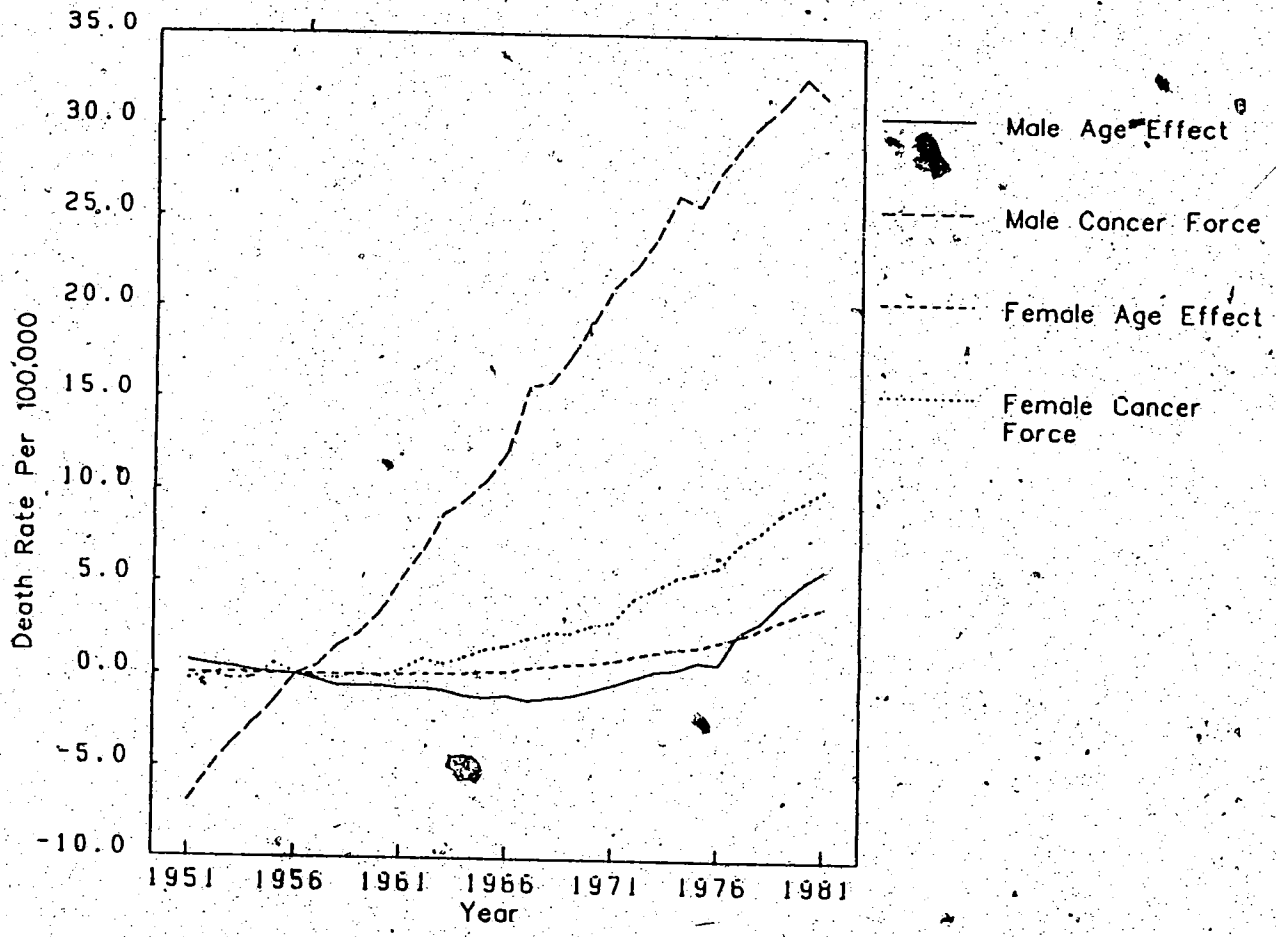
of total cancer mortality, where geometric means are much higher than crude or standardized death rates. High lung cancer mortality has indeed compensated for the deflationary effect of individuals younger than 25 years of age.

The patterns of male crude and standardized lung cancer death rates in Fig. 5.5 show great resemblance to those for total cancer mortality. From 1956 to 1973, male age adjusted rates are above the corresponding crude rates, denoting a period of high cancer force. Thereafter, the situation is reversed. This indicates that aging of the male population in the last decade has played an important role in increasing lung cancer death rates. Such a recent upswing in male age effect is clearly visible in Fig. 5.7.

For females, crude and standardized death rates, as well as the 1956 base rate, stayed fairly close to each other until 1960. Age structure and cancerous factors showed only marginal impacts on lung cancer mortality. In the following two decades, rising age effects and cancer force were present. Fig. 5.7 depicts a rapid increase in male cancer force throughout the entire period.

In contrast, the female cancer force gradient increased more gently. Large increases are registered from 1971 onwards and particularly after 1976. Lung cancer has played a dominant role in shaping these patterns. For both sexes, whether the risk factors involved in lung cancer are environmental or behavioral, the conditions obviously do not seem to be optimistic.

Fig. 5.7 Age Effect and Cancer Force of Lung Cancer Death Rates by Sex, Canada, 1951-1981



Source: Trend analysis.

As far as age effects are concerned, their impacts upon lung cancer are rather small. Similar to total cancer mortality, females in general had higher age effects than males but this has reversed since the mid 1970's. Some portions of fatalities due to carcinogenic exposures in the 1950's could have been delayed because of long latency periods, leading to a cohort effect.

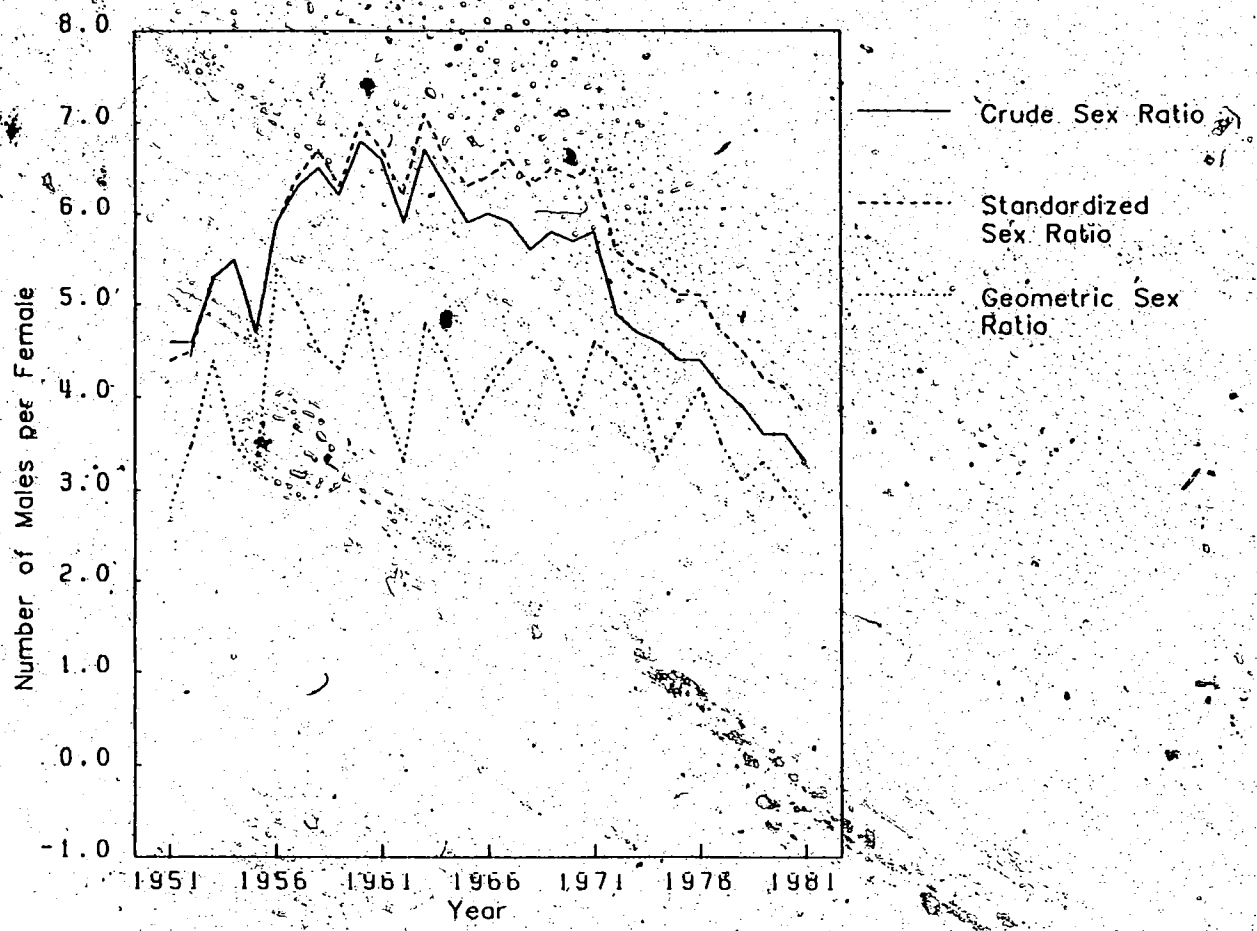
Turning to sex mortality differentials, patterns in Fig. 5.8 indicate a small excess in male lung cancer deaths in the last decade. From age effects and cancer force, we can infer that the narrowing gap was due to an increase in female mortality rather than to improvements among males. Of the three sex ratios, geometric means of populations 25 years of age and above yielded the smallest values. In other words, high excess male mortality was partly because of the inclusion of low risk age groups.

Sex mortality ratios computed from crude or standardized death rates paint a rather rosy picture for female lung cancer mortality. Geometric means offered a sex ratio of about 2.5 males for each female lung cancer death in 1981. It is not hard for this ratio to drop further if female cancer force continues to rise in upcoming years.

5.4 Cancer of the Stomach

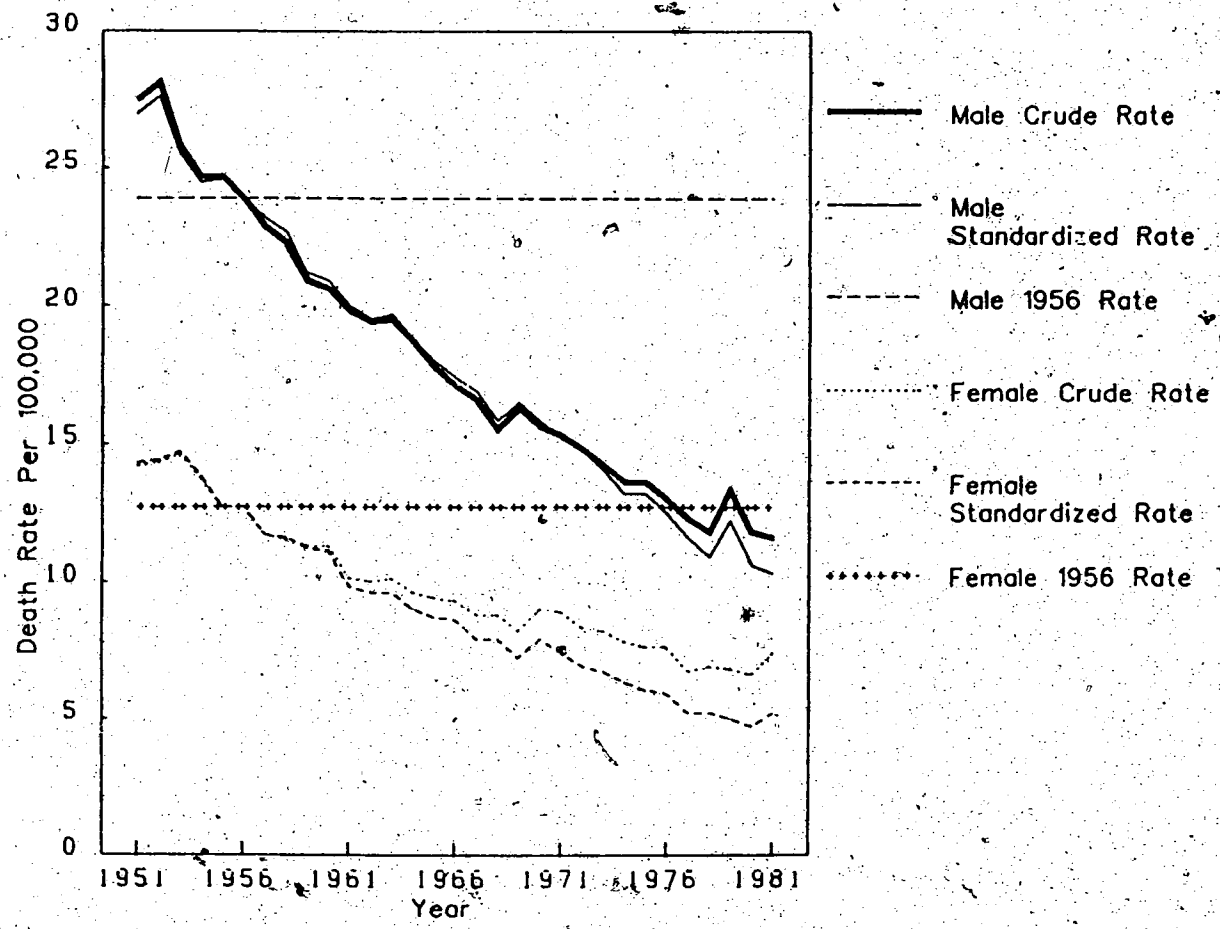
Compared to cancer of lung and trachea, mortality from stomach cancer has shown more encouraging improvements over time. Fig. 5.9 shows declining crude and standardized death

Fig. 5.8 Sex Differentials in Lung Cancer Mortality, Canada, 1951-1981.



Source: Trend analysis.

Fig. 5.9 Crude and Standardized Stomach Cancer Death Rates by Sex, Canada, 1951-1981



Source: Trend analysis.

rates for males, as well as for females. As before, male mortality was much higher, and the death rates stayed closer to each other. Geometric means of rates for age groups 30 years of age and above shown in Fig. 5.10 demonstrate even higher mortality. Yet, they are falling over time.

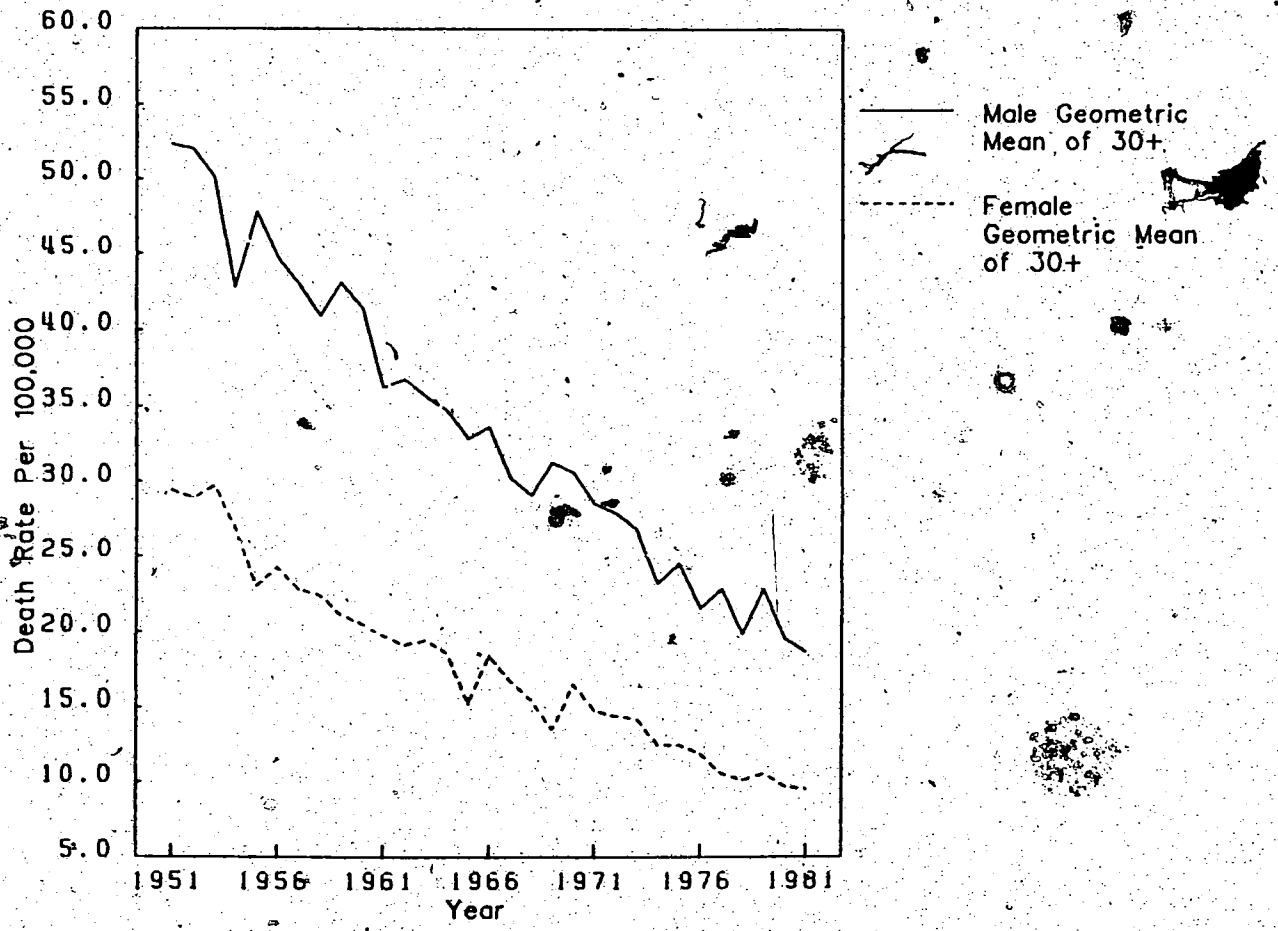
Whatever the risk factors for stomach cancer might be, their impacts have been reduced significantly during the past three decades. Nevertheless, progress was less impressive among females. As we can see in Fig. 5.11, female cancer force was substantially higher. It is likely that males and females differ in their risk factors or in their actions to reduce risk. Consequently, the degrees of recovery vary.

Age effects are on the rise over time and are higher among females. Evidence suggests that the aging population has prevented mortality from dropping to a more desirable level. The slightly upward-sloping trend implies that the age effect may possibly gain further importance in upcoming years.

The three types of sex ratios reveal for the first time dissimilar patterns of change. In Fig. 5.12, we see that the sex mortality ratios from crude death rates were falling over time. However, ascending trends beginning in the early 1970's are indicated by ratios from the standardized rates and geometric means.

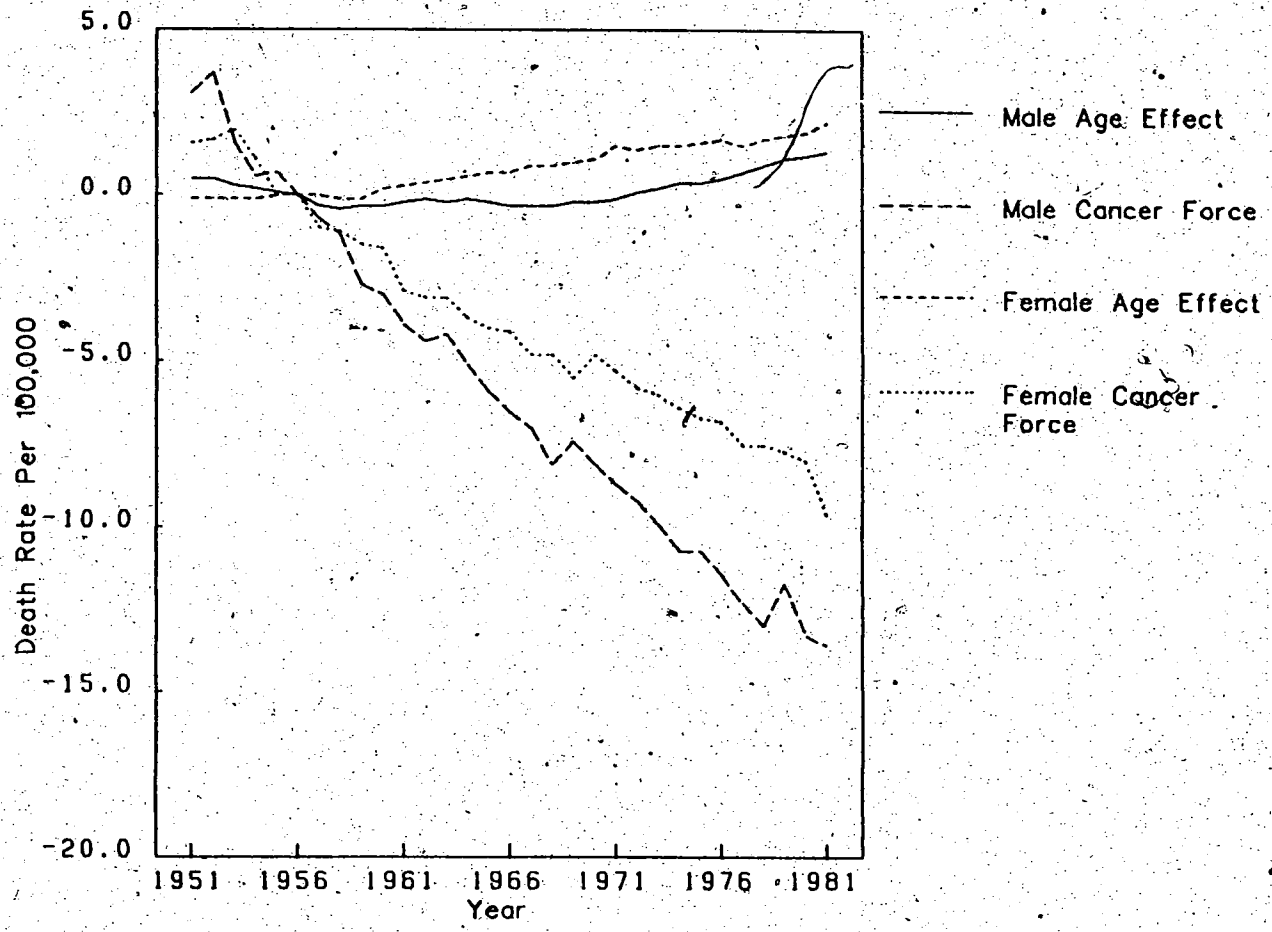
These opposing trend movements illustrate influences from the age structure, Faster aging of the female

Fig. 5.10 Geometric Means of Age Specific Stomach Cancer Death Rates by Sex, Canada, 1951-1981



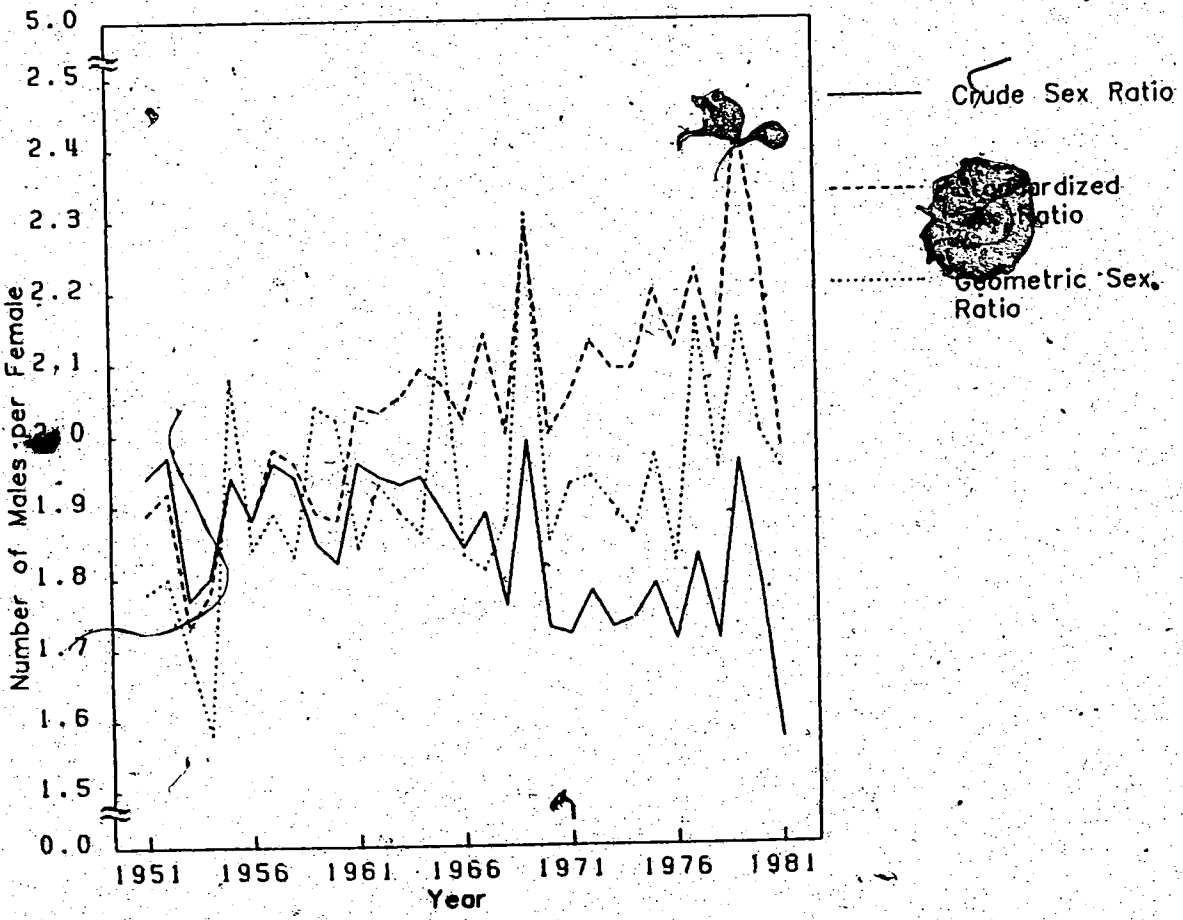
Source: Trend analysis.

Fig. 5.11 Age Effect and Cancer Force of Stomach Cancer Death Rates by Sex, Canada, 1951-1981



Source: Trend analysis.

Fig. 5.12 Sex Differentials in Stomach Cancer, Canada, 1951-1981



Source: Trend analysis.

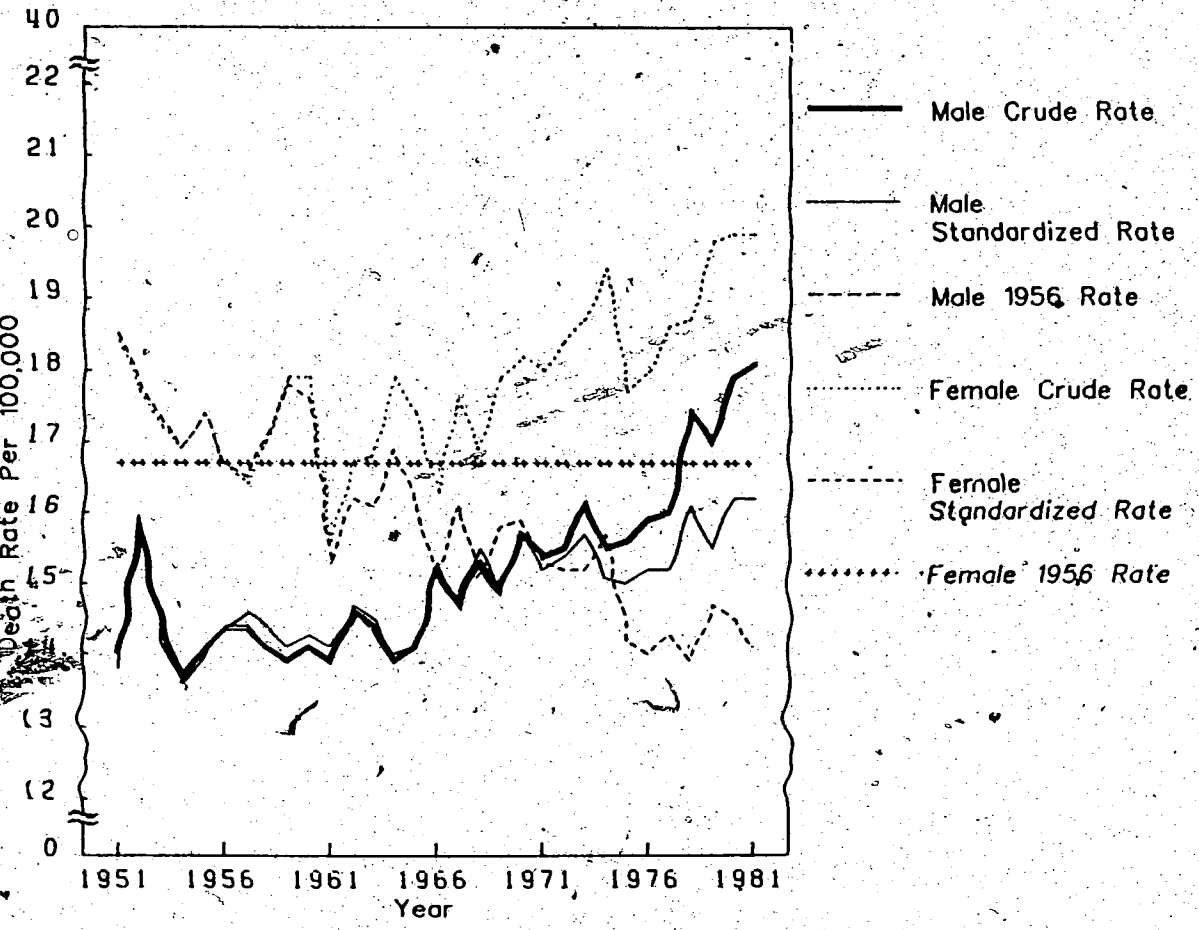
population could have produced higher crude death rates which in turn, lower the sex mortality ratio. The geometric means, which benefit from no age biases, indicate less excess male mortality than the adjusted rates.

5.5 Cancer of the Intestines

Cancer of the intestines is, perhaps, one of the few cancer sites for which females rather than males are experiencing more unfavourable conditions. Between 1951 and 1981, female crude death rates were consistently higher than for males. However, we can see in Fig. 5.13 that standardized death rates were declining. Also, they have fallen below the 1956 base rate since the mid 1960s. As a result, the female cancer force (see Fig. 5.14) was shrinking. Unfortunately, this decline is overpowered by the increasing age effects.

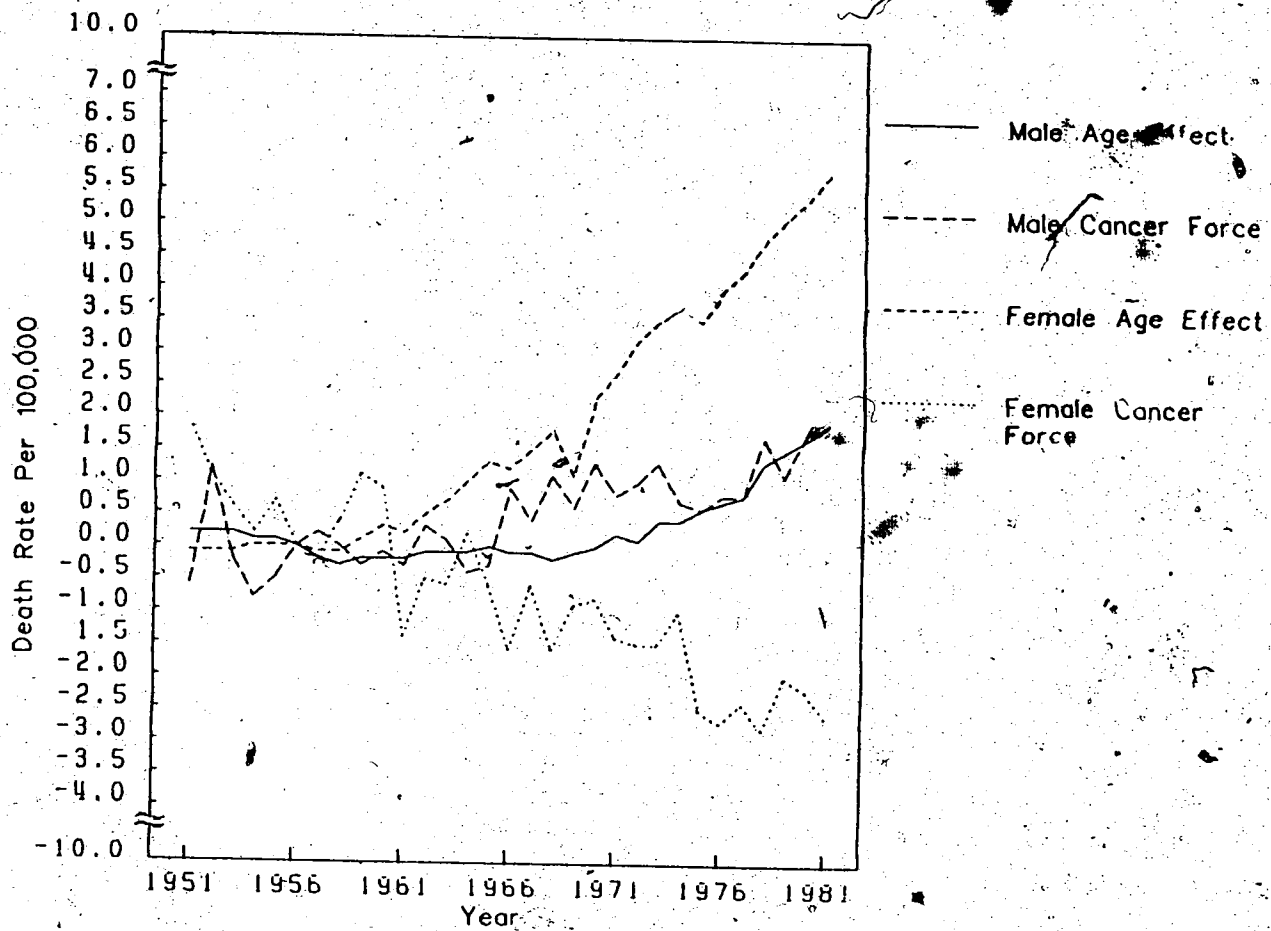
For males, the situation is not encouraging either. Both the crude and the standardized death rates are increasing. Therefore, the age effects and cancer force are also getting stronger. The drastic differences between male and female intestinal cancer can be seen easily if we exclude the low risk age groups. From Fig. 5.15, it is obvious that male geometric means were rising gradually but steadily. This rise is in sharp contrast to the fast-dropping female geometric means. A summing up of all the evidence, suggests that expanding sex differentials are unavoidable.

Fig. 5.13 Crude and Standardized Intestinal Cancer Death Rates by Sex, Canada, 1951-1981



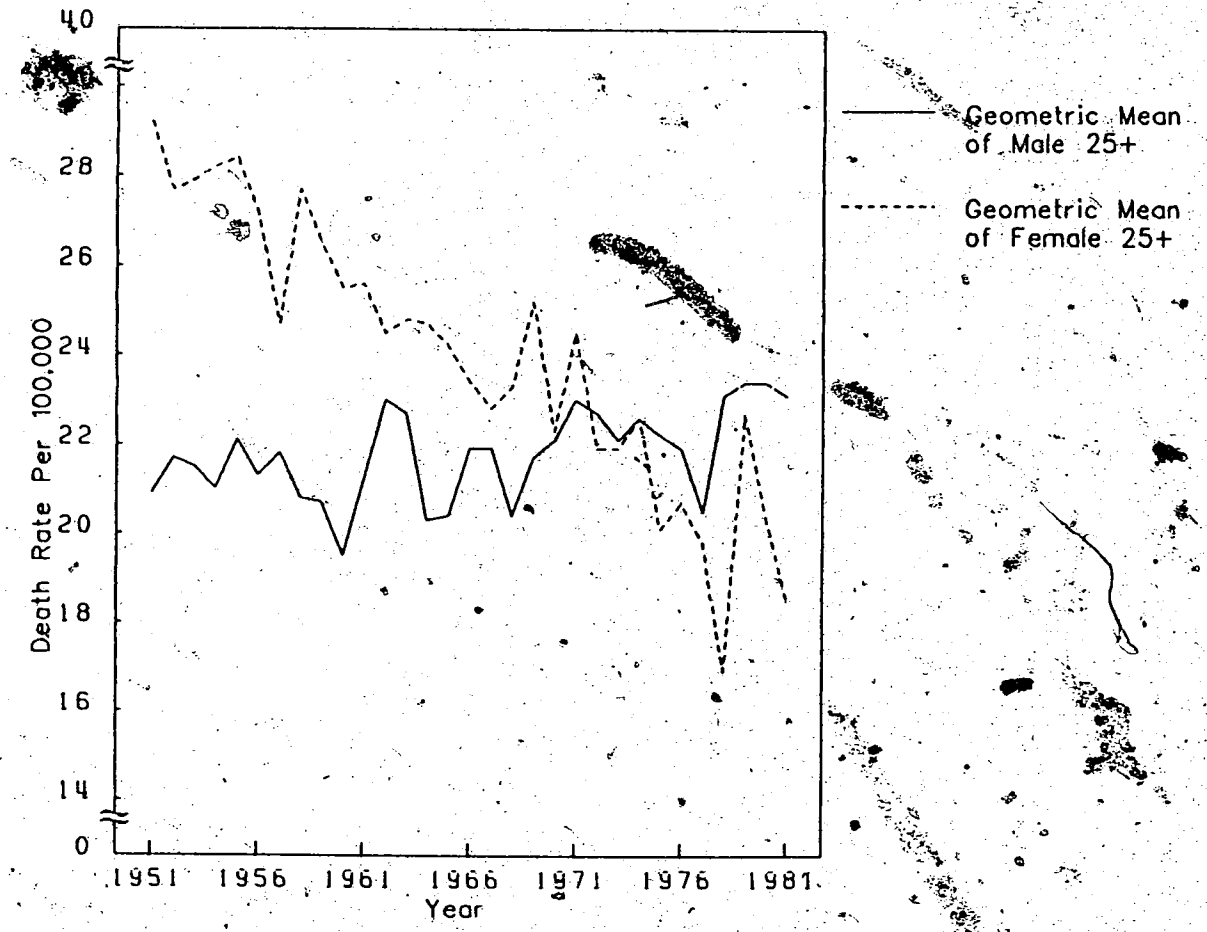
Source: Trend analysis.

Fig. 5.14 Age Effect and Cancer Force of Intestinal Cancer Death Rates by Sex, Canada, 1951-1981



Source: Trend analysis.

Fig. 5.15 Geometric Means of Age Specific Intestinal Cancer Death Rates by Sex, Canada, 1951-1981



Source: Trend analysis.

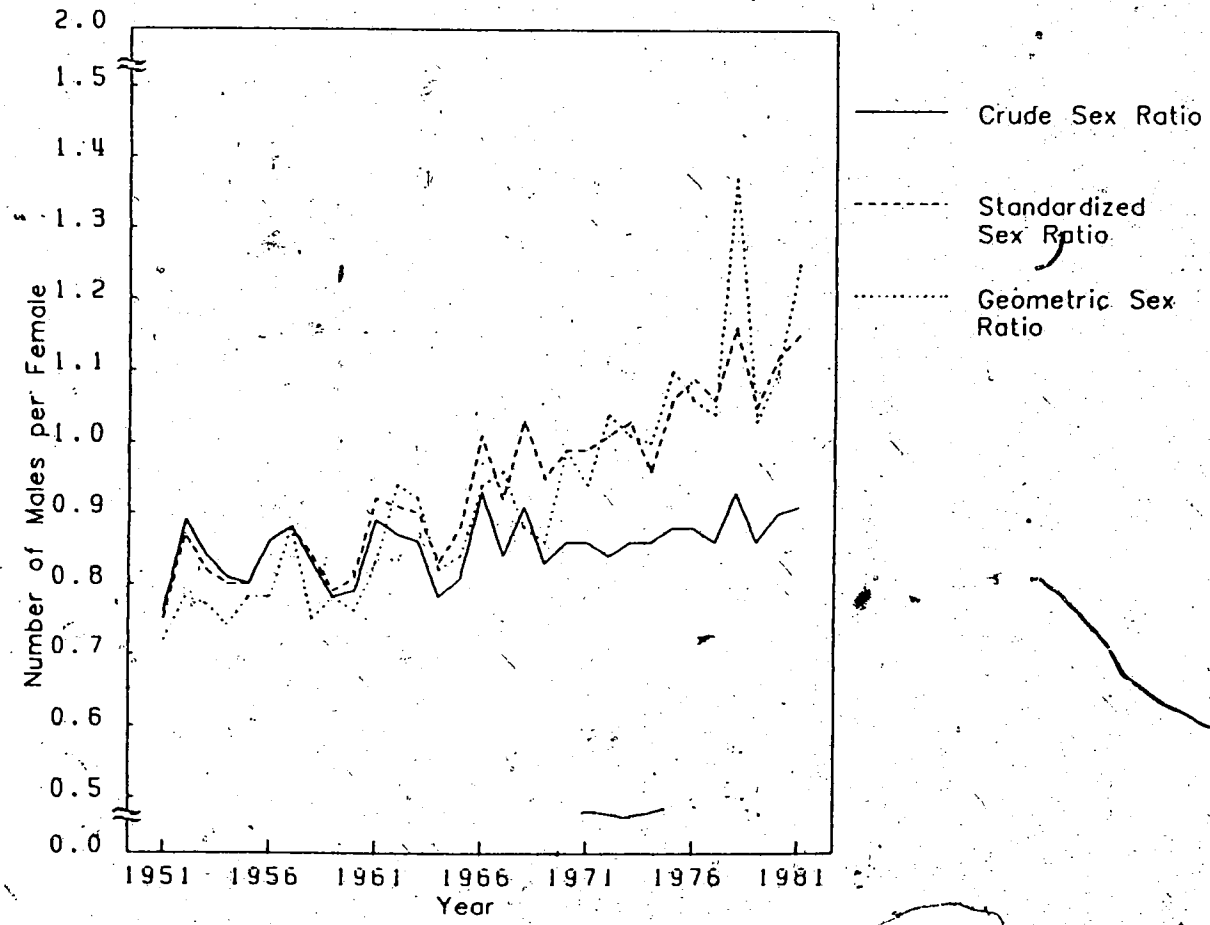
In Fig. 5.16, we observe that most of the three types of sex mortality ratios were below 1.0 prior to 1976. Large numbers of female deaths during this period had kept the ratios down. Since then, the ratios increased as male intestinal cancer deteriorated. The fastest growing ratio from geometric means indicates that conditions are less encouraging for male, and that excess male deaths can be anticipated in the near future.

5.6 Cancer of the Bladder

In comparison with other cancer sites, bladder cancer appears to be less threatening. Male death rates are about three times higher than for females, and have not increased by more than seven deaths per 100,000 throughout the period of 1951-1981. In Fig. 5.17, we can see that there were moderate increases in male crude and standardized death rates until 1971. Since then the trends, especially of the standardized rates, shift downward. Similar changes over time are exhibited for females, except for their stable crude death rates.

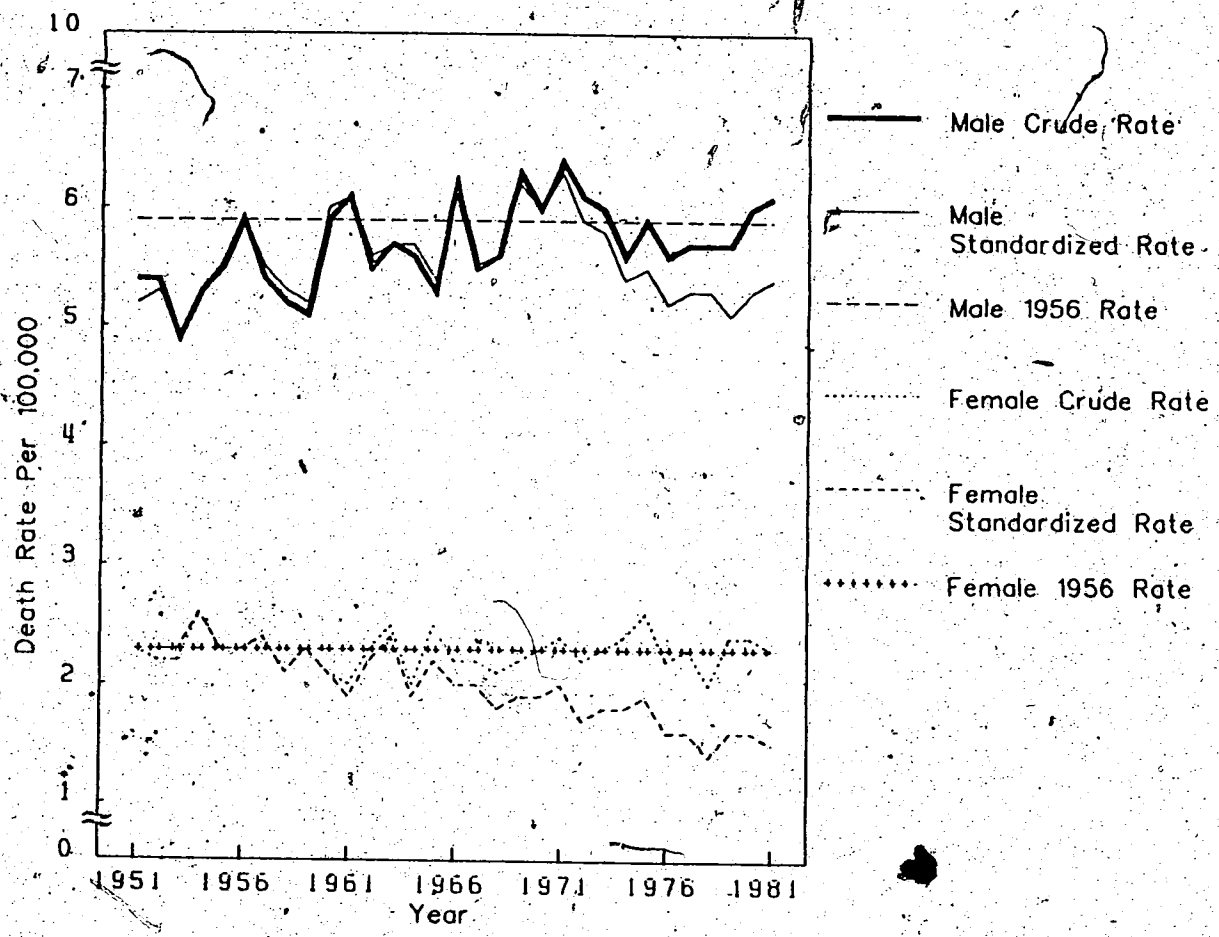
Considering only the high-risk age groups, the mortality patterns shown in Fig. 5.18 drastically alter our impression of bladder cancer. For both males and females, the geometric means of the rates are almost three times the crude and standardized death rates. Such differences reflect biases introduced by including large low-risk age groups, as in the case of crude and standardized rates. The actual

Fig. 5.16 Sex Differential in Intestinal Cancer Mortality, Canada, 1951-1981



Source: Trend analysis.

Fig. 5.17 Crude and Standardized Bladder Cancer Death Rates by Sex, Canada, 1951-1981



Source: Trend analysis.

mortality condition for persons middle-aged or older are not encouraging at all.

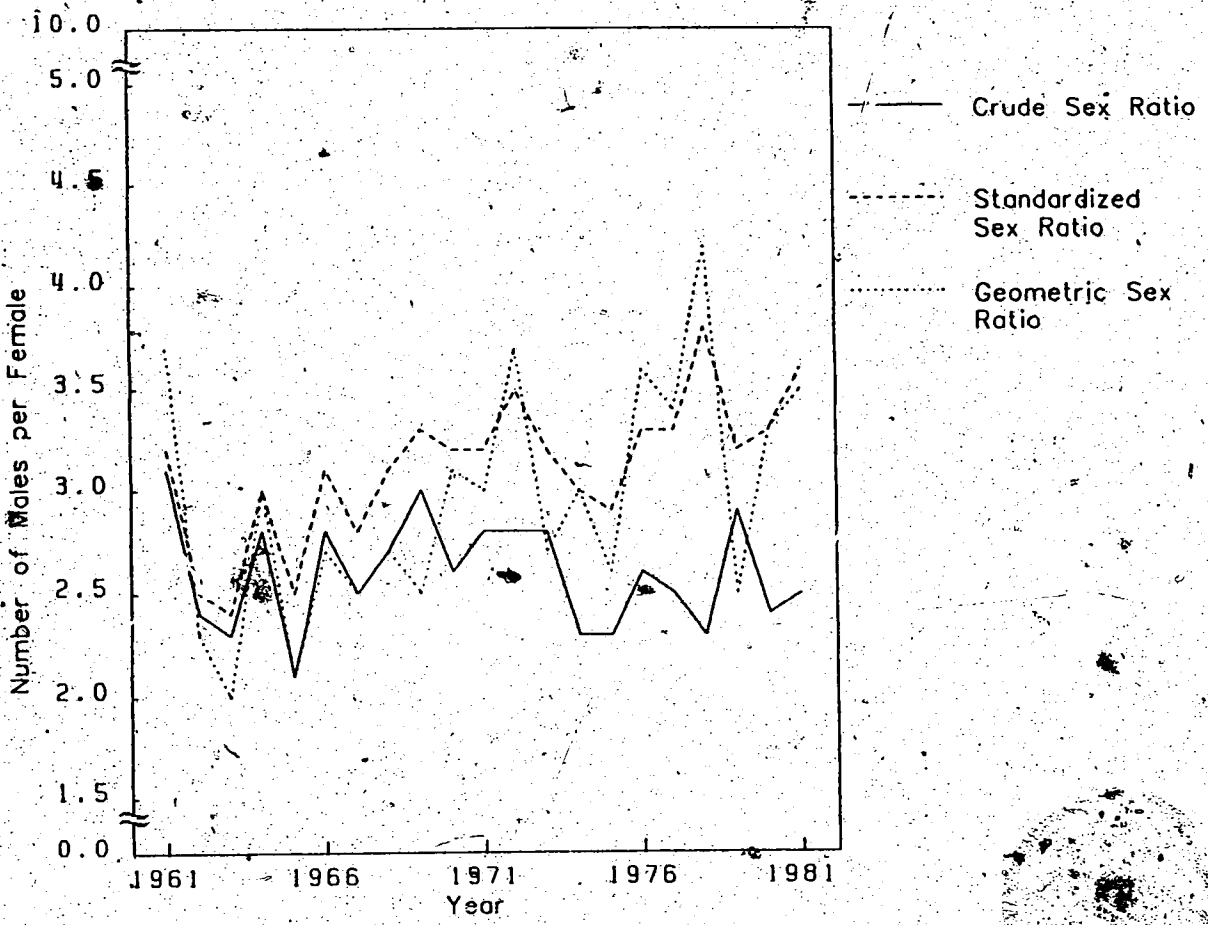
Due to the close proximities of crude and standardized rates, the age effects for both males and females are rather small. Nevertheless, the age effects have increased since the mid-1960's. On the contrary, cancer force has declined among females throughout the entire period but only since the early 1970's for males. This information is evident from Fig. 5.19. As far as the sex differential is concerned, the ratios of crude death rates (see Fig. 5.20) are again small.

The other two ratios are much higher and have remained fairly stable since the mid-1960's. In contrast to other cancer sites, the increase in bladder cancer for males and females alike has drawn momentum largely from the age structure. For this reason, the older age groups require careful monitoring for the actual factors causing their high risk.

5.7 Cancer of the Breast

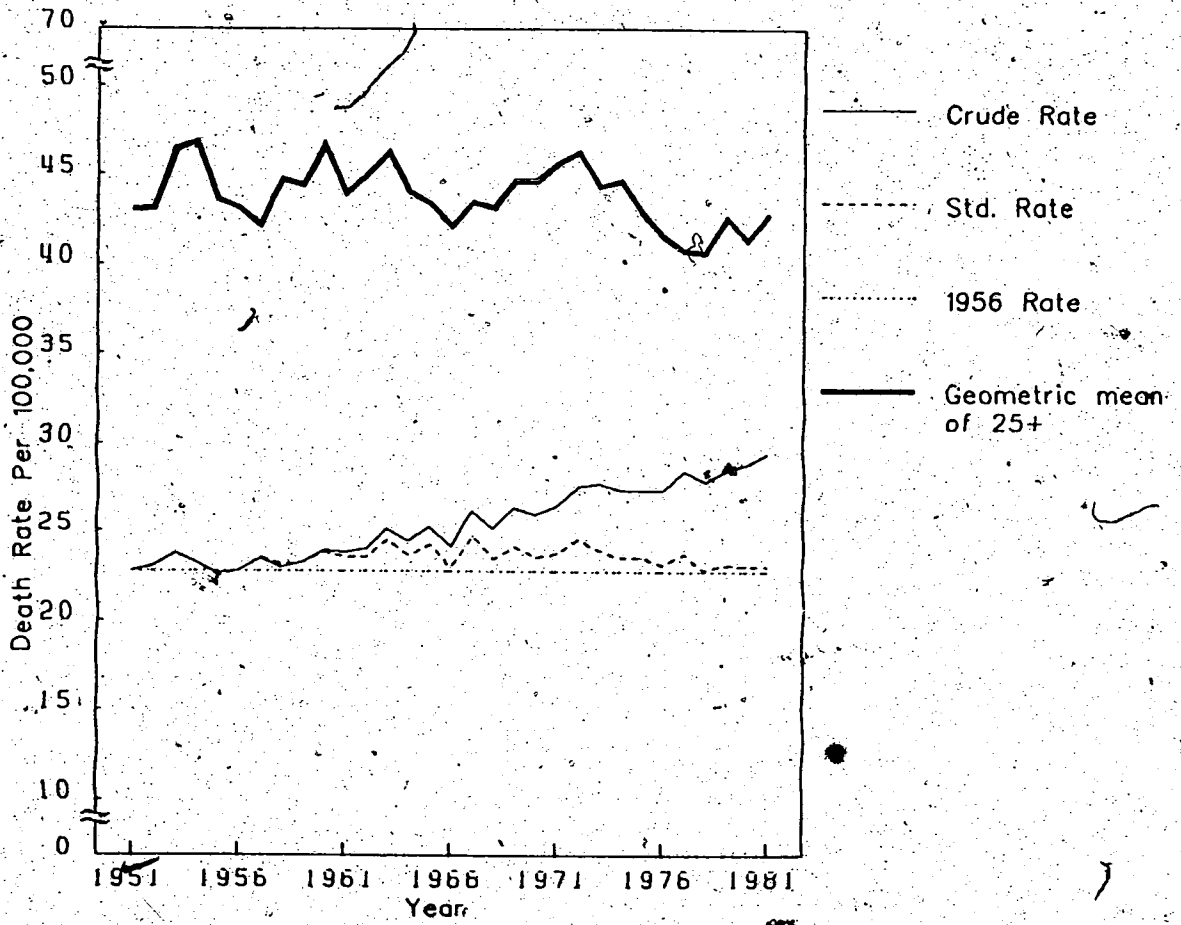
In 1951, approximately 19 per cent of all female cancer deaths were due to breast cancer. By 1981, this figure had increased marginally to 20 per cent. Despite the fluctuations within this period, death rates from this cancer site had experienced some improvements. The standardized death rates, as well as the geometric means shown in Fig. 5.21, reveal slight declines, notably from the early 1970's.

Fig. 5.20 Sex Differentials in Bladder Cancer Mortality, Canada, 1951-1981



Source: Trend analysis.

Fig. 5.21 Crude and Standardized Death Rates, and Geometric Mean of Age Specific Female Breast Cancer Death Rates, Canada, 1951-1981



Source: Trend analysis.

Regarding age effects, the increasing trend as seen in Fig. 5.22 clearly demonstrates the impact of the aging female population on breast cancer. On the other hand, cancer dorce was comparatively stable until the mid-1970's when it started to decline. More careful examination found that the cancer force was rather small in 1955-1961. Thus, there appears to be a gentle cycle in the cancer force in the three decades.

If fertility is one of the major factors in breast cancer, then recent decline in cancer force could be due to high fertility during the baby boom period. Women of childbearing ages during this time are now protected by their high fertility. Similarly, declining fertility recorded between 1901-1945 (Kalbach and McVey 1979) could have resulted in high cancer force during 1961-1973. To better identify the cohort differentials in breast cancer, a longer time series is needed.

5.8 Summary

In this analysis, estimates of age effects and cancer force are utilized, along with other types of death rates, to account for cancer mortality trends between 1951 and 1981. It is seen that cancer force for both sexes has declined with regard to cancer of the bladder and the stomach. Breast and the intestinal cancer have decreased for females. Only the cancer force for lung cancer in both sexes and for male intestinal cancer have significanctly increased

during this period.

Without exception, age effects are positive for all cancer sites. That is, an aging population structure has contributed to an increase in cancer mortality rates or has prevented them from falling to lower levels. In almost all cancer sites, females show higher age effects than males. If we assume that cancer force will remain unchanged for a sufficiently long period of time, and population aging continues in the same fashion, then we can anticipate that females will have higher cancer mortality than males because of their large age effects.

6. Statistical Model of Cancer Mortality

6.1 Introduction

The applications of age effects and cancer force have been demonstrated in our interpretations of cancer mortality trend. In addition, they can be utilized to set up statistical models to predict cancer death rates for upcoming years. The prediction of male and female total cancer mortality only will be attempted here. Statistical models for other cancer sites, pending success of the present efforts, are left for later analysis.

Recall from discussions of methodologies, that the age effects are expressed as functions of annual growth rates of three broad age groups, 25-44, 45-64, and 65 or older. Linear terms of these three age groups will form the basic equation. Higher order terms will be added if dictated by their relationships with the dependent variable. The cancer force is expressed as a function of time.

Conventional stepwise regression technique is used to fit the above models. In case of high collinearity, the method of ridge regression is used. Regression diagnostic methods are also employed in a stage by stage manner to maximize the goodness of fit of the models. When the equations are established, the two components will be first estimated. Then the equations can be rearranged algebraically to derive the estimate of future cancer death rates for males and females separately.

6.2 Male Age Effect

As shown in Table 6. there are moderate correlations between the age effect and t growth rates of the three age groups. The size of correlations is in the range of 0.6 to 0.7 and appears supportive of our hypothesized relationships. When we examine the distributions of these variables, age effect is the only one found highly skewed. The measurement of skewness is 1.7, indicating that not only are the above correlations deceptive, but the assumption of normality fundamental to regression analysis is violated.

In order to derive meaningful correlations, and more important, to conform to the regression assumptions, age effect must be transformed to achieve hormality. The most frequently adopted procedure is to compress the distribution by means of taking the logarithm of the original values.

For our situation, negative age effects prevent us from making this direct application since the logarithm of negative values is not defined. To circumvent the problem, an appropriate constant can be selected and added to the original values prior to taking the logarithm (Cook and Weisberg 1982). This will shift the scale of the variable but render no influences upon the nature of the data. For our analysis, a value of 5 is added to the age effect before the logarithm is taken.

Table 6.1 Initial Correlations of Male Age Effect and Growth Rates, Canada, 1950-1981

	Agemcc	Cm2544gr	Cm4564gr	Cm65gr
Agemcc	1.00			
Cm2544gr	0.625	1.00		
Cm4564gr	-0.749	-0.381	1.00	
Cm65gr	0.696	0.675	-0.700	1.00

Source: From initial multiple regression.

Table 6.2 Correlations of Male Age Effect and Growth Rates, Transformed Variables, Canada, 1950-1981

	Agemcc	Cm2544gr	Cm4564gr	Cm65gr
Agemcc	1.00			
Cm2544gr	0.720	1.00		
Cm4564gr	-0.811	-0.381	1.00	
Cm65gr	0.626	0.675	-0.700	1.00

Source: From multiple regression.

The transformed age effect has a skewness value of of 0.66, which is greatly improved. The correlations between this more normally distributed age effect and the independent variables also changed along with the transformation. The new correlations in Table 6.2 are are much stronger than those without the transformation. Unexpected though, is the negative correlation between age effects and the 45-64 age group. This could due to the declining annual growth rate of this group during the last decade. For the remaining two groups, the trends are increasing. The correlations among the three age groups are moderate and present no immediate threat of multicollinearity. Yet, such possibilities have to be carefully monitored to avoid unstable regression coefficients.

Before multiple regression is performed, each of the three age groups is tested on a bivariate basis. In all three cases, regression coefficients are highly significant. The R^2 values of about 0.67 are highest for the two oldest age groups. For the 25-44 groups, the R^2 is only around 0.5. This pattern of explained variations is well within our expectations. That is, the older age groups which have higher cancer risks explain a larger share of the observed age effects. A summary of bivariate results is provided in Appendix 1.

Turning our attention to multiple regression, we find that the curve fitting procedure is complicated by the

presence of some outliers. The influences of outlying points in regression analysis and their treatments have been dealt with in numerous publications (for example, Cook and weisberg 1982; Chatterjee and Price 1977; Daniel and Wood 1980). Deletion of outliers are discouraged unless they are true random fluctuations and are not results from special circumstances.

Since age effects are products of annual crude and standardized death rates, random fluctuations can be expected. The deletions of outlying points are therefore adopted. By doing so, however, we have the disadvantages of reduced sample size and variability.

Initial multiple regression results are presented in Table 6.3. Growth rates for males 65 years or above are first selected into the equation. It accounts for some 68 per cent of age effect variation. However, it becomes redundant as the other two groups are added to the equation. High correlation between 24-44 and 65 or above seems to be the reason of such changes.

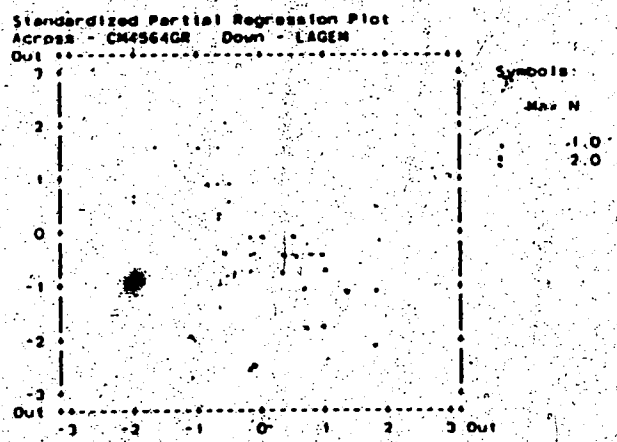
While this potential collinearity problem requires attention, it has been stressed that such issues in regression analysis should be tackled only after the model has been properly specified (Gunst and Mason 1980). Our equation at this stage thus consists of only two independent variables. Yet together they still produce a R^2 of 0.87. Nevertheless, evidence for needed improvements came from the rugged normal probability plot shown in Fig. 6.1. If a model

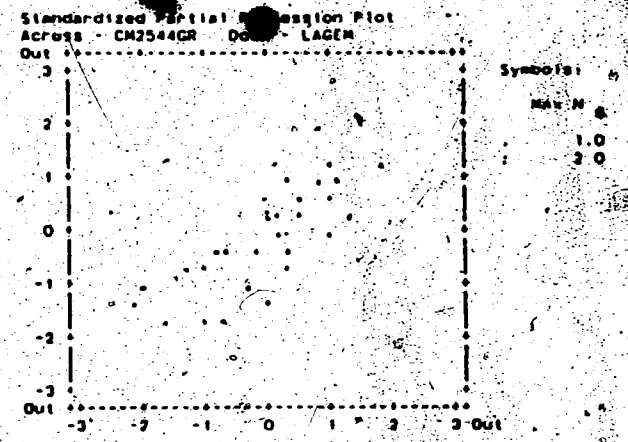
Table 6.3 Male Age Effect, Initial Regression Model, Canada, 1950-1981

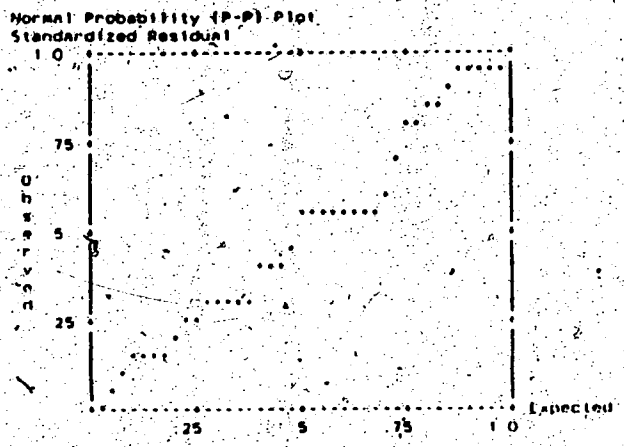
	Coefficient	T Statistics
Constant	2.5997	
25-44	0.3616	6.284
[illegible]64	-0.8225	-8.205
R^2 (adj.)	0.8449	
Standard Error	0.2647	
D-W Statistics	0.9295	
Number of Cases	32	

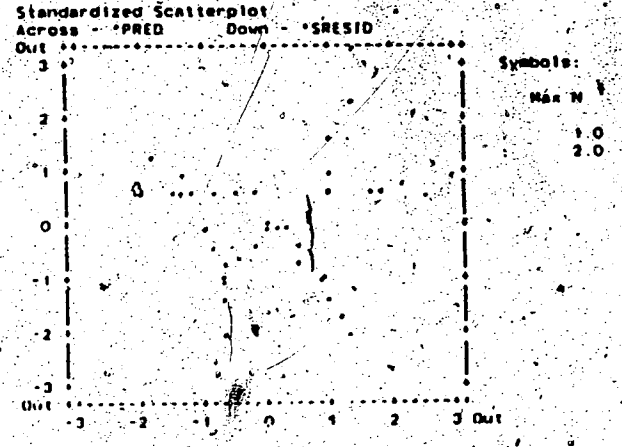
Source: From regression analysis

Fig. 6.1 Residual and Normal Probability Plots of Male Age Effect, Initial Regression Model, Canada, 1950-1981









Source: Regression analysis.

fits the data well, a pattern of residuals should fall more or less on a straight line except for minor fluctuations at either ends (Gunst and Mason 1980).

From the partial residual plots in Fig. 6.1, the growth rates specified in linear form seems adequate. But, two outlying residuals are observable in the diagram for the 45-64 group. Even more outliers can be found when the overall residuals are plotted against the predicted values. A group of six maverick points separates itself from the main cluster in the upper right corner.

It is apparent that this group should be distinguished from the rest. By means of the standardized predicted values, these outlying points are identified to include the years from 1976 to 1981. A dummy variable is therefore added to the model to represent the years after 1976. Regression is then repeated, and the results are presented in Table 6.4. The re-specification of this model brings substantial improvements. The overall residual plot in Fig. 6.2 gives more randomly distributed residuals.

Nevertheless, the normal probability plot given in the same diagram still indicates room for changes. Our next step is to check for possible outliers. For this, we would rely on formal residual statistics like the leverage value and the studentized deleted residual (or simply studentized residuals). The former, being the diagonal elements of the Hat matrix $X(X'X)^{-1}X'$ measures the influence of the i-th observed response on its predicted score (Gunst and Mason

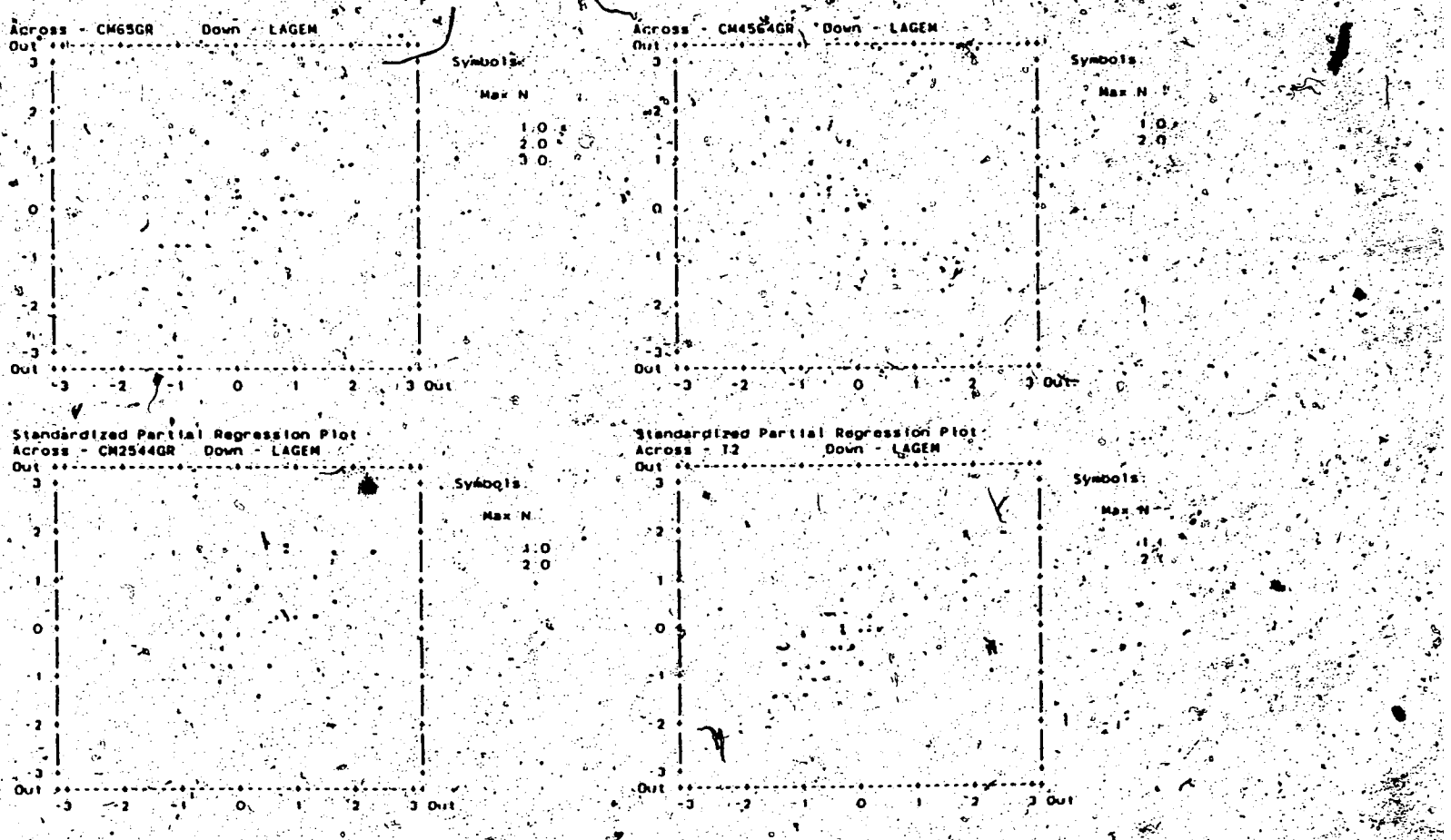
Table 6.4 Male Age Effect, Re-specified Regression Model, Canada, 1950-1981

	Coefficient	T Statistics
Constant	1.9623	
25-44	0.3154	10.911
45-64	-0.4682	-7.949
T	0.6064	8.284
R^2 (adj.)	0.9653	
Standard Error	0.1229	
D-W Statistics	1.7576	
Number of Cases	28	

Note: T stands for years after 1976 inclusive

Source: From regression analysis

Fig. 6.2 Residual and Normal Probability Plots of Male Age Effect, Re-specified Regression Model, Canada, 1950-1981



Source: Regression analysis.

1980). Values exceeding 2p/n have been suggested as potential outliers (Belsley et al. 1982).

The latter are modified versions of the usual residuals, but now assume a t-distribution with n-p-1 degrees of freedom. It measures how the regression equation would change if a particular suspected outlying point is removed from the analysis. Residuals greater than 2 are problematic (Belsley et al. 1980; Gunst and Mason 1980). Using these measurements, we notice that 1958 in the casewise plot in Appendix 2 has a large studentized residual.

This particular data point is then tested with a dummy variable. The testing finds it with a significant impact on both the slope and the intercept. It is therefore deleted from the analysis. Its removal, however, brings the data points for 1957, 1967, and 1966 as outliers. These three points are also eliminated after individual testing.

Regression results in Table 6.5, with four outliers deleted, show more improvements over those last reported. The R^2 has increased slightly from a previous 0.95 to 0.97, and the standard error of estimates is reduced quite markedly from 0.16 to 0.12. Not surprisingly, the age group of 65 and above, is removed from the equation. If we return to its partial residual plot in Fig. 6.2, we can see that the residuals, in fact, did not display an especially strong positive relationship with age effect. When the influential points are removed, this weak association is diminished.

Table 6.5 Male Age Effect, Final Regression Model, Canada, 1950-1981

	Coefficient	T Statistics
Constant	1.9623	
25-44	0.3154	10.911
45-64	-0.4682	-7.949
T	0.6064	8.284
R^2 (adj.)	0.9653	
Standard Error	0.1229	
D-W Statistics	1.7576	
Number of Cases	28	

Note: T stands for years after 1976 inclusive

Source: From regression analysis

With regarding to the D-W statistic, the current value of 1.76 is above the required 1.65 to free us from auto-correlations. Despite these encouraging results, the normal probability plot in Fig. 6.3 remains rather rough. Also, the overall residual plot shows two large negative and one positive studentized residuals. These three points are identified to be 1968 and 1976, and 1981, respectively.

Our next attempts are to test them with dummy variables. Although the results are all significant, their actual removals only yield barely noticeable changes. These, in fact, accord with their small leverages as compared to the maximum allowable 0.286. None of them actually has a dominative effect on the coefficients.

Also, from the partial residual plots in Fig. 6.3, and the casewise plots in Appendix 3, the independent variables indicate no abnormalities. The rugged normal probability plot might just indicate the inefficiency of regression technique in dealing with time series data that have substantial cyclic patterns. Therefore, no more cases are deleted, and the male age effect equation is as follows:

$$\text{Male Age Effect} = 1.9623 + 0.1354\ (\text{P.A.G. of } 25\text{-}44) - 0.4682\ (\text{P.A.G. of } 45\text{-}64) + 0.6064\ \text{Time}$$

where Time indicates the years after 1976.

Fig. 6.3 Residual And Normal Probability Plots of Male Age Effect, Final Regression Model, Canada, 1950-1981

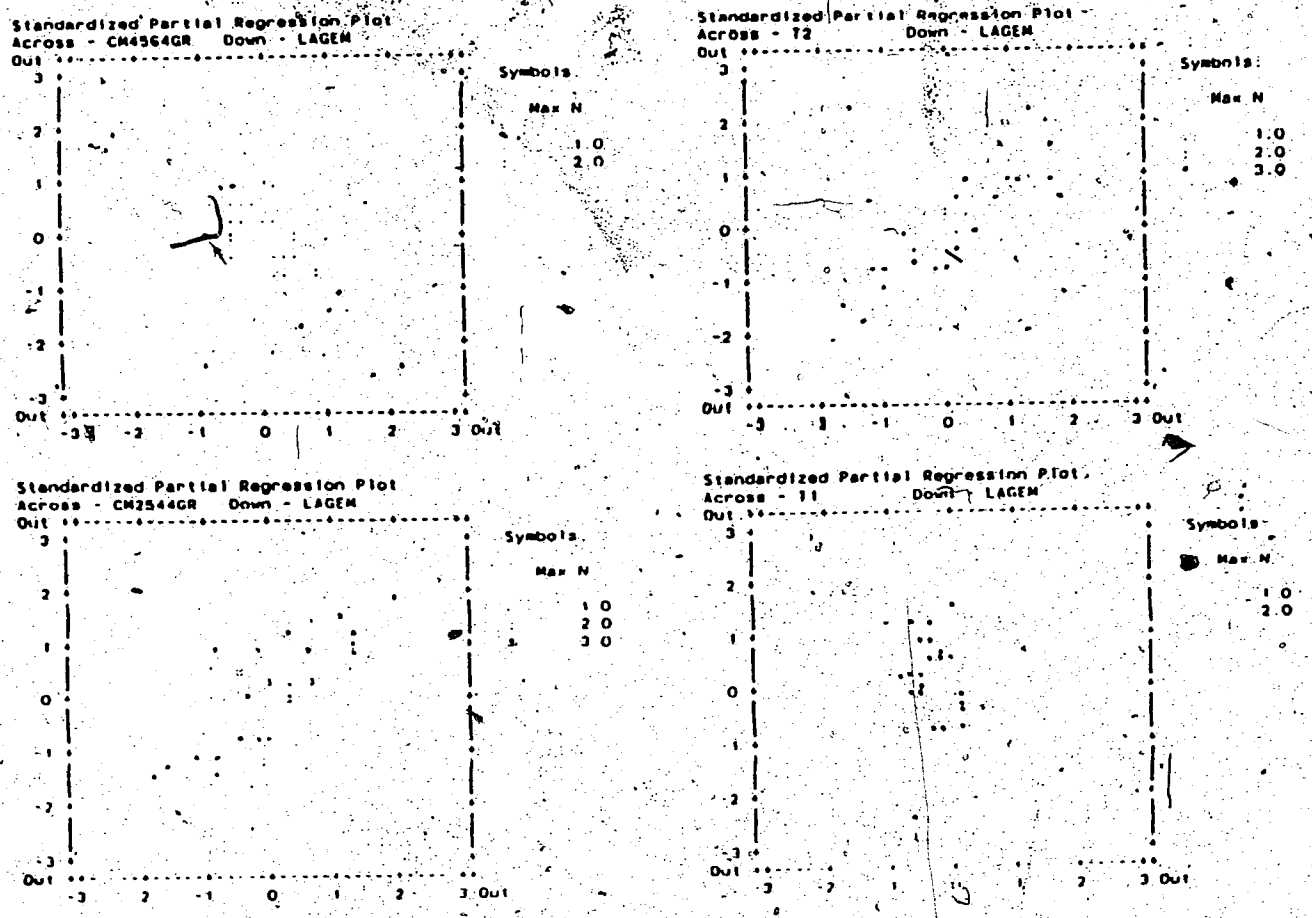
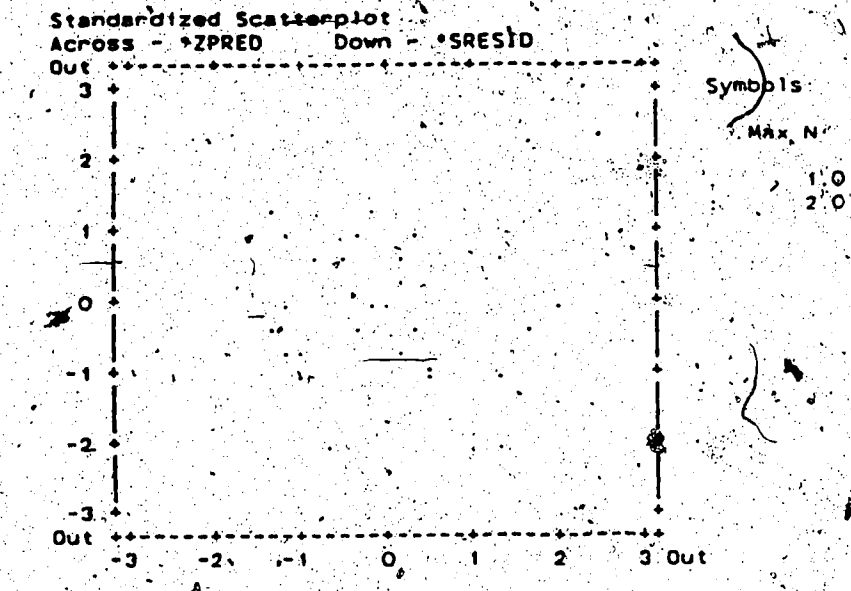
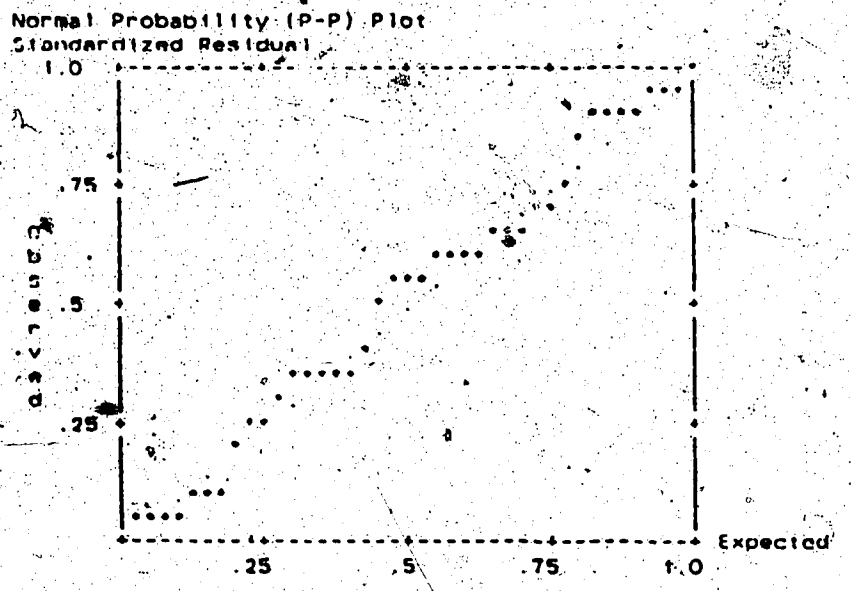


Fig. 6.3 (continue)





Source: Regression analysis...

6.3 Female Age Effect

Curve fitting for female age effects initially involves the same process as for males. A different technique, however, is necessary once the preliminary model is established. To begin, distributions of variables are checked for normality. Age effects and growth rates of 65 or above are found to be slightly skewed. A value of 5 is again added to the age effect before taking the logarithm.

After the transformation, the meausurement of skewness dropped from 0.80 to 0.16. For the elderly age group, the logarithm of original scores has a less satisfactory result. The skewness is reduced only from 1.5 to 1.0. Since other methods of transformation did not result in lower skewness, the values from the logarithm is used. Table 6.6 gives the correlations among these four basic variables.

Relationships between age effects and growth rates are very weak. The correlations among the independent variables are even stronger. Collinearity is potentially a problem. Similar to males, the 45-64 group is also negatively related to age effects. As we shall see later, this is dictated by the relationships with other variables. Despite the disappointing correlations, bivariate regression is carried out. The results in Appendix 4, show the growth rate of each age group explains, on average, merely about 20 per cent of the variations in age effects.

With multivariate regression, only the 45-64 group is significant. Others are discarded by the stepwise procedure.

Table 6.6 Correlations of Female Age* Effect and Growth Rates, Transformed Variables, Canada, 1950-1981

	Agemcc	Cm2544gr	Cm4564gr	Cm65gr
Agemcc	1.00			
Cm2544gr	0.398	1.00		
Cm4564gr	-0.526	-0.685	1.00	
Cm65gr	0.385	0.365	-0.634	1.00

Note: Cm2544gr = male 25-44 growth rate.
Cm4564gr = male 45-64 growth rate.
Cm65gr = male 65+ growth rate.
Source: Regression analysis.

Nonetheless, these findings are not surprising at all. The two excluded age groups have very low correlations with the age effect on one hand, but are highly associated with the 45-64 group, on the other. The correlation is high between age effect and this middle-age group.

Therefore, the 25-44 and the 65 plus age group become redundant once the remaining age group is selected into the equation. Compared with the significant results of all three age groups in bivariate regression, there is firm evidence of collinearity. An additional problem of auto-correlation reveals itself with a low D-W statistics of only 0.14.

At this stage, it seems that the model for female age effect is going out of control. Yet, the residual pattern offers clues for modifications. The casewise plot in Appendix 5 shows an upward trend of residuals after 1964. To be exact, two more sections can be identified. Between 1950-1958, the residuals are negative but decreasing over time. During 1959 and 1963, the residuals are stablized slightly below 0.

In general, the residuals from these two periods are negative. They imply that the predicted age effects are overestimated. Contrarily, predictions after 1964 are underestimated. Based on such distinctions, separating the entire trend into two portions seems appropiate. As far as the outliers are concerned, the studentized residuals indicate that 1950 and 1951 are potential maverick points. Instead of dealing with them first as we did before, the

model must be re-specified. The two time trends should not be combined and treated as one.

Instead of repeating the regression procedures for years before and after 1964, a dummy time variable is incorporated into the model. It has a value of 1 for years greater than or equal to 1964, and 0 otherwise. The categories are so designated because we are more interested in the age effects after 1964. By itself, this dummy variable measures impacts of the latter period on the intercept, or it indicates whether the post-1964 years on the whole differs from the previous years in their age effects.

To assess the impacts on slopes, the time indicator is multiplied with the growth rates of the three age groups. If they are significant, then the growth rates after 1964 have special influences on the observed age effects. These modifications increase the number of independent variables from the original three to the current seven. Their correlations in Table 6.7 disclose much more encouraging results.

All growth rate and time interactions, except those from the middle-age group, are highly correlated with age effect. Undesirably, however, there are high correlations among the variables. The only exclusion is between 25-44 and 45-64. Multicollinearity is definitely a problem in our analysis.

Table 6.7 Correlations of Female Age Effect and Growth Rates, Trends Added, Canada, 1950-1981

	LAGEF	CF2544GR	CF4564GR	LCF65	T	TCF25	TCF45	TCF65
LAGEF	1.000	.375	-.497	.406	.878	.933	.585	.917
CF2544GR	.375	1.000	-.673	.380	.203	.614	-.101	.232
CF4564GR	-.497	-.673	1.000	-.680	-.223	-.547	.193	-.332
LCF65	.406	.380	-.680	1.000	.238	.291	-.050	.403
T	.878	.203	-.223	.238	1.000	.817	.882	.978
TCF25	.933	.614	-.547	.291	.817	1.000	.512	.823
TCF45	.585	-.101	.193	-.050	.882	.512	1.000	.798
TCF65	.917	.232	-.332	.403	.978	.823	.798	1.000

Note: LAGEF= Age Effect; CF2544GR= 25-44 Growth Rate;
CF4564GR= 45-64 Growth Rate; LCF65= 65+ Growth Rate;
T= Trend, 1964+; TCF25, TCF45 and TCF65 are
Interactions of T and the three growth rates.
Source: From Regression Analysis.

Despite the collinearity, regression results are impressive. The value of R^2 increases from a low of 0.27 before to the present 0.98. The standard error of residuals also reduces from 0.67 to 0.13. Although the residuals in the casewise plot in Appendix 6 appear more random, two distinct patterns emerge. In terms of the variables, not all of them are significant. Only the three interactions and the 25-44 growth rate are useful. It is not unexpected to find the importance of the interactions, since the female age effect is increasing rapidly, primarily since the early 1960's.

The maverick points, 1959 and 1969, have been replaced by 1950 and 1951 as the outliers. In the partial residual plots of Fig. 6.4, the growth rate of 25-44 by itself or through interaction with time appear to be curvilinear. The graphs suggest that adding quadratic terms to our basic equation might offer a better fit. Model modifications begin with the testing of 1959 and 1969 as the outliers using two dummy variables and their interactions with the other independent variables. Results indicate that only 1959 is a valid outlier and should be eliminated.

We can see from Table 6.8, that R^2, after the deletion remains around 0.98. Yet, the standard error of the residual is down to 0.12, and the normal plot in Fig. 6.5 becomes smoother. Also, the casewise plot in Appendix 7 reveals almost no apparent patterns. Only one potential outlier, 1966, emerges in this third regression procedure. Later

Fig. 6.4 Residual and Normal Probability Plots of Female Age Effect, Final Regression Model, Canada, 1950-1981

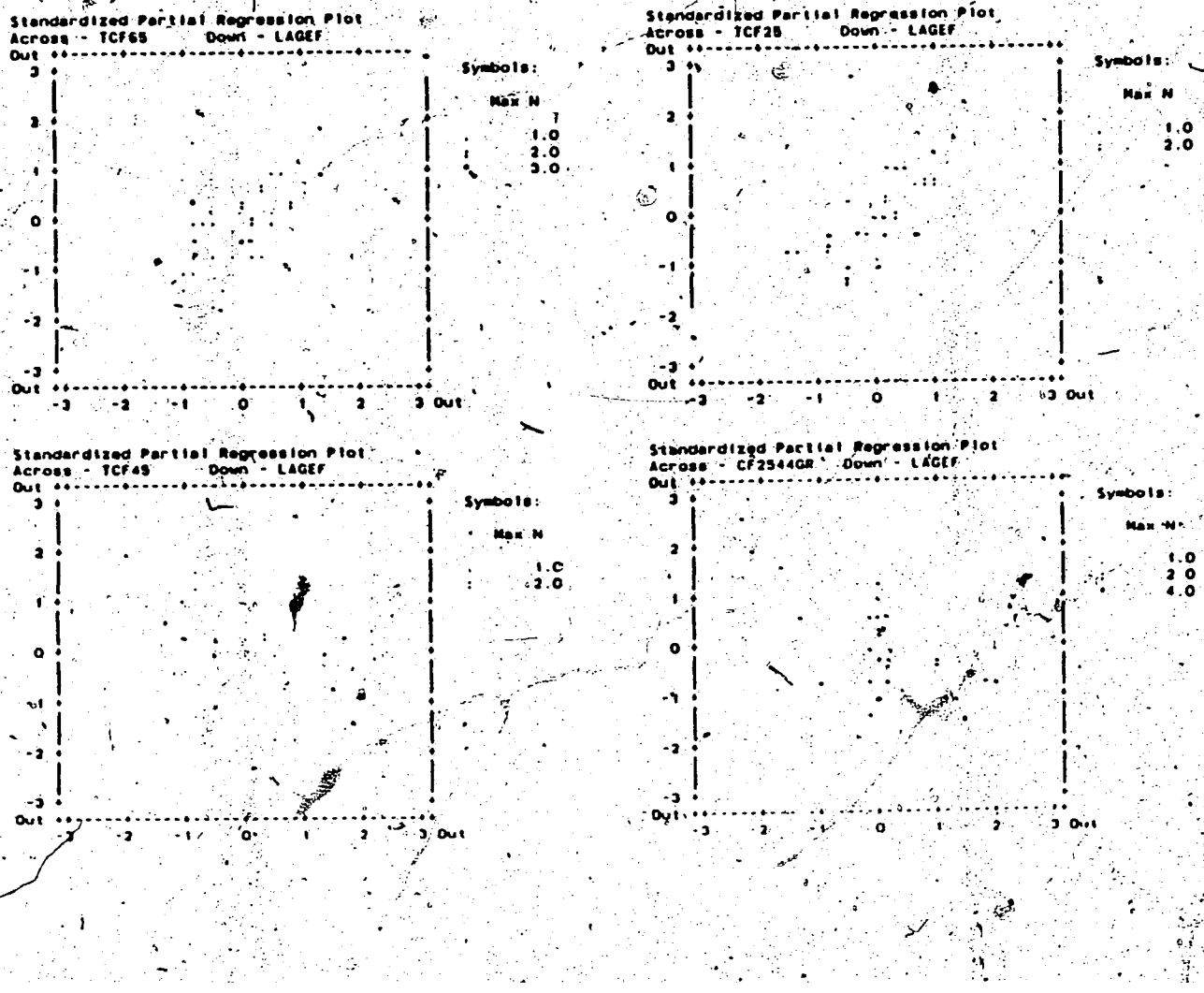
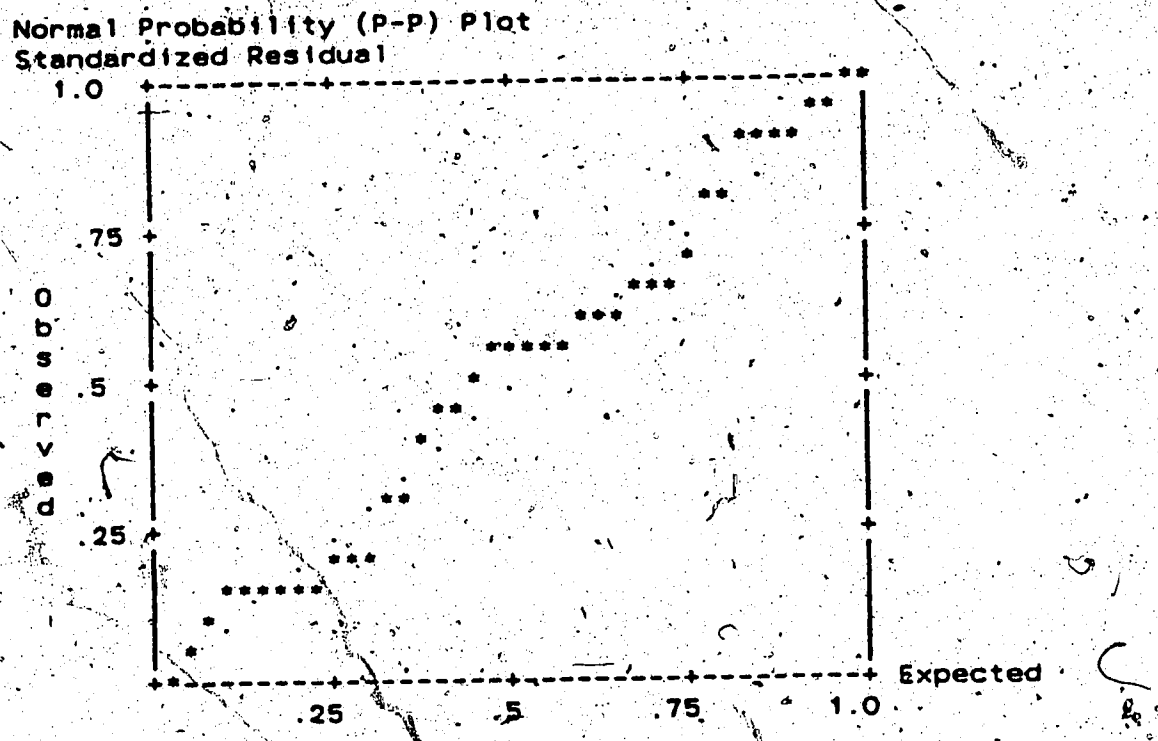


Fig 6.4 (continue)



Source: Regression analysis.

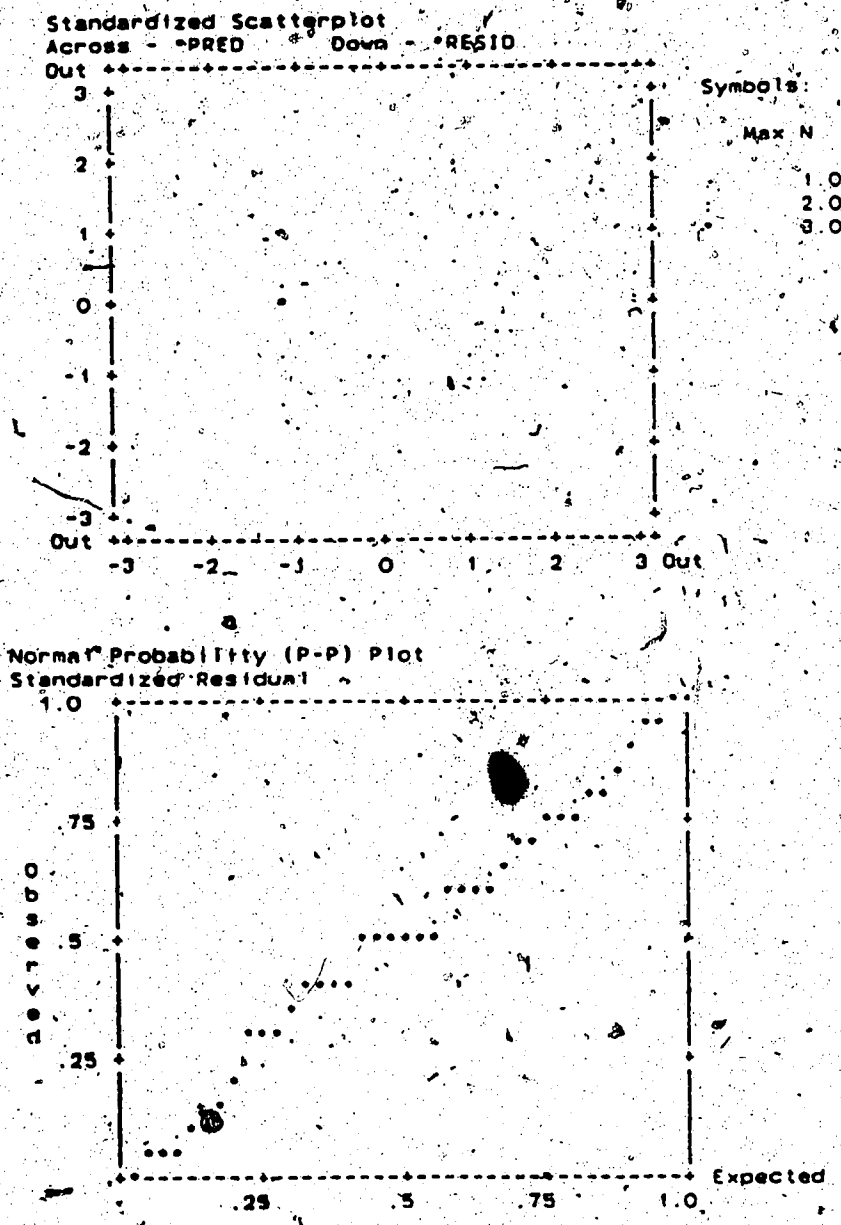
Table 6.8 Female Age Effect, Re-specified Regression Model with 1959 Deleted, Canada 1950-1981

	Coefficient	T Statistics
Constant	0.9513	
25-44	-0.3055	-10.841
65+	1.1909	7.808
25-44 (T)	0.6392	31.583
R^2(adj.)	0.9795	
Standard Error	0.1162	
D-W Statistics	1.494	
Number of Cases	31	

Note: T stands for years after 1964 inclusive

Source: From regression analysis

Fig. 6.5 Residual and Normal Probability Plots of Female Age-Effect, 1959 Deleted, Canada, 1950-1981.



Source: Regression ahalysis.

testing shows that it is not influential and can be retained. At this stage, it seems that a final model has been derived.

Unfortunately, the regression coefficients in Table 6.8 indicate the opposite. Only two of the previously significant variables; growth rate of 25-44 and its interactions with time, continue to be so. The age group of 65 plus, which was excluded from the equation earlier, is now important. The significance of the variables was drastically changed with the deletion of just one outlier, 1959. This is one of the signs that the long suspected collinearity is at work (Chatterjee and Price 1977).

Further investigation of the correlations in Appendix 8 (1959 removed) reveals that the 25-44 and the 65 plus groups actually have a very low correlation with age effect. Their significance in the equation is due to the high correlations with other variables. For example, the interaction term of 25-44 has the highest correlation with age effect. At the same time, it also correlates more with the other two interactions than they do among themselves. Consequently, it is the only one of the three time-growth variables selected into the equation.

Similar scenarios can be observed for the other two significant independent variables. Such multicollinearity precludes one from concluding on the individual effects of the explanatory variables. It also renders the regression coefficients unstable and their test of significance

unreliable because of their large variances. Furthermore, the stepwise regression procedure would be affected (Belsley et al 1982; Gunst and Mason 1980; Chatterjee and Price 1977).

Moreover, to use this model to predict future age effect would be conditioned on a similar collinearity structure among the input data to be used to generate the estimates (Gunst and Mason 1980). Apparently, this is not to be guarranteed by all means. Under these restrictions, the present model has limited predictive value. To remedy this problem, other types of regression techniques are called for.

Among the alternatives, ridge regression is chosen. We may recall that this technique has the advantage of providing estimates of coefficients closest to the true values in the population, if a proper ridge constant is selected. Since collinearity affects the stepwise procedure, the ridge regression is applied to all six variables and not just to those selected in our last model. They are the growth rates of the three age groups and their interactions with time.

For our initial testing, the chosen constants (K) are 0.0 and 0.1. When the ridge constant is 0, the results are the same as from the usual regression. Yet, ridge regression utilizes forced entry, which includes all variables meeting a preset criterion in the equation. When K = 0, all six variables except the 45-64 age group are significant. The R^2

is 0.98, but the condition number is 202. The latter is an index of collinearity. If it is between 10-30, then multicollinearity is minor or moderate. The higher the index, the more serious is collinearity. Our current situation is obviously not good.

When k is changed to 0.1, the condition number declines to 27.8. Although collinearity is still manifest, it is clearly alleviated. Also, the 45-64 age group, which had positive coefficients before, is now negative. Sign change is one of the symbols that denotes a collinear predictor variable (Gunst and Mason 1980). By adding the ridge constant, however, R^2 is reduced to 0.96, and the standard error increases from 0.12 to 0.17.

Although collinearity has now become moderate, the high standard error seems hard to tolerate. Therefore, it is decided not to introduce an even higher ridge constant. Instead, a small interval of increment between 0 and 0.1 is used to pinpoint the best constant that produces the acceptable parameter estimates and standard error. With 0.02 as the increment, we found that most regression coefficients stabilize when K is 0.08.

Settling down at this level, the condition number is 36.0. The standard error is 0.16, and R^2 is 0.97. We are now accepting slightly higher collinearity in exchange for lower standard error. This is comparatively more important for our prediction purposes. The final ridge regression results are reported in Table 6.9. Ridge regression has one additional

advantage of which we can make use to obtain the best equation.

The standardized coefficients can be plotted against their corresponding ridge constants in a diagram noted as the ridge trace. This plotting technique provides a means to delete the variables that are unstable or moving towards zero as K increases (Gunst and Mason 1980; Chatterjee and Price 1977). From the ridge trace in Fig. 6.6, the growth rate of 45-64 and its interaction with time have the most drastic changes with K. They are eliminated from the final equation.

Also removed is the interaction of time with the 65 or above group. This coefficient has quite substantial changes and a tendency to vary its directions of movement when K increases. As a result, the final equation contains only three variables. The coefficients are the values retained from Table 6.9. No additional ridge regression is necessary for the selected variables (Chatterjee and Price 1977). The final equation for female age effect is as follows:

$$\begin{aligned}\text{Ln (Female Age Effect)} = {} & 1.80 - 0.129\ (\text{P.A.G. 25-44}) \\ & + 0.359\ (\text{Time})\ (\text{P.A.G. 25-44}) \\ & + 0.355\ (\text{P.A.G. 65+})\end{aligned}$$

where P.A.G. stands for percentage of annual population growth, and Time represents the years after 1964.

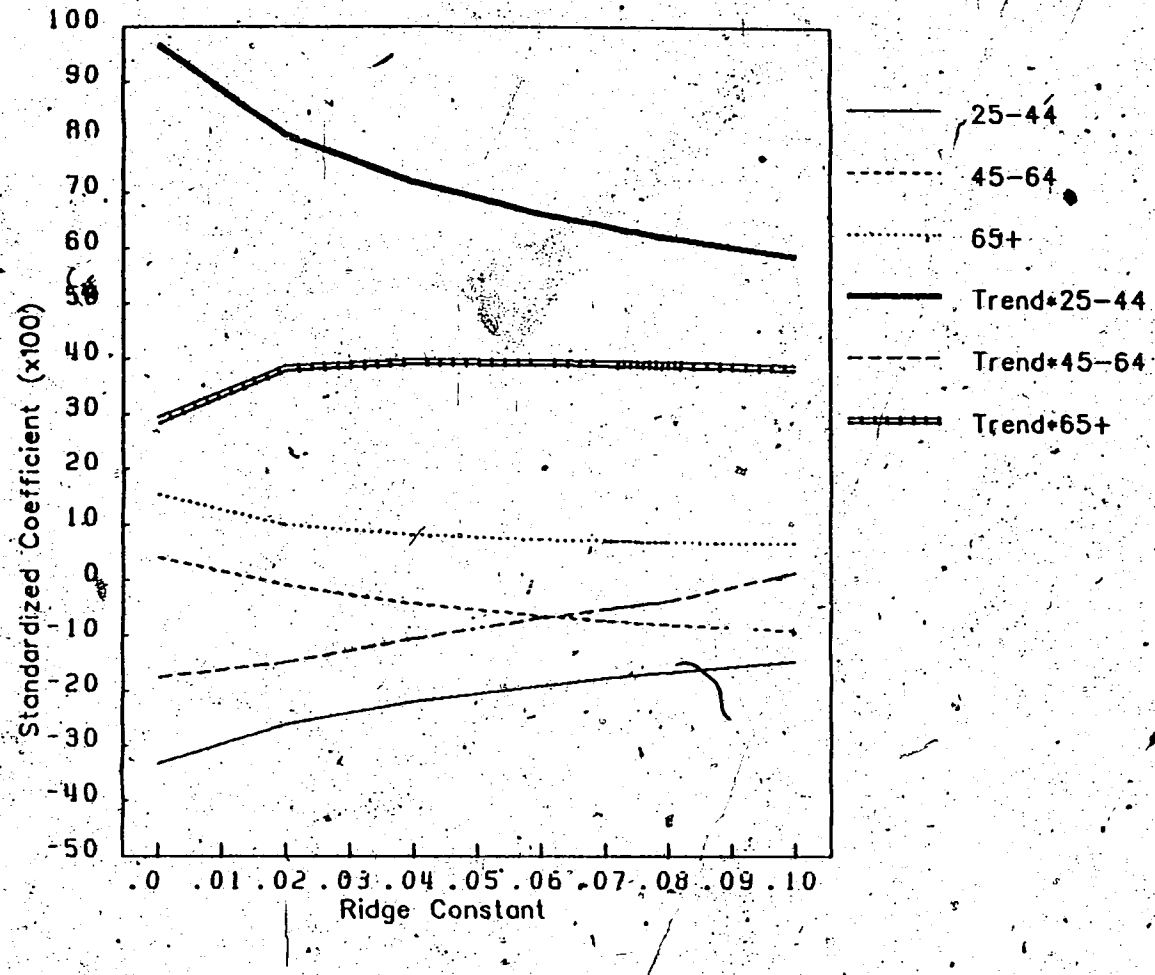
Table 6.9 Female Age Effect, Final Ridge Regression Model, Canada, 1950-1981

	Coefficient	Standard Error
Constant	1.8003	
25-44	-0.1296	0.025
45-64	-0.0911	0.043
65+	0.3547	0.158
25-44 (T)	0.3585	0.020
45-64 (T)	-0.0228	0.034
65+ (T)	0.3821	0.039
R^2 (adj.)	0.9651	
Standard Error	0.1605	
Condition Number	33.45	
Number of Cases	31	

Note: T stands for years after 1964 inclusive. Since the ridge regression program does not produce T statistics, the standard error of the coefficients are given. Significant coefficient should be at least twice the size of its standard error.

Source: From ridge regression analysis

Fig. 6.6 Ridge Trace of the Variables in Female Age Effect, Canada, 1950-1981



Source: Ridge regression analysis.

6.4 Male Cancer Force

Fitting the model for cancer force begins with simple bivariate regression. The only independent variable in the equation is the number of years measured from 1956. However, as the model develops, multivariate analysis is necessary. The initial inspection of the distribution of male cancer force shows normality. For the time variable, as each observation can not occur more than once, the distribution is therefore a straight upward sloping line. Transformations is not required for either one of these two variables.

The correlation between time and cancer force is very high, about 0.987. Such a positive relationship is encouraging since cancer force is hypothesized to rise as time progresses from 1956. That is, whether carcinogenic exposures were before or after 1956, the impacts will be manifest and more serious as time advances. This complies with the notion of a long latency period between contact and fatality.

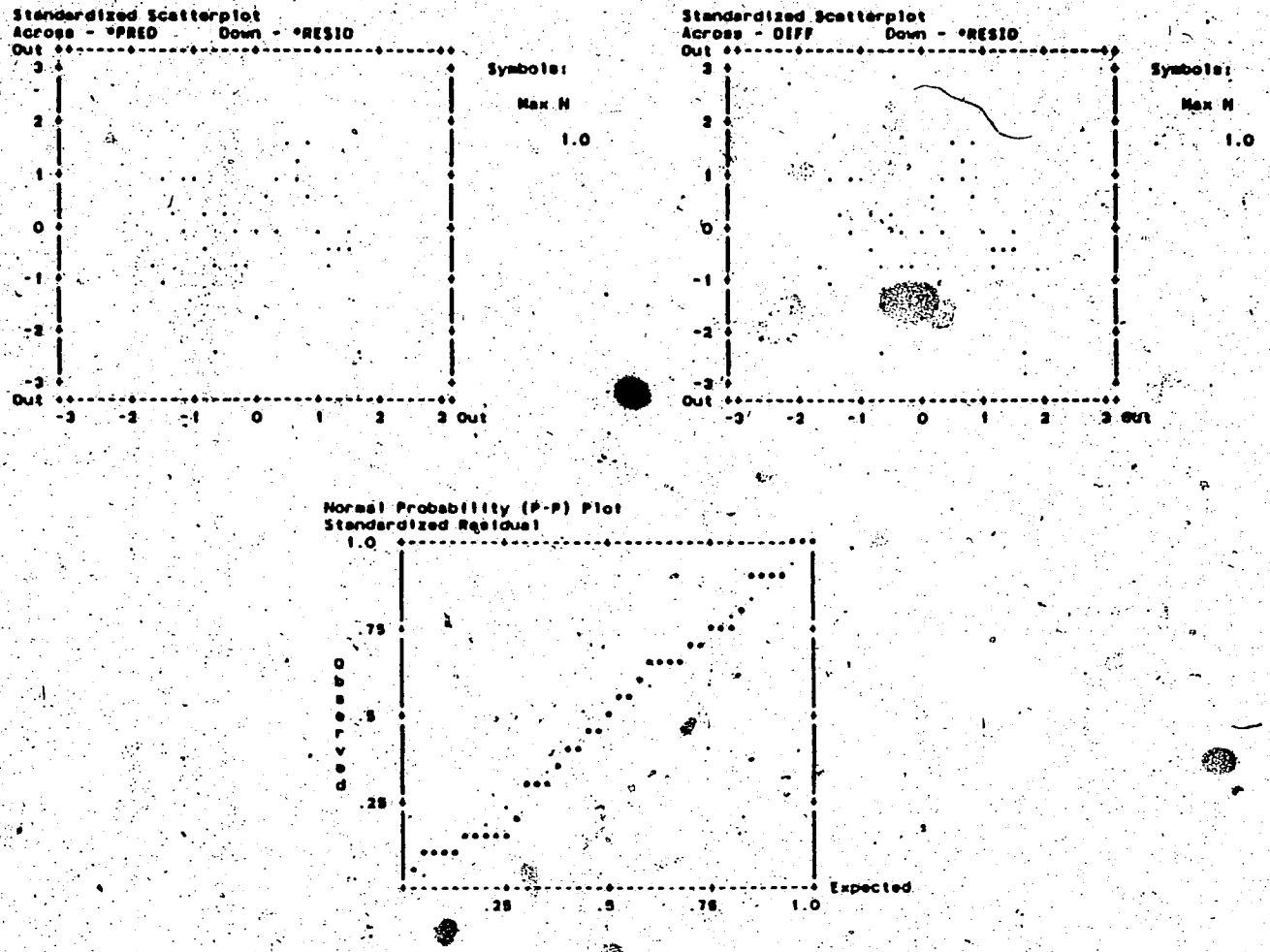
The bivariate results in Table 6.10 seem to be promising at first glance. A R^2 of 0.97 indicates that only about three per cent of the variations in cancer force could not be explained by changes in time. Ignoring some moderate fluctuations, the normal probability plot in Fig. 6.7 is almost a straight line. The model seems satisfactory despite the low D-W statistic of 0.897, which suggests a problem of autocorrelation. A minimum of 1.50 is necessary in order to be free of autocorrelation.

Table 6.10 Male Cancer Force, Initial Regression Model, Canada, 1950-1981

	Coefficient	T Value
Constant	-1.9222	
Time	1.3609	36.831
R^2 (adj.)	0.9736	
Standard Error	2.1010	
D-W Statistics	0.8977	
Number of Cases	32	

Source: From regression analysis

Fig. 6.7 Residual and Normal Probability Plots of Male Cancer Force, Initial Regression Model, Canada, 1950-1981



Source: Regression analysis.

If we focus on the residual plot in Fig. 6.7, we can see not only outliers, but also two main clusters. The latter indicate that some trends are impeded within cancer force. In the casewise plot in Appendix 8, the residuals can be more easily distinguished by three clusters: 1950-1964, 1965-1973, and 1974-1981. From studentized residuals provided with the diagram, 1959 and 1981 are obviously the outlying points. Evidence indicates that the present model is far from satisfactory.

The modification begins with the addition of two dummy trend indicators. The first variable takes on the value of 1 if the years are between 1965 and 1973, and is 0 otherwise. The second variable represents the years of 1974 and later with a value of 1, and 0 for otherwise. A third dummy variable for 1950-1964 is not necessary. Exact collinearity would be the consequence if it were added to the equation. As before, these two dummy variables are each multiplied with the time variable to test for their influences on the slope (coefficient of the time variable). Multiple regression is then performed on this new set of variables.

The new model reveals great improvements. The D-W statistic increases to 1.15, and the standard error is down from the previous 2.10 to 1.05. Nevertheless, the two outliers of 1959 and 1981 still prevail. When they are tested with dummy variables, they are found to be valid outliers. As they are deleted for a new regression, 1950, 1951 and 1965 emerge with large studentized residuals. These

three potential outliers are again tested, individually and collectively, using dummy variables. They are all proven highly influential and subsequently eliminated.

Multiple regression is then performed on the remaining 27 cases. The model now consists of the original time variable, the two trend indicators and their interactions. The results in Table 6.11 show that R^2 for our new model has been increased slightly to 0.996, and the standard error is reduced to 0.87. For the latter, the improvement is substantial.

Furthermore, the present D-W statistic of 1.92 is well-above the required 1.08 to free the model from autocorrelation. The residuals in the casewise plot, as well as when plotted against the predicted values, reveal no distinguishable pattern or trend. The normal probability plot is also straightened but still has some fluctuations. Since no more outliers and abnormalities are present, the model is then finalized. These three residual and casewise plots are provided in Fig. 6.8 and Appendix 9, respectively.

With regard to the regression coefficients, only four of the five are significant. The interaction of the post-1974 period is excluded from the equation. Even so, the significance of the trend indicators shows that the male cancer force between 1951-1981 involves different patterns of movements. Despite the addition of trend indicators and their interaction terms, the original time variable persists in being the most important variable with the largest impact

Table 6.11 Male Cancer Force, Final Regression Model, Canada, 1950-1981

	Coefficient	T Statistics
Constant	-1.2422	
Time	1.1307	19.220
T1	-5.0757	-2.794
Time (T1)	0.7002	4.870
T2	3.8829	3.524
R^2 (adj.)	0.9957	
Standard Error	0.8504	
D-W Statistics	1.9165	
Number of Cases	27	

Note: T1 stands for years prior 1965-1973
T2 Stands for years after 1974 inclusive

Source: From regression analysis

Fig. 6.8 Residual and Normal Probability Plots of Male Cancer Force, Final Regression Model, Canada, 1950-1981

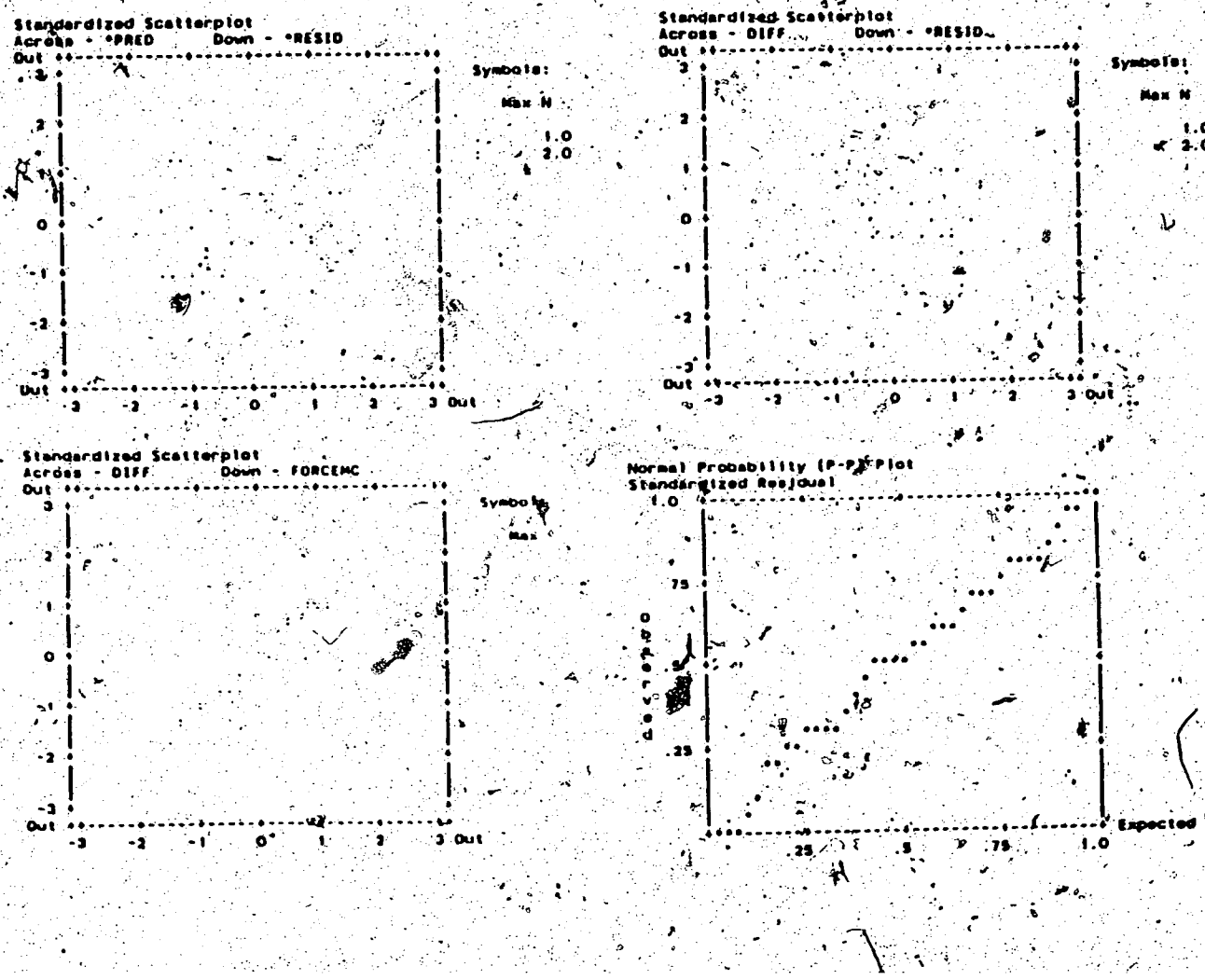
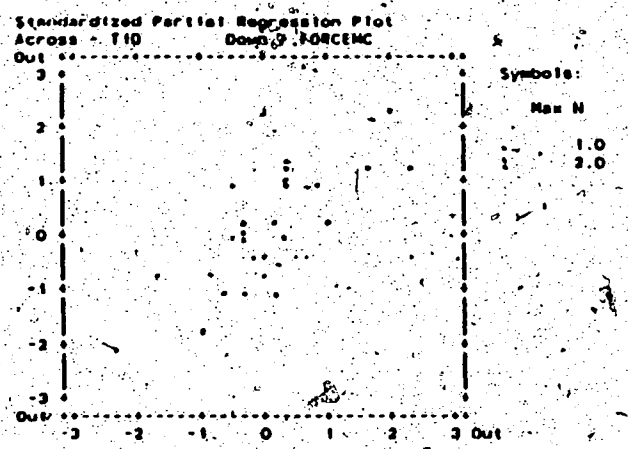
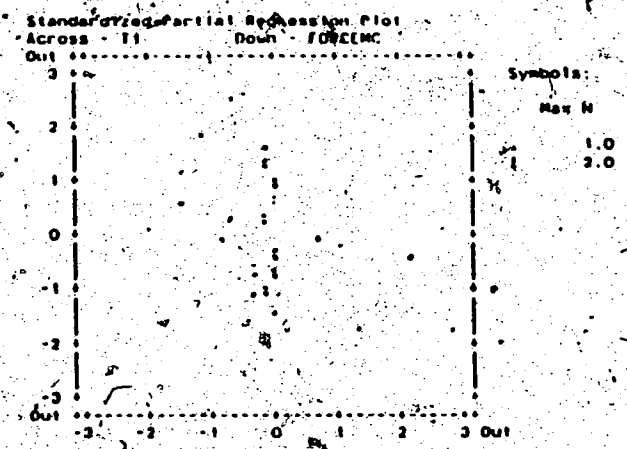
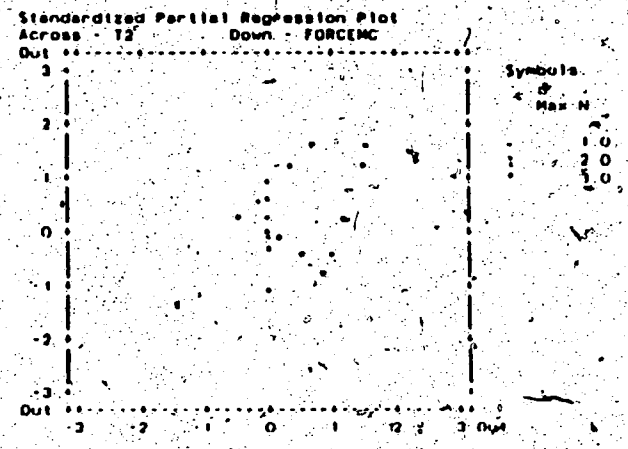
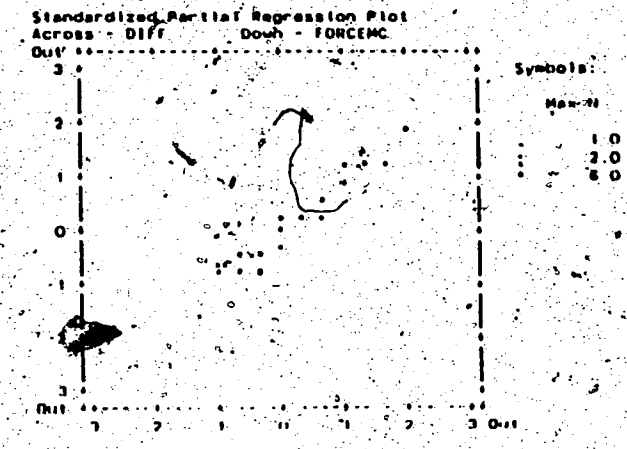


Fig. 6.8 (continue)









Source: Regression analysis.

on male cancer force. The final equation is as follow:

$$\text{Male Cancer Force} = -1.242 + 1.131\ \text{Time} - 5.076\ T1 + 0.70\ T1\ (\text{Time}) + 3.88\ T2$$

For predicting cancer force after 1974, the equation becomes:

$$\text{Male Cancer Force} = -1.242 + 1.131\ \text{Time} + 3.88\ T2$$

where T2 stands for years after 1974.

6.5 Female Cancer Force

Fitting the model for female cancer force is more time consuming than for males. Regressions must be repeated in order to test for time trends that are known to exists but never apparent enough for exact location. The model employs untransformed data since their distributions reveal no harmful non-normality.

The correlation between cancer force and the time variable is very high, -0.945. The negative sign is as anticipated, because female cancer force in general has been declining in the last three decades. Bivariate regression shows that the time variable accounts for about 89 per cent of the variations in female cancer force. The regression results are given in Table 6.12.

Table 6.12 Female Cancer Force, Initial Regression Model, Canada, 1950-1981

	Coefficient	T Value
Constant	-0.0378	
Time	-0.4444	-15.860
R^2(adj.)	0.8899	
Standard Error	1.4634	
D-W Statistics	1.5654	
Number of Cases	32	

Source: From regression analysis

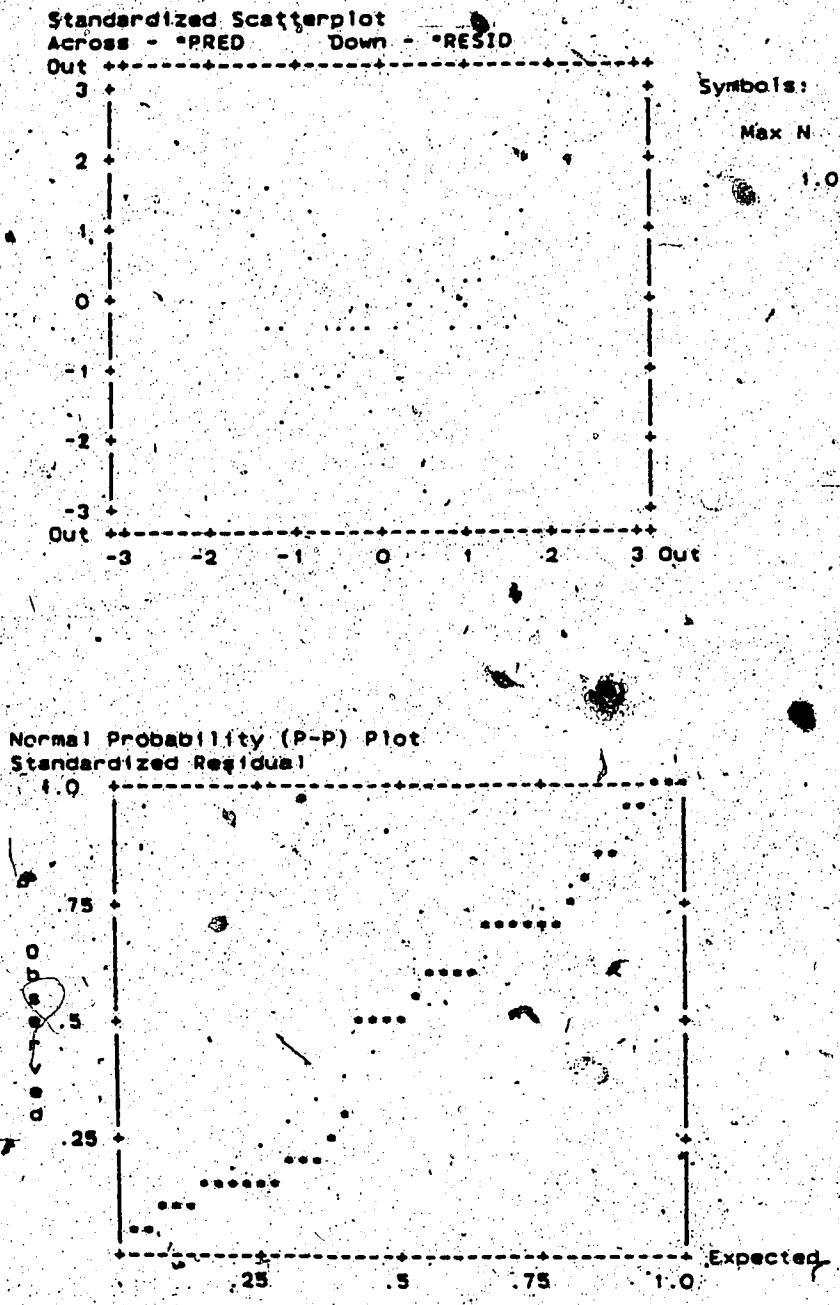
When the residual statistics in Appendix 10 are check, only 1961 and 1966 are found to be possible outliers. Their studentized residulas were larger than preferred. Despite the high R^2, the normal probability plot in Fig. 6.9 suggests that the model is not properly specified. There are systematic variations in the residuals layout. Also, the casewise plot in Appendix 10 shows that there might be two or three latent trends.

Taking 1970 as the cutting point, residuals thereafter appear with greater variations than before. Heteroscedasticity is likely to be a problem. If 1964-1970 is considered as the central period with comparatively small residuals, then three patterns can be distinguished. Regardless of the trends, however, the D-W statistic of 1.56 expresses no specific threat of autocorrelation.

Modification of the model is necessary. The main concern is to specify the trends, which are not as manifest as those among the male cancer force. Regressions are therefore utilized on a trial and error basis to test for different trend specifications. Initially, the trends of 1964-1969 and 1970-1981 are tested with dummy variables in the usual manner, while 1950-1963 is made the reference point. This model specification, disappointedly, provides few changes. The normal plot reveals almost no improvements. New specifications must be tried.

In our subsequent attempt, using 1975 as the cutting point, not much improvement occurred either. More careful

Fig. 6.9 Residual and Normal Probability Plots of Female Cancer Force, Initial Regression Model, Canada, 1950-1981



Source: Regression analysis.

examination of the original casewise plot led to the specification of a trend indicator with 1971 as the center point. The results indicate some breakthroughs. When the outliers of 1961 and 1966 are deleted, each of the four years from 1975 to 1978 appears consecutively as the outliers.

If all six outliers were deleted, there were substantial improvements in the model. Yet, the need to remove six data points is rather uncomfortable. The sequential appearances of 1975-1978 suggest that they should not be treated as outliers but rather as a short-term fluctuation. For such a possibility, three time trends are now designated and tested with two dummy variables. For the two periods of 1951-1971 and 1975-1978, they are represented with indicators with a value of 1.0. The remaining years are made the reference point.

From the new regression, the years of 1961, 1966, and 1971 are found to have large studentized residuals. Subsequent testing led to their removal. The results with these three cases removed are summarized Table 6.13. The stepwise procedure has excluded the two trend indicator, but retained their interactions. In other words, the trends affect only the slopes and not the intercept.

The three significant variables account for some 98 per cent of variations in female cancer force. The normal probability plot in Fig. 6.10 reflects clearly a much better fit, despite the slight increase in standard error to 0.64.

The casewise plot in Appendix 11 reveals no specific patterns as it did before.

Although the studentized residuals for 1952 and 1976 are quite large, they are not eliminated as their dummy variable test did not provide further positive changes in the fit of the present model. As a matter of fact, these two outlying points appear as the extensions of the main cluster and should therefore, be considered in the model as well. Since there are no more modifications, the final equation for female cancer force is as follows:

$$\text{Female Cancer Force} = 0.3879 - 0.124\ T1\ (\text{Time}) - 0.37\ T2\ (\text{Time}) - 0.388\ \text{Time}$$

where T1 and T2 represents the uears from 1951 to 1971 and from 1975 to 1978, represectively. For estimating cancer force after 1981, the equation is simply:

$$\text{Female Cancer Force} = 0.3879 - 0.388\ \text{Time}$$

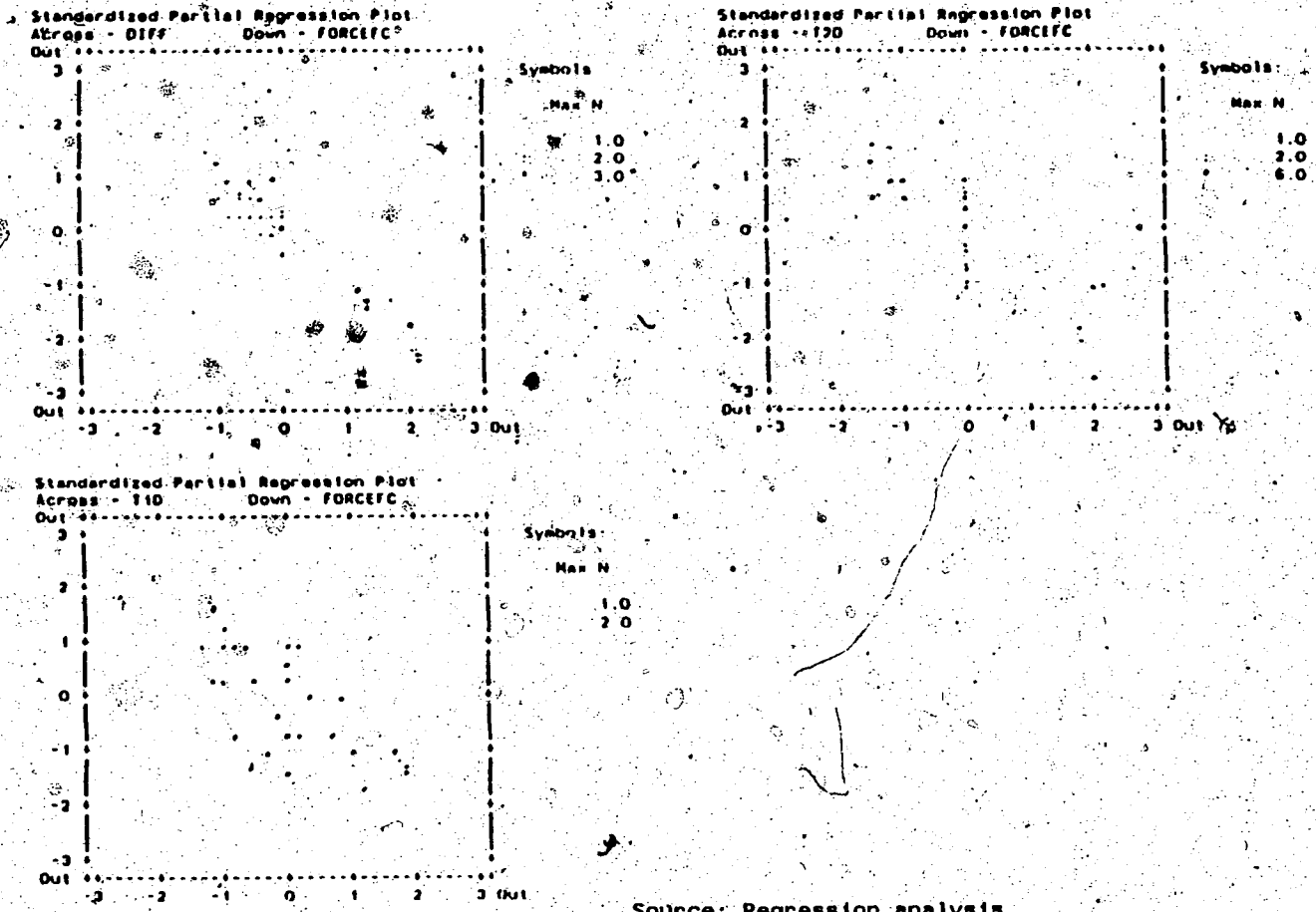
Table 6.13 Female Cancer Force, Re-specified Regression model, Canada, 1950-1981

	Coefficient	T Statistics
Constant	0.3879	
Time	-0.3854	-26.036
Time (T1)	-0.1238	-5.125
Time (T2)	-0.1366	-6.862
R^2 (adj.)	0.9800	
Standard Error	0.6364	
D-W Statistics	2.043	
Number of Cases	29	

Note: T1 stands for years prior to 1970
T2 Stands for years 1975-1978

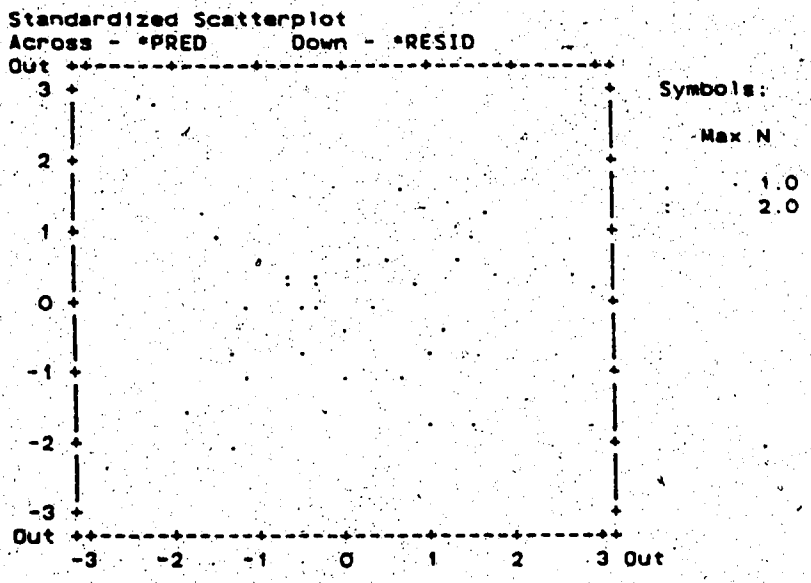
Source: From regression analysis

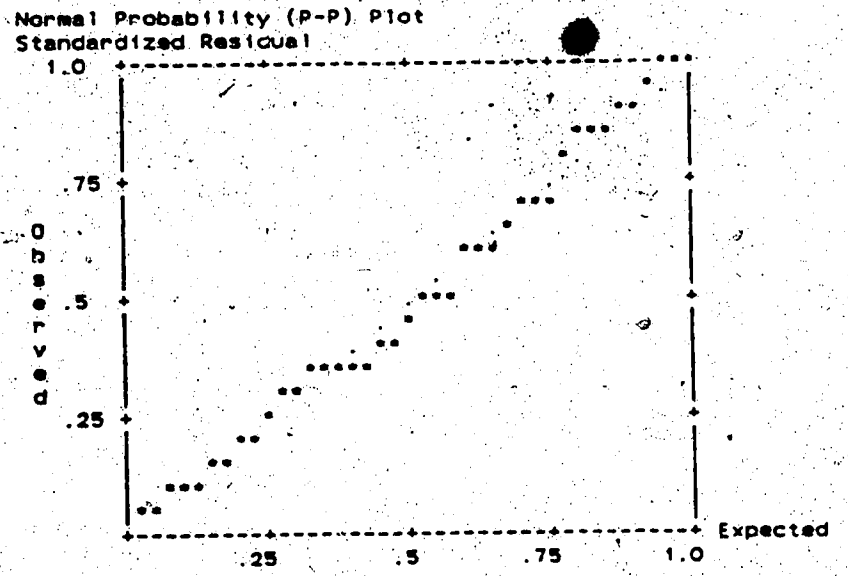
Fig. 6.10 Residual and Normal Probability Plots of Female Cancer Force, Final Regression Model, Canada, 1950-1981



Source: Regression analysis.

Fig. 6.10 (coninued)





6.6 Summary of the Statistical Models

To compare male and female age effects and cancer force, the standardized coefficients are provided in Table 6.14. For all four equations, R^2 is very high. Nonetheless, such high values seem not to be very well matched with the moderately rugged normal probability plots. In view of the trend factors involved, more detailed time series analysis techniques would be able to handle the data problems more efficiently and intricately.

In spite of these shortcomings, regression analysis still provides us with useful prediction equations. In general, the age effect has much lower standard errors of estimate than in the case of cancer force. Yet, it is the latter that has smoother normal plots. The coefficients are not of the type one would expect from the age pattern of cancer risk. The oldest male age group has not been included in the equation.

For both sexes, the 25-44 age group displays the largest impact on age effect. For females, this age group has a negative effect if the entire time series is examined as a whole. Positive impacts are found for the years after 1964 only. Such changes are well supported by the upswing in female age effect since 1958, shown in Fig. 5.22. The differences in the trend indicators, 1976 for males and 1964 for females, illustrate perhaps different cohort effects. Again, the time series technique can explore this aspect in greater detail.

Table 6.14 Comparison of Male and Female Age Effect and Cancer Force Regression Results, Canada, 1950-1981

Coefficients	Age Effect	
	Male	Female
P.A.G 25-44	0.456	-0.167
P.A.G 45-64	-0.366	---
P.A.G 65+	---	0.069
Male Trend (1976 on)	0.388	---
Female Trend (1964 on)	---	---
Trend (25-44)	---	0.621
Trend (65+)	---	---
Standard error of Estimate	0.122	0.161
	Cancer Force	
Time	0.824	-0.800
Male Trend (1974 on)	0.146	---
Standard Error of Estimate	0.850	0.636

Source: From multiple and ridge regression.

6.7 Male and Female Cancer Mortality, 1982-2003

With the equations for age effects and cancer force established for males and females, respectively, cancer mortality rates can be predicted. The major limitation of these predictions, however, is that they could not foresee fluctuations that might occur in the future. For them to be valid or reliable, we first have to assume that the trends of cancer force during the past thirty years will either persist or continue with only gradual changes in the upcoming decades.

Secondly, we have to assume that there will be no significant changes in the population size. This assumption is essential for the crude death rates. Any large influx of young immigrants, for example, could reduce the death rates and vise versa. With the exception of migration, our assumptions perhaps, are not easy to defend. Yet, recent progress in medical treatment, and the willingness of individuals to modify their behaviors do not pose an immediate threat to the reliability of our estimates.

The predictions are made in two stages. Age effects and cancer force are first estimate, and the crude and standard death rates computed using equations (4-6) and (4-7). The 1956 base rates used in our estimates are 139.5 and 119.8 for males and females, respectively. It should be noted that the estimated standardized rates are based on the 1956 age structure. The estimated age effects, the cancer force, and the crude and standardized death rates are given in Appendix

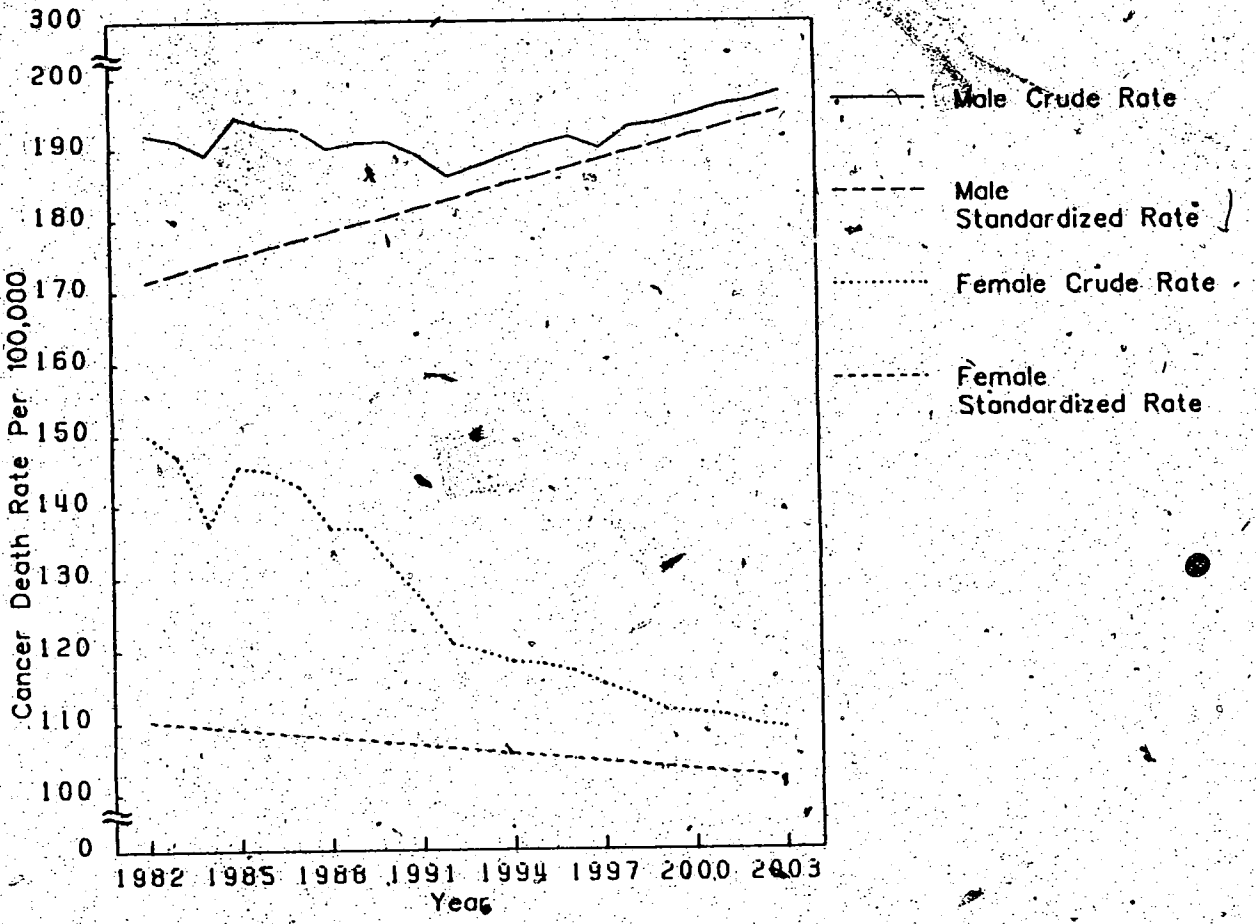
12. The two death rates are plotted in Fig. 6.11, and the predicted age effect and cancer force are shown in Fig. 6.12.

The estimates show that male age effect will decline, while cancer force will continue to grow in the coming 20 years or so. For females, both components will decrease. The declining age effects are as expected, to some extent, since fertility has subsided since the 1960's. As a result, the supply of risk population and its growths are diminishing over time. Cancer mortality, therefore, will draw on a smaller pool of victims.

The downward trend in female cancer force suggests an absence of influence by the rising lung cancer death rate. Perhaps mortality from this recently-deteriorating cancer site is still relatively minor and its effect has been suppressed by the reductions in cancer force from other sites. Another possible explanation is the long latency period which could have prevented lung cancer from showing its impact at the present stage. If this is the case, then female cancer force could rise substantially once the current young cohort reaches late adulthood.

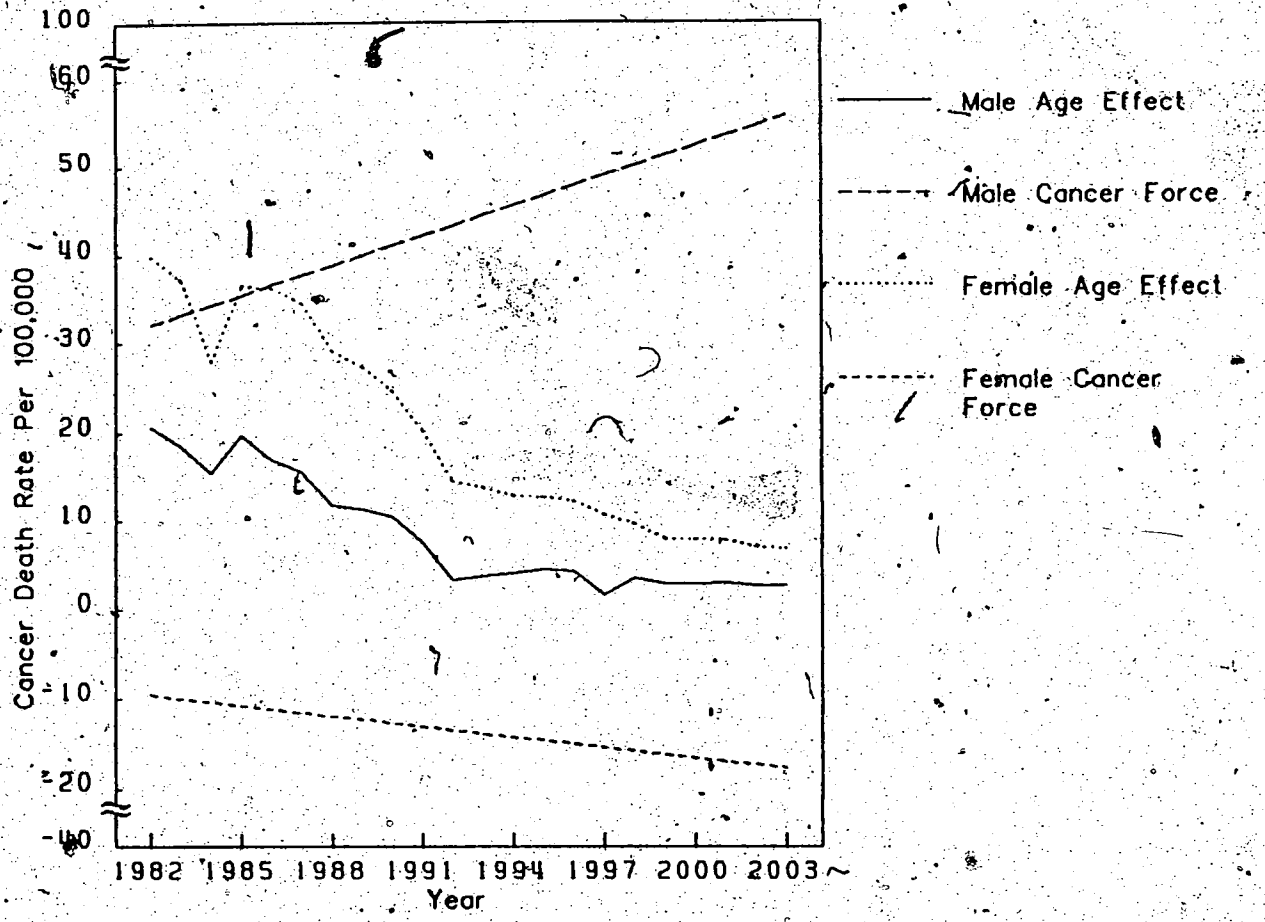
As can be deduced from the above age effect and cancer force components, the estimated male and female cancer death rates are necessarily moving in opposite directions. However, the trend in male crude cancer mortality rates is increasing curvilinearly. When the estimates for the two sexes are contrasted, males outperform the females.

Fig. 6.11 Predicted Crude and Standardized Cancer Death Rates by Sex, Canada; 1982-2003



Source: Regression analysis.

Fig. 6.12 Predicted Age Effect and Cancer Force in Cancer Mortality by Sex, Canada, 1982-2003



Source: Regression analysis.

For example, male and female crude cancer death rates registered in 1982 are 192.1 and 149.0, respectively. From our equations, the corresponding estimates are 192.13 and 150.0, respectively. While the figure for male is exactly as reported, that for female is off the target by only one death per 100,000 population. For 1983 and 1984, the reported rates are 192.1 for males and 148.4 for females. They are still more or less contained within a 95 per cent confidence interval. Even when they exceed the given range, the difference is minimal.

Considering that the differences between the estimates and the reported figures come to only one or two deaths per 100,000 population, the predictions seem tolerable. Using the lower boundary of the interval, we can gain some idea of the minimum cancer death rates to be expected for the coming decades. Given the fact of reduced fertility, and a subsequently smaller work force, the increasingly heavy burden of providing adequate future health care facilities for cancer patients is not hard to imagine. The present results are useful in health care planning.

7. Socioeconomic Analysis of Cancer Mortality

7.1 Introduction

We may recall from our earlier review of literature that cancer mortality is not homogeneous across all population subgroups. Differentials exist between the young and the old and between males and females. Studies also indicate that individuals of different ethnic groups or occupations are not equally susceptible to the same types of cancer.

Research on socioeconomic classes commonly unveils an inverse relationship with cancer mortality. Such an association persists, more often than not, regardless of how social class is measured (by income, or education, or occupation status, or their combinations). Exceptions emerge, however, when age, sex, or ethnicity are being taken into consideration. The results also change when cancer by sites is examined.

Putting these findings together, it [illegible] obvious that cancer mortality and differentials alike, dwell within a complex socioeconomic framework. Not only does the individual cancer site react unequally to the socioeconomic environment, but their response may be further affected by many demographic characteristics as, for example, gender. As a result, factors that are found supportive of high cancer death rates among a specific subgroup may not affect other groups, even if the conditions are similar. Likewise, some

factors can be more influential, or some become active, only under the pressure of other elements.

Under these circumstances, mere comparisons of the mortality experience of some population subgroups with the general public may not be sufficient to identify the actual factors causing high cancer death rates or the differentials. It is therefore necessary to assess simultaneously as many relevant factors as possible. By doing so, the real contributions of each cancer inducing factors in the presence of others can be best determined. The following study attempts to examine the effect of four groups of selected factors on cancer mortality, namely, socioeconomic level, ethnicity, age, and occupation.

7.2 Summary of Regression Procedures

The analysis is carried out for males and females separately using regression procedures. Besides total cancer, four specific cancer sites, stomach, intestines, bladder, and lung, are chosen for the present study. In the case of females, cancer of the breast is also analysed. Death rates per 100,000 population from the above cancer sites are the dependent variables for the 11 regression equations involved in our model fitting. Due to this rather large number of equations, it has been decided not to describe the lengthy process by which each equation arrives at its final stage. Instead, the measures taken to derive the best models are summarized here.

The analysis begin with 20 basic explanatory variables for males and 22 variables for females. These variables have been described in Chapter 4.4. Prior to statistical analysis, the variables with skewed distributions are transformed to obtain normality. Excluded from the transformations, however, are female Asians and Scandinavians. Their skewed distributions cannot be normalized substantially through transformations. These two variables are, therefore, each replaced by a dummy variable. A value of 1 will stand for the presence of these ethnic groups in a particular Census Division and 0 otherwise.

When correlations are computed, further modifications of some variables are found required. First, percentage of the French and the English population is highly but negatively correlated ($r = -0.815$). In other words, the English population decreases as the percentage of French rises. This is due to the opposing patterns of concentration of these two major ethnic groups in Canada.

In terms of variable selection, to include both ethnic groups in the equations introduces redundancy. The effect of either ethnic group can be captured by the sign of the coefficient of the other group selected for the analysis. Consequently, only the percentage of French is retained. Its selection is due to the well-known high cancer death rate among the French.

A similar situation also occurs between the primary industry and the other industrial variables. Their negative

correlations reflect the opposing developments of the agricultural and the industrial or financial sectors. Therefore, the primary industry is excluded from the analysis, despite its fairly high negative correlation with overall cancer mortality.

There are also high correlations between the industries of health and welfare, education, and personal service, as well as between finance and trade. In order to minimize collinearity, the first three categories are collapsed into a single category of communication and the latter two into commerce. These groupings are also employed in the Census reports.

The above changes reduce the number of basic variables from 20 to 14 for males and from 22 to 16 for females. At this stage, regression analysis could have proceeded. However, cancer inducing factors tend not to operate by themselves alone. Possible interactions between the variables must be introduced as well. In our present situation, it is neither possible nor practical to study all the interactions among the 14 independent variables.

To find the most likely interactions, the Census Divisions are categorized into high and low levels of income, education, and manufacturing which includes also the medium level. Only these variables are selected because of the findings of their association with high cancer mortality or differentials.

On the other, as interactions (first order) are symmetric (Fox 1984), it is not necessary to select additional variables like age and repeatedly examine how its effect changes with level of manufacturing, for example. To test regional variations, Census Divisions are grouped into the Atlantic Provinces (P.E.I., New Brunswick, Nova Scotia), Ontario, Quebec, the Prairie Provinces (Alberta, Saskatchewan, Manitoba), and British Columbia.

Regression is then carried out for each of these categories. The dependent variables employed in these tests are male and female total cancer mortality. The idea here is to see how the explanatory variables alter their magnitudes or even their signs when different controls are introduced.

Aside from the interactions, a noteworthy result of the tests is the correlation between high level of income and education. The new coefficients of 0.536 and 0.343 for males and females, respectively, are lower than the 0.690 and 0.430 of the original variables. To reduce collinearity, the dummy variables of high income and education level areas are adopted instead. The low level areas are kept as reference groups.

The interaction tests also reveal that levels of manufacturing labour force change the size of their effects as other controlling factors are introduced, and vice versa. It is therefore decided to replace the original variable (percentage of manufacturing work force) with the high and low level of manufacturing areas. The medium group is left

as the reference group.

We also observe that the regions in Canada should be distinguished, or even tested, as independent variables. However, the adding of four regional dummy variables (for Quebec, Ontario, the Prairie Provinces, and British Columbia) renders the correlation matrix singular. Under this condition, the determinant of the matrix cannot be solved and no parameter estimates in the regression equation can be computed. Therefore, regional effect is tested in a different manner.

These four regional indicators are each combined with mining, the most versatile variable under regional groupings, to form interactions. In subsequent regression analysis, the Prairie Provinces are joined by British Columbia to form the Western Provinces for their similiar influences on cancer mortality.

In the case of females, percentage of labour force engaged in the mining and construction industry are found to have almost no impact on cancer mortality. These two variables are therefore removed from our model to reduce redundancy. It is necessary to remove all ineffective variables before best possible subset regression is applied.

The reason for doing so being that the Mallow's Cp statistic in subset regression is affected by the variance of the complete model with all variables. The latter can be inflated by the inclusion of unnecessary variables (Gunst and Mason 1980; Daniel and Wood 1977; Draper and Smith

1981). For example, including mining interactions for the Prairie Provinces and British Columbia separately in the equation could unnecessarly inflate the variance of the full model, and thereby introduce biase to the subsets of equations.

The final pools of variables ready for the best subset regression, for males and females respectively, are given in Tables 7.1 and 7-2. A total of 26 and 29 variables are involved in the regression equations for males and females, respectively. The dummy variables in the Tables are indicated with an asterisk.

For all interaction terms, the two involved variables are first centerized. That is, their means are removed. This step is taken to ensure that the cross-products will be less collinear with the original linear variables (Gunst and Mason 1980; Cohen and Cohen 1983). It is necessary to point out that the interactions are derived with total cancer mortality as the dependent variable. Yet, they are used for all cancer sites selected in this study.

Ideally, interactions should be designated individually for the cancer sites. Nevertheless, using different sets of variables makes comparisons across the various cancer sites very difficult, because variables are selected not only on their own merits, but also on the merits of the accompanying variables (Mosteller and Tuky 1977). Consequently, the more time-consuming process of setting up interactions for each

Table 7.1 Variables for the Socioeconomic Analysis of Male Cancer Mortality by Selected Sites

Basic Variables	Interaction Terms With
% of 25-44 (1)	(4), (7), (8), (9), (10)
% of 45-64 (2)	(7)
% of 65+ (3)	(7)
% French (4)	(7), (8)
% Asian (5)	N/A
% Scandinavian (6)	N/A
High Income (7)	See above
High Education (8)	See above
High Manufacturing (9)	See above
Low Manufacturing (10)	See above
Construction (11)	N/A
Communication (12)	N/A
Commerce (13)	N/A
Mining (14)	Quebec, Ontario, West (the Prairie Provinces and British Columbia)

Note: For mining, the Atlantic Provinces are left as the reference group.
The total number of variables is 26.

Table 7.2 Variables for the Socioeconomic Analysis of Female Cancer Mortality by Selected Sites

Basaic Variables		Interaction Terms With
% of 25-44	(1)	(4), (7), (8), (9), (10)
% of 45-64	(2)	(7)
% of 65+	(3)	(7)
% French	(4)	(3), (7), (8), (9), (10)
Asian	(5)	N/A
Scandinavian	(6)	N/A
High Income	(7)	See above
High Education	(8)	See above
High Manufacturing	(9)	See above
Low Manufacturing	(10)	See above
Communication	(12)	N/A
Commerce	(13)	N/A
Child-Woman Ratio	(14)	N/A
Number of Children	(15)	(7), (8)

Note: Child-Woman Ratio (14) is the number of children less than 4 years of age to the number of women aged 25-44.
Number of children (15) is the average number of children under 24 years of age and staying at home.
There is a total of 29 variables.

cancer site is left for later analysis.

In the following discussion, regression results are based on the best subset equations selected for their low Cp statistics and high coefficient of determination. To arrive at these equations, data points with large Mahalanobis distance or Cook's D (Fox 1984) are tested for removal.

Data points will be treated as valid outliers and eliminated from the analysis only when that results in better fit of the model either by increasing the R^2 or reducing the standard error of estimate. For this analysis, normal probability plots are not available from the statistical package for best subset analysis.

As the R^2 are relatively low, which is typical for socioeconomic studies, we do not expect that the normal plots, if available would indicate any smooth patterns, primarily because we have not included all possible socioeconomic variables that could affect cancer mortality.

7.3 Male Regression Results

In Table 7.3, the best subsets of variables selected for the the different cancer sites are provided along with the corresponding summary statistics. For comparison, the value of R^2 and standard error of estimate without case deletions are also given. The variables listed are based on the largest subset of total cancer mortality, which includes 13 variables. Variables that are not chosen for a particular cancer site are left blank in the Table.

Table 7.3 Best-Possible Subset Equations for Male Cancer Mortality by Selected Sites, Canada, 1976

	All Cancer	Stomach	Intestine	Bladder	Lung
Intercept	8.04	7.86	3.40	3.13	0.93
25-44	----	-0.82 [e]	----	----	----
45-64	0.38 [e]	----	-0.14 [a]	0.33 [d]	0.68 [e]
65+	-0.32 [d]	-0.55 [e]	----	-0.34 [e]	-0.40 [e]
Scand.	-0.15 [a]	0.16 [a]	-0.32 [e]	-0.13 [+]	-0.22 [c]
French	----	0.22 [c]	-0.38 [e]	-0.28 [d]	-0.25 [e]
French (25-44)	0.16 [a]	----	0.32 [e]	----	----
High Income	5.71 [a]	-1.48 [*]	----	0.65 [a]	6.47 [e]
Hi. Income (25-44)	----	----	----	----	0.21 [c]
Hi. Income (45-64)	----	----	----	0.21 [d]	----
Hi. Income (French)	0.19 [b]	----	----	0.21 [c]	----
Hi. Ed.	----	-1.74 [a]	----	----	-2.20 [f]
Hi. Ed. (25-44)	0.12 [a]	0.13 [*]	0.12 [a]	----	----
Hi. Ed. (French)	----	----	----	----	0.15 [a]
Hi. Manuf.	8.07 [c]	----	2.34 [e]	----	----
Lo. Manuf.	----	1.88 [a]	----	----	-3.14 [c]
Lo. Manuf. (25-44)	0.11 [a]	----	0.18 [c]	----	0.11 [f]
Construction	----	0.11 [a]	----	-0.09 [*]	----
Communication	----	----	----	-0.11 [a]	----
Mining	----	----	-0.13 [a]	----	----
Ont. Mine.	0.08 [a]	----	----	-0.18 [d]	0.19 [e]
West. Mine.	-0.22 [e]	-0.12 [a]	----	-0.17 [c]	-0.08 [f]
Finance-Trade	0.07 [*]	----	0.20 [e]	----	----
Bladder	N/A	N/A	N/A	-6.60	N/A
R	0.58	0.46	0.38	0.37	0.46
R (adj.)	0.56	0.44	0.36	0.34	0.43
Std. E.E	14.53	4.90	3.74	1.90	7.11
Cp.	3.34	3.90	7.26	4.64	7.86
Cases Deleted	13	11	6	4	6
R (adj.)	0.42	0.36	0.31	0.14	0.36
Std. E.E	18.53	6.18	4.05	2.22	7.90

Significance

a: $P < 0.05$
b: $P < 0.01$
c: $P < 0.005$
d: $P < 0.001$
e: $P < 0.0001$
f: $P < 0.1$
*: Insigificant

Source: Best possible subset regression analysis.

Not listed in the Table are variables designed for this analysis but never selected for any subsets. With the exception of the dummy variables, the regression coefficients are standardized coefficients. The reason for reporting unstandardized coefficients for the dummy variables is that these category variables, having values of 1 and 0, cannot increase by one standard deviation. Reporting their standardizd coefficients is discouraged (Fox 1984).

From the given R^2, variables selected from our given set of variables do only moderately well. The largest value is just 0.57 of total cancer mortality. Unfortunately, it is also associated with the largest standard error of estimate. As we move on to the four specific cancer sites, we find that the standard errors of estimate decline much faster than the values of R^2.

These changes caution us that it is not always best to combine all cancer sites into a single category, whether the objectives be identification of cancerous factors or differential analysis. Individual cancer sites potentially react in their own particular way to various socioeconomic and demographic conditions. Evidently, this did indeed take place in the five subsets. We can find almost no distinguishable pattern of appearance for the variables other than the three age categories. Even when they team up with other variables, their effects and their being selected differ from when they are by themselves.

In terms of policy-making or cancer prevention programs, informations received from overall cancer mortality analysis can, at times, be impractical. Only when the types of cancer are specific, can we determine who or which kinds of areas (as in our present study) have unusualy high risks, and under what conditions. These limitations of total cancer mortality must be kept in mind when we compare its significant variables with those of the individual cancer sites.

As suggested earlier, age appears rather consistently across all five equations. The exception is the 25-44 age group, which shows up with a negative effect in stomach cancer. Also carrying negative signs is the oldest group of individuals 65 and above. Only in cancer of the intestines has it failed to be selected. The unexpected negative coefficients, especially those for the oldest age group, appear to contradict the fact that cancer mortality increases with age.

Yet, we must take note that attributes of aggregate and individual data must be differentiated. The present results merely indicate that an increase in the proportion of males over 65 would lower the cancer death rates in a census division. It would be erroneous to infer, that cancer mortality declines with age. The question to be resolved, nonetheless, is why the concentration of elderly males has a negative effect on local cancer death rates.

The answers are evident from the correlations presented in Appendix 13. The figures show that the proportion of males above 65 is inversely related to high income, and manufacturing areas, both of which are largely positively associated with cancer mortality. Given these statistical properties, the negative coefficients are not unanticipated.

From the perspective of life cycle, the negative relationships emerge, understandably, as males have their income reduced when they begin to drop out of the work force after retirement. This is likely followed by relocation away from industralized and expensive areas. A correlation of 0.454 between proportion of elderly males and labour force in primary industry (not used in regression) reveals, instead of the elderlies' involvement in the primary sector, their tendency to retire to more rural areas.

These correlations denote that positive effects from those 65 and above could have been masked by the characteristics of the Census Divisions in which they are residing, which are less affluent, low manufacturing, and rural areas. To put it differently, the coefficients could portray a collection of the features that distinguish low cancer death rate areas.

Even so, age effects should not be dismissed. The marked decline of stomach cancer noted between 1960 and 1974 (National Health and Welfare 1979; Statistics Canada 1977) appear to be well matched by a large negative coefficient. In fact, the youngest age group of 25-44 also displays the

same negative sign and has more or less equal strength.

Regarding the middle age group of 45-64, slightly mixed results are found. While a small negative coefficient is registered for intestinal cancer, lung cancer scores the largest positive coefficient. Compared to the oldest age group, the signs of the coefficients depict much better the movements of cancer mortality between 1966 and 1976. Studies by National Health and Welfare (1976) and Statistics Canada (1977) have both indicated a sharp increase of lung cancer mortality, particularly in males above 35 years of age.

Although the latter has also reported no significant changes in intestinal and bladder cancer for the 45-79 and the under 65 age groups, respectively, the age effects of these two cancer sites shown in Fig. 5.14 and 5.19, do partially support the directions of their regression coefficients. We can observe in these diagrams that the age effects of the two cancer sites are moving slowly away from negative values in 1966 to 1976. The pace of movement is slightly faster in bladder cancer than in cancer of the intestine. Consequently, the positive and negative coefficients for bladder and intestinal cancer are reasonable.

In contrast to the performance of the three age groups, several points demand our attentions. First, males between 25 and 44 years of age are in their prime working years, and they can be expected to be exposed to most occupational hazards, including those that are cancer inducing. Yet, the

percentage of males in this age group has the least effect on the five types of cancer. This could be a result of the long incubation period required by most cancer carcinogens.

Males between 45 and 64, on the contrary, are in the latter half of their working years, but their proportion shows the most impact. Such contrasts indirectly underline that, if considering age effect alone on cancer mortality, a latency period of about 20 years should be allowed. Cole and Goldman (1975) have recommended the same incubation period for most carcinogens present in the working environment.

Second, the proportion of males in the middle age group is more likely than the proportion in other age groups to reflect the high risk of cancer. This group's regression coefficients closely follow the directions of cancer mortality movements during the decade of 1966-1976. The rationale for such performance could be that males who survive to these ages have accumulated sufficient carcinogens from the work environment or other sources. Also, enough time related to the latency period has elapsed for cancer to emerge. As a result, personal risk dominates.

Lastly, and contrary to the middle age group, the coefficients for the proportion of elderly males largely echo features of low cancer mortality areas. Selective relocation after retirement is a main factor. It is also possible that, once cancer is detected, patients return to urban centers where medical facilities are more comprehensive. This again intensifies the inverse

relationship already discussed.

In his study on spatial distribution of Canadian mortality, including cancer, Field (1980) reports that this age group of 65 and above is particularly influential. Our current findings suggest that, at least for cancer and for males, a large concentration of elderlies does not warrant high death rates.

Regarding ethnicities, the Asians (Chinese and Japanese) have been precluded from all five equations. The Scandinavians, on the contrary, have negative effects on total cancer and on cancer of the intestines and lung. Their only positive impacts on stomach cancer are well documented in previous findings (Choi, 1968; Newman and Spengler, 1984). For the French, results are more complex. Basically, an increase in this ethnic group will raise the death rate from stomach cancer but lower the rate from intestine, bladder, and lung cancer. Reversed patterns of these influences can be suggested for the English, since their spatial distribution is inversely correlated with the French.

Compared with actual regional variations, our results at this stage are just partially supported. Evidence reveals that from 1960 to 1973, stomach cancer death rate is indeed highest in Quebec, where the French proportion is the largest. However, in Ontario, Manitoba, and British Columbia, with English as the major ethnic group, lung cancer exceeds the national level (Statistics Canada 1977).

For intestinal and bladder cancers, however, there is little compliance between their coefficients and their high mortality in Quebec In the case of total cancer mortality, the percentage of French is not selected at all.

Apparently, the effects of French ethnicity after filtering out influences from other sources, do not always align with observed regional differentiations. In other words, ethnicity can work independently, perhaps through cultural habits, as well as jointly with other factors on cancer mortality. This becomes evident when we evaluate income and education. Regression results show that these two socioeconomic variables by themselves have opposing effects. Turning first to level of income, areas that are above the average have significantly higher death rates in overall cancer and in bladder and lung cancer. The impact on lung cancer is particularly outstanding.

In comparison to previous income differential studies, our findings are rather conflictual. Only Lerner and Stutz (1977) have reported a direct connection between income and cancer mortality in the United States for 1959-1961 and 1969-1971. Yet, neither sex differentiation nor household income is employed in their analysis. Comparability is therefore limited. More relevant to our analysis are the studies by Wigle (1977) and the Health and Welfare Canada (1980). Both have reported a negative relationship between level of census tract income and cancer death rate.

To explain such diversities is not easy, however, speculation is in order. First, the methodologies and units of analysis are completely different. Using the Census Divisions as the unit of analysis may have introduced greater heterogeneity into our results or information could be lost, as Census Tracts are aggregated. In other words, the relationship between income and cancer mortality could vary as areal units differ.

Second, it is not unlikely that the level of income actually has a positive relationship with cancer mortality. There is a common notion that cancer is primarily associated with an affluent life style. This latent positive correlation emerges only when the influences of other factors are removed.

Studies by McMichael (1981) warn against the confounding and interactive effects embedded in social class analysis of cancer mortality. In our case, undesired influences are statistically eliminated, and the real pattern is reflected by the positive partial regression coefficients. Lastly, differences in the cutting point of average household income can produce the conflicting results.

For example, if our national average of $8617 is used to separate high and low census tracts in the two Ontario cities examined by Wigle (1977), then the higher number of lung cancer deaths (150 versus 138) is also in the more affluent areas. Based on these possibilities, our present

findings are not without any foundations. Support is found when we consider the interactive variables of income.

Two types of income interactions are evaluated with age and ethnicity. For the former, men aged 25-44 and 45-64 in high income areas have a positive impact on lung and bladder cancer, respectively. No more interactions are selected for the remaining cancer sites. These results suggest that economic well-being has no unique pattern of influence but depends on individuals' life cycles and the specific cancer sites being examined. Regarding ethnicity, the proportion of French which previously has had no effect on total cancer but negative effect on bladder cancer, now displays positive impacts on both sites for those in high income areas.

Since the percentage of English increases as French decreases, we can infer that the former have lower overall and bladder cancer mortality, even for the same levels of income. This provides evidence that income differentials without taking ethnic background into consideration might lead to biased findings. At this point, all interaction terms involving the proportion of French have signs in line with the high cancer mortality in Quebec. As a result, there is evidence in our findings of a direct relationship between income and cancer death rate.

For the next socioeconomic variable, level of education, the findings are less controvsial. For this variable by itself, an inverse relationship is observed with stomach and lung cancer. Of particular interest are the two

extreme effects of income and education on lung cancer. Their opposing impacts reveal, perhaps, that while income determines one's possible life-style, education helps in deciding how to fulfill one's desires, given the alternatives. For example, one must weight the brief satisfaction gained from cigarette smoking and the long-term health hazard.

With regard to the interaction between education and age, our results are once again different. For males aged 25-44 and in high education areas, they have positive influences on total cancer and cancer of the intestines. The study by Kitagawa and Hauser (1973) on the United states in 1960 report an inverse relationship between years of schooling and cancer death rates for males in the 25-64 age bracket. Unfortunately, no Canadian studies on this subject are available for comparison.

Further discrepancy appears as we look at the interaction between education and ethnicity. The proportion the French in high education areas has significantly higher lung cancer mortality. Nevertheless, the coefficient aligns with the high lung cancer mortality and the large number of regular cigarette smokers in Quebec (Health Protection Branch 1976).

An examination of the industrial variables reveals that the percentages of the labour force engaged in construction, communication, finance and trade have merely marginal effects on the selected cancer sites. Of all the significant

results, only cancer of the stomach and intestines have registered positive coefficients. The latter is consistent with the findings by Howe and Lindsay (1983) of excess intestinal cancer deaths among the workers in the trade industry.

The low-level manufacturing areas have smaller cancer death rates, mainly in cancer of the stomach and the lung. Rather surprisingly, the proportion of males in the 25-44 group together with a low level of manufacturing have a positive influence on death rates from intestinal and lung cancer. These changes denote that the advantages from low manufacturing activities do not benefit all ages equally. Especially for males in prime working years, involvement in industries other than manufacturing have escalated their risk in those two cancer sites. Apparently, there is a need for future inquiries to look into the industrial characteristics of these areas and their relationship with cancer mortality.

As far as the mining industry is concerned, the study by Howe and Lindsay (1983) on the Canadian labor force from 1965 to 1973 found no statistically significant excess deaths among its workers. Initially, our regression analysis yields more or less the same result. Percentage of mining labour force has just a small negative impact on intestinal cancer.

More diversified effects arise when the regions in Canada are distinguished. For the West, which includes

British Columbia and the Prairie Provinces, mining has negative influences on overall cancer, as well as stomach and bladder cancer. In Ontario, however, mining decreases deaths from bladder cancer but increases those from lung cancer, and all cancer sites combined.

The positive impact of Ontario mining on lung cancer demands special attention. Studies by Finkelstein et al. (1982) and Hewitt (1976) have noted escalated cancer risk, including lung cancer, among Ontario uranium miners and those having silicosis. Therefore, it seems that the absence of the mining effect in the study by Howe and Lindsay (1983) is due not to an insufficient latency period, but rather to the lack of regional differentiation. In other words, it is important to recognize the types and locations of mining involved. Simply assessing overall impact would be ineffectual if policy implications are to be considered.

7.4 Female Regression Results

Compared to the males, equations for women in general have fewer variables and a lower R^2. The regression results are given in Table 7.4. The correlations are given in Appendix 14. Except for bladder cancer, values of R^2 range from a high of 0.47 of total cancer to a meager 0.19 of lung cancer. Bladder cancer, which has an impressive R^2 of 0.67, actually would have the least of its variation explained if not for the dummy variable separating areas with no bladder cancer deaths from the rest.

Table 7.4 Best Possible Subset Equations for Female Cancer Mortality by Selected Sites, canada, 1976

	All Cancer	Stomach	Intestine	Bladder	Lung	Breast
Intercept	7.33	5.48	-1.85	-0.73	3.82	3.01
25-44	0.85 [c]	----	0.62 [e]	----	----	0.22 [d]
45-64	----	0.15 [f]	-0.32 [b]	----	0.39 [e]	----
65+	0.31 [e]	-0.33 [c]	0.71 [e]	----	-0.39 [c]	----
Scand.	----	-0.38 [c]	-1.47 [a]	----	----	----
Asian	----	-0.22 [b]	-1.46 [a]	0.09	----	----
French	----	0.24 [c]	----	----	-0.17 [b]	----
Chil	----	N/A	N/A	N/A	N/A	-0.17 [a]
High Income	-0.21 [c]	-0.16 [a]	----	0.17 [*]	----	----
Hi. Income (25-44)	-0.26 [b]	----	----	----	-0.21	----
Hi. Income (French)	0.25 [e]	----	0.22 [e]	----	----	----
Hi. Income (Child)	-0.16 [c]	N/A	N/A	N/A	N/A	-0.12 [*]
Hi. Ed.	----	----	----	----	0.22 [d]	0.13 [a]
Hi. Ed. (25-44)	0.13 [b]	----	-0.31 [d]	0.10 [b]	0.19 [b]	----
Hi. Ed. (French)	----	----	----	----	----	0.17 [d]
Hi.Ed. (Chil)	0.13 [f]	N/A	N/A	N/A	N/A	----
Hi. Manuf.	----	-0.16 [a]	----	0.07 [b]	----	0.16 [b]
Lo. Manuf. (French)	0.21 [e]	----	0.14 [a]	----	----	0.16 [b]
Communic-ation	0.10 [f]	----	----	-0.10 [b]	----	0.18 [b]
Finance-Trade	----	----	0.10	----	----	-0.11 [*]
Hi-Bladder	N/A	N/A	N/A	1.48 [e]	N/A	N/A
R	0.41	0.37	0.30	0.67	0.19	0.27
R (adj.)	0.39	0.35	0.28	0.66	0.18	0.24
Std. E.E	0.54	0.49	4.07	0.37	0.48	4.12
Cp	4.18	2.57	8.18	-1.01	0.93	7.39
Cases Deleted	14	11	3	5	8	4
R (adj.)	0.24	0.29	0.23	0.07	0.11	0.24
Std. E.E	0.67	0.54	4.25	0.44	0.62	4.57

Significance

a: P < 0.05
b: P < 0.01
c: P < 0.005
d: P < 0.001
e: P < 0.0001
f: P < 0.1
*: Insigificant

Source: Best possible subset regression analysis.

To account for the poor performance of the model, fewer females participating in hazardous behaviours and occupations can minimize the explanatory power of the chosen socioeconomic variables. Also, a major portion of female cancer deaths are from breast cancer, and the fact that it is more biologically than socioeconomically induced can further lower R^2.

Turning our attention to the age groups, we would not be too surprised to find that the youngest group of 25-44 has more importance for females than for males. This is mainly because more females died from cancer at these ages, largely from breast cancer. Such patterns are correctly reflected by the positive coefficients for both total and breast cancer. For intestinal cancer, which has the remaining positive coefficient, the present finding suggests that women 25-44 years of age still face considerable risk, despite improvements in their death rates (Statistics Canada 1977).

Regarding the 45-64 age group, significant coefficients are found for intestinal and lung cancer only. In comparison to males, it is not hard to note that the proportion of middle aged females exerts less influence on cancer mortality. This could be accounted for by the age-sex differential of cancer mortality. With few exceptions, empirical evidence generally reveals higher female than male cancer death rates prior to age 45-50, but the situation is reversed after these ages. As far as the oldest age group of

65 and above is concerned, the pattern of effects also departs from that observed among the males. Instead of all negative coefficients, positive impacts are now found on total and intestinal cancers.

Different explanations account for the two negative effects. The remarkable declining trend since the 1960's (Statistics Canada 1977) could be the main reason for the negative coefficient associated with stomach cancer. For lung cancer, the proportion of elderly females could have paralleled their male counterparts by reflecting their residential areal characteristics. These are low income, manufacturing areas with low cancer mortality.

The longer female life expectancy might make indirect contributions to the positive impacts. This appears to be the case for total cancer mortality. As greater numbers of females survive to advanced ages, the result is not only in larger elderly population at risk, but also a longer period of exposure and incubation. For total and intestinal cancers, the outstandingly high cancer risk outweights the effects of areal features and therefore turns in positive coefficients.

Moving to the ethnic variables, we first notice the negative impact of the Scandinavians on stomach cancer. The result is unexpected, and it clearly contradicts the positive impact uncovered for males. Such contradiction cautions us that it is not necessary for both sexes of an ethnic group to have equal risk in the same cancer site. As

for the Asians, their concentration in an area tends to lower stomach and intestinal cancer death rates, but raise rates from bladder cancer. Since some studies report higher stomach cancer mortality among the Japanese (King and Haenszel 1973; King et al. 1985), it may not be wise to combine them with the Chinese into one single group.

Unfortunately, available data do not permit us to analyse them individually. Despite these shortcomings, the absence of effects on breast cancer still parallels the observation of low breast cancer deaths among these two ethnic groups in the United States (King and Haenszel 1973). The proportion of female French population affects stomach and lung cancer in the same way as for males. Also, the patterns of influences comply with the observed regional differentiations. That is, high stomach cancer mortality occurs in areas of French concentration, and high lung cancer death rates in areas with a large English population.

Checking on the socioeconomic variables, we once again find they have different impacts on female cancer mortality. First, the level of income affects fewer cancer sites than they it does for males. This, in turn, provides partial support to our earlier suggestion that females are less likely than males to have their cancer mortality affected by socioeconomic environment. Second, high income areas actually have lower death rates in overall cancer and in cancer of the stomach. For males, except for the insignificant negative coefficient of stomach cancer, the

relationships revealed are all positive.

Even when compared with the past analysis, our present results find themselves with little support. The study by the Health and Welfare Canada (1980) did not observe any noticeable income variations in female total and bladder cancer mortality. For this, differences in the categorization of income level cannot be ruled out as one of the underlying factors. Given the lack of evidence, we can only speculate that male and female cancer mortality react unequally to the same income status, which could be due, possibly, to of the diversified life-styles and occupational hazards.

An inverse relationship emerges when we consider the interaction between income level and age. Females 25-44 years of age in high income areas have fewer overall cancer death. This result seems to suggest that females in this category behave rather differently, resulting in lower cancer mortality, from those in low income areas. When we consider the interaction of income and ethnicity, the effects to be observed share little in common with.

Of the three ethnic group selected for this study, only the French proportion has significant interactive effects. Positive impacts are found on total cancer and on cancer of the intestines. Obviously, such impact departs drastically from the negative effects we reported for level of income alone. Even so, both are in line with the regional differentiations, i.e. death rates of these two cancer sites

are always higher in Quebec. From these findings, there are reasons to believe that the negative impacts of income level are more than statistical artefacts; they are the latent effects that arise only when other influences are removed.

For education, the remaining socioeconomic variable, the results are not as controversial. In fact, its positive effect on cancers of the lung and breast are well supported by previous reports on direct relationships between social class and female cigarette smoking and breast cancer (Graham, 1960; Kitagawa and Hauser, 1973; U.N. 1982). As far as the latter is concerned, low fertility among high social status women has been attributed as the prime factor. With respect to the interactions of education and age, the most interesting observaton is its direct impact on lung cancer. Females aged 25 to 44 in high education areas have significantly higher lung cancer mortality than those living elsewhere.

It might be noted that this age group is most likely to be exposed to hazards from the working environment, such as cigarette smoking, etc. The report on the smoking habits of Canadians (Health and Welfare Canada 1976b) reveals that the largest percentage of female smokers during 1965-1974 is in the 20-45 age bracket. As a result, females of these young ages would have a much higher risk of lung cancer. We may recall the direct relationship between social class and female smoking, and, hence, a positive interaction effect of education and age is well possible.

Turning to the interaction of education and ethnicity, only the effect from the proportion of French changes as education level is varied, and only breast cancer is affected. At this point, we may observe that the proportion of French by itself has not exerted any influences on breast cancer, although death rate is highest in areas of French concentration.

Yet, when the level of education is differentiated, positive impacts that accord with regional variations arise. Similar changes can also be observed for total cancer, for which the French population is influential only when level of income is distinguished. All these show that the true cancer risk for any ethnic group is revealed only when socioeconomic conditions are considered simultaneously.

With regard to the group of industrial variables, only a few have significant impacts on selected cancer sites. Discarded from the analysis are mining, construction, finance, and trade. Of the remaining variables, the high manufacturing areas have higher death rates from cancer of the bladder and the breast. Unlike the results for males, total cancer mortality is not affected. Such diversity indicates that the kinds of manufacturing are likely to be different for the two sexes and possibly less hazardous to the health of females.

The low manufacturing areas are effective mainly in conjunction with the proportion of French. Total cancer and cancer of the intestines and the breast are all positively

affected by this interaction term. Except for breast cancer, it is possible that the French population in low manufacturing areas is involved in other occupations or industries that have adverse effects on intestinal cancer and on sites that are not included in the present study. The latter could, therefore, result in positive impacts on overall cancer mortality.

For breast cancer, the fact that it is more biologically-induced requires us to consider a different interpretation of the significant industrial variables. They should be treated as indicators of women participating in the labor force, which consequently lead to lower fertility or delayed child bearing. The last two factors are the prime suspects in relation to breast cancer.

To test the effect of fertility on this chief cancer site among females, the average number of children under 18 and staying at home are used. Though it is a crude measure of fertility, the result is an expected negative coefficient. Unfortunately, the interactions between socioeconomic conditions and number of children fail to be selected, and the differential effects cannot be tested.

Regarding the interactions of the number of children and the socioeconomic variables, only that with level of income show some influence. Although the negative coefficient is statistically insignificant, its presence calls for speculation. We may remember that number of children by itself already has an inverse effect on breast

cancer death rates. The interaction term indicates that this "children effect" would be even greater in high income areas. This suggests that there might be some special characteristics of the families in more affluent areas that contribute to even lower breast cancer mortality.

One of the many possibilities could be the larger family size in high income areas. It is not unlikely that, given the economic capabilities, women of higher income status choose to have more children. Consequently, the "children effect" is stronger in affluent areas. If this interpretation is valid, then we can find two opposing effects operating on female breast cancer. While the level of income indirectly reduces breast cancer through larger number of children, education level increases breast cancer by lowering fertility by encouraging female labor force participation.

If the above relationships are true, then the class differential studies on breast cancer using a composite index developed from income and education could lead to biased results. Under such circumstances, the findings would depend on the comparative strengths of the two socioeconomic variables. The major shortcoming of the above argument lies in the crude measurement of fertility used. Since the number of children used in this analysis actually refers to those under 24 years of age and staying at home, it is not impossible that in affluent areas, more young and unmarried adults choose to stay home for a longer period of time.

To put it differently, our measurement of fertility favours the high income areas. As a result, the "children effect" is more pronounce in such areas. If this is so, then the interaction term would introduce a bias to our study of breast cancer mortality. The fact that it is not significant saves us from such a risk. Although the above speculations do not allow us to arrive at any concrete solutions regarding the role of income in breast cancer, they do highlight the need to treat these two socioeconomic variables with caution.

8. Summary and Suggestions for Future Studies

The reductions in human mortality and the corresponding increase in life expectancy are made possible largely by socioeconomic developments and medical inventions. Therefore, the structure of causes of death is gradually changed as well. Among the developed countries, infectious or communicable diseases have been almost eradicted. In their place, degenerative diseases have become the dominant causes of death.

Chronic or degenerative diseases are robust to current medical technologies, and they attack mostly mature adults. These features, in combination with the aging of the population in the developed countries, result in the anticipation of no substantial life expectancy gains in the coming years. While most of the degenerative diseases are difficult to cure, their causes have been largely related to hazardous life-styles.

In fact, 80-90 per cent of all cancer has been regarded as preventable. It is recognized that the high risk of cancer can be due to diet, excessive alcohol consumption, cigarette smoking, pollutants in different working environments, and low fertility. Since these elements are embedded in the social milieu, the socioeconomic differentials in cancer mortality need to be examined.

Studies carried out in Canada and elsewhere generally suggest an inverse relationship between social class and cancer death rates. Exceptions arise when other demographic

factors are taken into consideration. In order to better understand cancer mortality differentials, variations reported by age, sex, and ethnicity have to be looked into as well. In general, differential analysis studying only one social, demographic, or occupational aspect at a time is not sufficient to expose the true effects on cancer mortality. This thesis examines several factors acting on cancer mortality simultaneously.

A trend analysis reveals that the age effect and cancer force have different dominant powers in male and female cancer death rates. The high male cancer force but large female age effect reflect the differentials in cancer sites and other responsible factors among the two sexes. It is also observed that both components need not act in the same direction to account for the changes in the death rate from a specific cancer site.

The future cancer death rates in Canada are predicted using results from the trend analysis. Both the predicted crude and standardized death rates indicate that cancer in this country will continue to rise. The prediction equations are developed for male and female total cancer death rates only. This process can be repeated for other cancer sites as well. In fact, if this is done for all major cancer sites, then future Canadian cancer mortality can be better depicted.

Based on ecological data, the socioeconomic analysis of Canadian cancer death rates is rather complex. We find that

cancer death rates show differentials by areas of high and low income or education. However, the socioeconomic forces causing these differentials do not work individually. Their interactions with other variables are found to play crucial roles. An unexpected positive relationship is observed between income and male cancer death rates. Such findings differ from most of the previous results, and they are attributed to be the actual impact of income on cancer mortality after removing the confounding and interactive factors.

For females, income and education operates differently than in the case of males. This is largely because of breast cancer, which does not react in the same way to these socioeconomic variables. Even on this specific cancer site, income and education have opposing effects. The coefficient of income and fertility interaction term, though statistically insignificant, tends to reduce breast cancer. Level of education, on the other hand, helps to increase deaths from this cancer site. While the positive impact of education is understandable, the potential negative effect of income demands further inquiry.

Of the industrial variables, mining has shown some interesting results. Only mining in Ontario has a positive impact on cancer death rates, particularly from lung cancer. Although some earlier studies have already identified some specific types of mining, further inquiries into the pecularities of the mining industry in this province would

be beneficial.

Throughout this analysis, the French ethnic group has always stood out by itself with positive influences on cancer death rates. Its high risk obviously deserves special attention. Furthermore, it is important to find out whether such a high risk of cancer is culturally or behaviorally related or whether it is due to occupational hazards.

In terms of possible policy applications, differences between males and females in terms of their income and education effect on lung cancer suggest that a single preventive program which applies to all migh not achieve the desired effects. If cigarette smoking is attributed as the main cause of lung cancer, then perhaps anti-smoking programs that begin during early schooling and are directed at males might have more curtailing effects in the long run. This approach is suggested in view of the negative coefficient between level of education and male lung cancer.

For females, however, who have a positive education impact on lung cancer, another tactic should be adopted. Perhaps anti-smoking commercials on television employing popular celebrities might help to stop cigarette smoking before it becomes a habit. The main concept here is that research of this kind can help to identify high cancer risk groups more correctly and in greater detail. Information received is more practical in terms of policy design.

Although the present study has shown some encouraging results, there is room for improvement. The most apparent

could be the employment of more refined areal units in order to reduce heterogeneity within the unit of analysis. Also, more refined measurements of fertility, income, and education should be adopted.

The use of ecological data in this analysis has limited us to identifying the characteristics of high cancer mortality areas instead of population subgroups. To assess the persons at high cancer risk, individual data have to be employed. In fact, individual records of those deceased can be linked to earlier Census records or even to income tax and industrial employment records.

Such linkage could provide larger numbers of socioeconomic variables for more complete assessment of mortality at the individual level. The kinds of linkage studies carried out in the United States have already proven successful in providing information on mortality differentials or on the identification of risk factors. The shortage of information on Canadian mortality prvides strong rationale for encouraging analysis of this nature.

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Appendix 1 Bivariate Regression Results of Male Age Effect

Age Group	Regression Coefficient (Standardized)	t-statistics	R^2
25-44 GR	0.719	5.68	0.52
45-64 GR	-0.811	-7.59	0.66
65+ GR	0.825	8.01	0.68

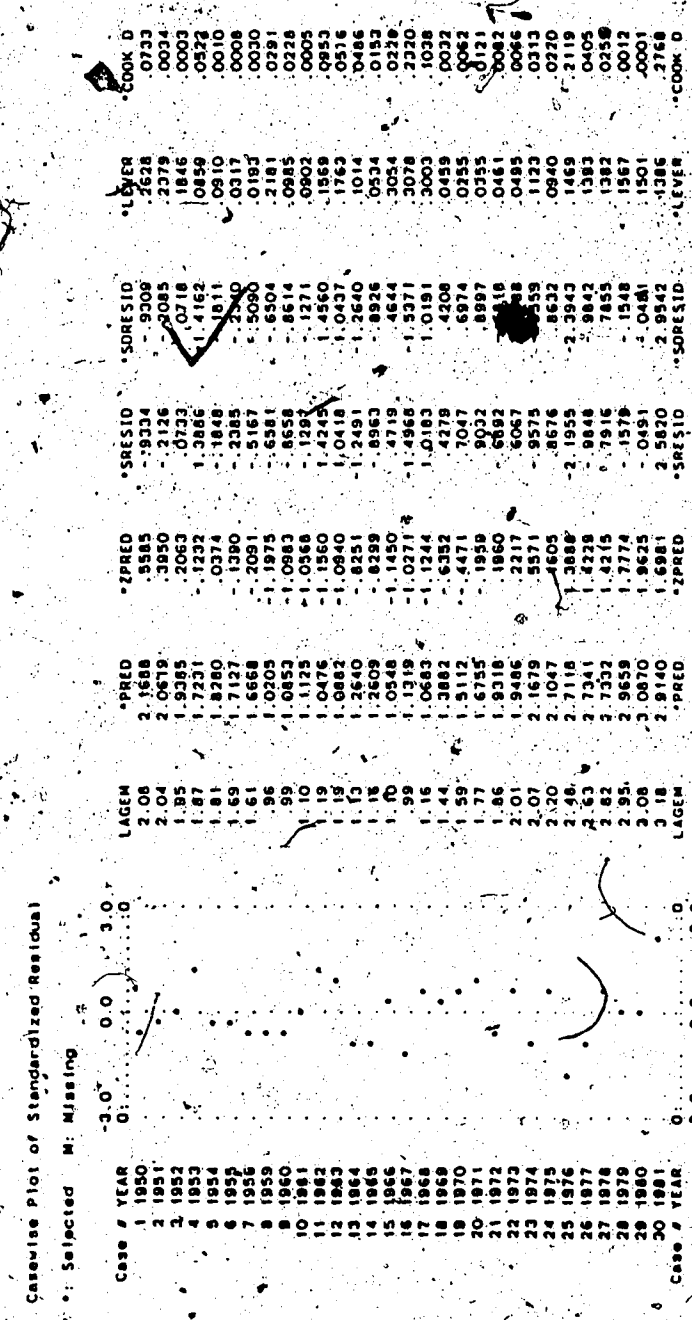
Source: From bivariate regression.

Appendix 2 Casewise Residual Plot of Male Age Effect, Re-Specified Model, Canada, 1950-1981

Case #	YEAR	-3.0 0.0 3.0	LAGEM	*PRED	*ZPRED	*SRESID	*SDRESID	*LEVER	*COOK D
1	1950	0	2.08	2.2392	.7412	-1.2236	-1.2360	.3117	.1303
2	1951		2.04	2.0679	.4799	-.1974	-.1937	.2667	.0028
3	1952		1.95	2.0633	.4729	-.9495	-.9476	.1947	.1047
4	1953		1.87	1.6823	-.1081	1.2558	1.2706	.0912	.0367
5	1954		1.81	1.8269	.1123	-.1244	-.1220	.1092	.0004
6	1955		1.69	1.5818	-.2615	.6695	.6623	.0277	.0047
7	1956		1.61	1.5351	-.3327	.4765	.4693	.0304	.0025
8	1957		1.16	1.3384	-.6327	-1.1970	-1.2075	.1410	.0500
9	1958		.83	1.1666	-.8948	-2.2353	-2.4388	.1095	.1365
10	1959		.96	.9362	-1.2462	.1464	.1436	.3005	.0018
11	1960		.99	1.0282	-1.1057	-.2341	-.2298	.1020	.0014
12	1961		1.10	1.1404	-.9347	-.2830	-.2780	.1265	.0025
13	1962		1.19	1.1413	-.9333	.3949	.3884	.2852	.0120
14	1963		1.19	1.1927	-.8551	.0098	.0096	.3379	.0000
15	1964		1.13	1.2008	-.8425	-.4725	-.4653	.1352	.0074
16	1965		1.16	1.0925	-1.0078	.4649	.4577	.0794	.0045
17	1966		1.10	1.1506	-.9192	-.3481	-.3421	.1091	.0033
18	1967		.99	1.2323	-.7947	-1.5630	-1.6329	.0898	.0575
19	1968		1.16	1.1836	-.8688	-.1348	-.1322	.0769	.0004
20	1969		1.16	1.1650	-.8972	1.7620	1.8412	.0631	.0539
21	1970		1.59	1.3022	-.6881	1.8606	1.9536	.0508	.0516
22	1971		1.77	1.7391	-.0216	.2575	.2529	.2217	.0037
23	1972		1.86	1.9552	.3080	-.6563	-.6490	.0940	.0103
24	1973		2.01	1.8122	.0900	1.3349	1.3563	.0802	.0373
25	1974		2.07	2.1028	.5332	-.2621	-.2574	.2429	.0043
26	1975		2.20	2.0809	.4998	.7910	.7852	.1355	.0208
27	1976		2.48	2.6845	1.4204	-1.3732	-1.3982	.1545	.0717
28	1977		2.63	2.7156	1.4678	-.5756	-.5683	.1548	.0126
29	1978		2.82	2.6847	1.4208	.9022	.8989	.1598	.0320
30	1979		2.95	2.8879	1.8832	-.2637	-.2588	.1697	.0027
31	1980		3.09	3.1562	2.1399	-.5239	-.5204	.2057	.0144
32	1981		3.16	2.9172	1.7754	1.8374	1.9315	.1669	.1390
Case #	YEAR	0.0 0.0 3.0	LAGEM	*PRED	*ZPRED	*SRESID	*SDRESID	*LEVER	*COOK D

Source: Regression analysis.

Appendix 3 Casewise Residual Plot of Male Age Effect, Final Model, Canada, 1950-1981



Casewise Plot of Standardized Residual

*: Selected M: Missing

-3.0 0.0 3.0

Case #	YEAR									
1	1950	2.08	2.1688	.5585	-.9334	-.9309	.7628	.0733	*COOK D
2	1951	2.04	2.0679	.3850	-.2126	-.2085	.2379	.0034	
3	1952	1.95	1.9385	.2063	.0733	.0718	.1846	.0003	
4	1953	1.87	1.7231	.1232	1.3866	1.4162	.0859	.0522	
5	1954	1.81	1.8280	.0374	-.1848	-.1811	.0910	.0010	
6	1955	1.69	1.7127	.1390	-.2385	-.2340	.0317	.0008	
7	1956	1.61	1.6668	.2091	-.5167	-.5090	.0193	.0030	
8	1959	.96	1.0205	1.1975	-.6581	-.6504	.2181	.0291	
9	1960	.99	1.0853	1.0983	-.8658	-.8614	.0985	.0228	
10	1961	1.10	1.1125	1.0568	-.1294	-.1271	.0802	.0005	
11	1962	1.19	1.0476	1.0840	1.4245	1.4560	.1569	.0953	
12	1963	1.19	1.0882	1.0251	1.0418	1.0437	.1763	.0516	
13	1964	1.13	1.2640	.8251	-1.2491	-1.2640	.1014	.0486	
14	1965	1.18	1.2609	.8299	-.8963	-.8926	.0534	.0153	
15	1966	1.00	1.0548	1.1450	.4719	.4644	.3034	.0228	
16	1967	.99	1.1319	1.0271	-1.4968	-1.5371	.3078	.2320	
17	1968	1.16	1.0683	1.1244	1.0183	1.0191	.3003	.1038	
18	1969	1.44	1.3882	.6352	.4278	.4208	.0459	.0032	
19	1970	1.59	1.5112	.4471	.7047	.6974	.0255	.0062	
20	1971	1.77	1.6755	.1959	.9032	.8997	.0355	.0121	
21	1972	1.86	1.9318	.1860	.6892	.6818	.0461	.0082	
22	1973	2.01	1.9486	.2217	.6067	.5959	.0485	.0066	
23	1974	2.07	2.1679	.5571	-.9575	-.8632	.1123	.0313	
24	1975	2.20	2.1047	.1605	.8676	.8632	.0940	.0220	
25	1976	2.46	2.7118	.3888	-2.1955	-2.3943	.1469	.2119	
26	1977	2.63	2.7341	.4228	-.9848	-.9842	.1383	.0405	
27	1978	2.82	2.7332	.4215	.7916	.7855	.1382	.0258	
28	1979	2.95	2.9659	.7774	-.1578	-.1548	.1567	.0012	
29	1980	3.08	3.0870	.9625	-.0491	-.0481	.1501	.0001	
30	1981	3.18	2.9140	.6981	2.5820	2.9542	.1386	.2768	
Case #	YEAR	LAGEM	*PRED	*ZPRED	*SRESID	*SDRESID	*LEVER	*COOK D	

Source: Regression analysis.

Appendix 4 Bivariate Regression Results of Female Age Effect, Canada, 1950-1981

Age Group	Regression Coefficient (Standardized)	t-statistics	R
25-44 GR	0.397	2.37	0.16
45-64 GR	-0.526	-3.39	0.27
65+ GR	0.384	2.28	0.15

Source: From bivariate regression.

Appendix 5 Casewise Residual Plot of Female Age Effect, Initial Model, Canada, 1950-1981

Casewise Plot of Standardized Residual

*: Selected M: Missing

Case #	YEAR	-3.0 ... 0.0 ... 3.0	LAGEF	*PRED	*RESID	*SRESID	*LEVER	*COOK D
1	1950		1.55	2.8678	-1.3203	-2.1569	.0297	.1347
2	1951		1.59	3.0144	-1.4252	-2.2300	.0562	.2383
3	1952		1.61	2.2816	-.6722	-1.0256	.0074	.0211
4	1953		1.63	2.4575	-.8283	-1.2707	.0001	.0256
5	1954		1.65	2.2875	-.6388	-.9728	.0070	.0188
6	1955		1.63	2.5806	-.9514	-1.4463	.0021	.0361
7	1956		1.61	2.7565	-1.1471	-1.7557	.0152	.0751
8	1957		1.57	1.9885	-.4199	-.6535	.0463	.0180
9	1958		1.50	2.1468	-.6427	-.9869	.0212	.0269
10	1959		1.59	1.8302	-.2410	-.3824	.0813	.0093
11	1960		1.84	2.0237	-.1831	-.2840	.0399	.0031
12	1961		1.97	2.1644	-.1903	-.2919	.0190	.0023
13	1962		2.07	2.2172	-.1503	-.2288	.0131	.0012
14	1963		2.19	2.3520	-.1659	-.2524	.0030	.0011
15	1964		2.27	2.2582	.0139	.0213	.0093	.0000
16	1965		2.38	2.1937	.1858	.2801	.0156	.0020
17	1966		2.47	2.1292	.3389	.5146	.0235	.0079
18	1967		2.58	2.0296	.5907	.8496	.0389	.0275
19	1968		2.71	2.0471	.6609	1.0236	.0359	.0376
20	1969		2.83	2.0471	.7861	1.2267	.0359	.0532
21	1970		2.90	2.1703	.7312	1.1260	.0183	.0327
22	1971		3.00	2.4399	.5608	.8477	.0003	.0118
23	1972		3.11	2.5630	.5505	.8323	.0014	.0116
24	1973		3.20	2.6158	.5829	.8836	.0038	.0143
25	1974		3.25	2.6275	.6267	.9524	.0044	.0168
26	1975		3.27	2.6334	.6362	.9675	.0048	.0175
27	1976		3.37	2.8268	.5439	.8320	.0238	.0204
28	1977		3.42	3.0027	.4183	.6472	.0538	.0199
29	1978		3.52	3.0079	.4826	.7516	.0612	.0292
30	1979		3.60	3.3076	.2897	.4679	.1342	.0223
31	1980		3.66	3.2489	.4146	.6648	.1159	.0389
32	1981		3.71	3.1088	.6028	.9531	.0775	.0556
Case #	YEAR	-3.0 ... 0.0 ... 3.0	LAGEF	*PRED	*RESID	*SRESID	*LEVER	*COOK D

Source: Regression analysis.

Appendix 6 Casewise Residual Plot of Female Age Effect, Trend Added, Canada, 1950-1981

Case #	YEAR	LAGEF	*PRED	*RESID	*SRESID	*SDRESID	*LEVER	*COOK D
1	1950	1.55	1.5684	-.0209	-.4709	-.1678	.0364	.0009
2	1951	1.59	1.4938	.0958	.8164	.8112	.1702	.0336
3	1952	1.61	1.4920	.1175	1.0062	1.0064	.1724	.0518
4	1953	1.63	1.5994	.0288	.2412	.2369	.0748	.0014
5	1954	1.65	1.5976	.0511	.4131	.4067	.0759	.0044
6	1955	1.63	1.6631	-.0339	-.2699	-.2652	.0466	.0012
7	1956	1.61	1.6996	-.0901	-.7150	-.7064	.0404	.0079
8	1957	1.57	1.5848	-.0162	-.1318	-.1294	.0443	.0005
9	1958	1.50	1.7378	-.2237	-.8555	-.8483	.0418	.0543
10	1959	1.59	1.8362	-.2469	-2.0039	-1.9734	.0818	.1023
11	1960	1.84	1.8580	-.0775	-.1430	-.1404	.0979	.0006
12	1961	1.97	1.8944	.0796	.6649	.6579	.1305	.0171
13	1962	2.07	1.9545	.1123	.9787	.9785	.2000	.0577
14	1963	2.19	1.9818	.2042	1.8259	1.9139	.2381	.2459
15	1964	2.27	2.4740	-.2019	-1.8637	-1.7233	.1084	.0899
16	1965	2.38	2.5134	-.1339	-1.0981	-1.1025	.1006	.0366
17	1966	2.47	2.6530	-.1849	-1.5101	-1.5487	.0932	.0648
18	1967	2.58	2.6256	-.0454	-.3800	-.3739	.1337	.0057
19	1968	2.71	2.6182	.0898	.7426	.7363	.1143	.0188
20	1969	2.83	2.6000	.2332	1.9300	2.0359	.1161	.1288
21	1970	2.90	2.7292	.1722	1.4002	1.4268	.0853	.0517
22	1971	3.00	2.8828	.1179	.7635	.8397	.7075	.7587
23	1972	3.11	3.1905	-.0767	-.6273	-.6201	.0946	.0113
24	1973	3.20	3.1393	.0594	.4721	.4652	.0445	.0037
25	1974	3.25	3.2976	-.0434	-.3556	-.3498	.0994	.0038
26	1975	3.27	3.2782	-8.5928E-03	-.0608	-.0685	.0836	.0001
27	1976	3.37	3.4252	-.0544	-.4365	-.4298	.0604	.0038
28	1977	3.42	3.4872	-.0662	-.5382	-.5310	.0844	.0076
29	1978	3.52	3.4422	.0782	.6336	.6265	.0781	.0099
30	1979	3.60	3.6359	-.0385	-.3298	-.3242	.1706	.0055
31	1980	3.66	3.7258	-.0622	-.5302	-.5230	.1648	.0137
32	1981	3.71	3.5765	.1347	1.1102	1.1152	.1093	.0403
Case #	YEAR	LAGEF	*PRED	*RESID	*SRESID	*SDRESID	*LEVER	*COOK D

Casewise Plot of Standardized Residual

*: Selected, M: Missing

Case #	YEAR	-3.0	0.0	3.0
1	1950	0	.	:0
2	1951		.	:0
3	1952		.	:0
4	1953		.	:0
5	1954		.	:0
6	1955		.	:0
7	1956		.	:0
8	1957		.	:0
9	1958		.	:0
10	1959		.	:0
11	1960		.	:0
12	1961		.	:0
13	1962		.	:0
14	1963		.	:0
15	1964		.	:0
16	1965		.	:0
17	1966		.	:0
18	1967		.	:0
19	1968		.	:0
20	1969		.	:0
21	1970		.	:0
22	1971		.	:0
23	1972		.	:0
24	1973		.	:0
25	1974		.	:0
26	1975		.	:0
27	1976		.	:0
28	1977		.	:0
29	1978		.	:0
30	1979		.	:0
31	1980		.	:0
32	1981		.	:0
Case #	YEAR	-3.0	0.0	3.0

Source: Regression analysis.

Appendix 7 Casewise Residual Plot of Female Age Effect, Trend Added, Final Model, Canada, 1950-1981

Casewise Plot of Standardized Residual

. Selected M Missing

Case #	YEAR	-3.0 0.0 3.0	LAGEF	*PRED	*RESID	*SRESID	*SDRESID	*LEVER	*COOK D
1	1950	0 : : : 0	1.55	1.7328	-.1852	-1.7405	-1.8127	.1285	.1451
2	1951	. : : .	1.59	1.6585	-.0692	-.6793	-.6724	.1979	.0345
3	1952	. : : .	1.61	1.4848	.1247	1.1881	1.1987	.1527	.0802
4	1953	. : : .	1.63	1.5782	.0500	.4559	.4481	.0752	.0068
5	1954	. : : .	1.65	1.6328	.0159	.1447	.1421	.0767	.0006
6	1955	. : : .	1.63	1.6267	2.5646E-03	.0231	.0226	.0515	.0000
7	1956	. : : .	1.61	1.5948	.0146	.1323	.1298	.0620	.0005
8	1957	. : : .	1.57	1.4068	.1618	1.8143	1.5534	.1209	.1037
9	1958	. : : .	1.50	1.6501	-.1461	-1.3164	-1.3354	.0551	.0435
10	1960	. : : .	1.84	1.8650	-.0244	-.2199	-.2160	.0539	.0011
11	1961	. : : .	1.97	2.0779	-.1039	-.9409	-.9388	.0646	.0237
12	1962	. : : .	2.07	1.9913	.0756	.6984	.6916	.1001	.0186
13	1963	. : : .	2.19	2.0901	.0960	.8919	.8884	.1091	.0327
14	1964	. : : .	2.27	2.3622	-.0900	-.8217	-.8166	.0774	.0208
15	1965	. : : .	2.38	2.4483	-.0688	-.6218	-.6146	.0606	.0089
16	1966	. : : .	2.47	2.6020	-.2138	-1.9079	-2.0128	.0361	.0668
17	1967	. : : .	2.58	2.6771	-.0969	-.8715	-.8675	.0515	.0174
18	1968	. : : .	2.71	2.6610	.0471	.4209	.4144	.0404	.0035
19	1968	. : : .	2.83	2.6346	.1886	1.7798	1.8590	.0447	.0660
20	1970	. : : .	2.90	2.7615	.1389	1.2416	1.2612	.0359	.0285
21	1971	. : : .	3.00	2.9027	.0980	1.5340	1.5756	.6651	1.3556
22	1972	. : : .	3.11	3.2336	-.1201	-1.0940	-1.0982	.0750	.0359
23	1973	. : : .	3.20	3.1398	.0588	.5266	.5194	.0426	.0056
24	1974	. : : .	3.25	3.3586	-.1044	-.9534	-.9517	.0782	.0285
25	1975	. : : .	3.27	3.3290	-.0595	-.5400	-.5327	.0687	.0082
26	1976	. : : .	3.37	3.4606	-.0859	-.8158	-.8106	.0684	.0186
27	1977	. : : .	3.42	3.4674	-.0464	-.4219	-.4148	.0688	.0050
28	1978	. : : .	3.52	3.3845	.1359	1.2228	1.2346	.0515	.0342
29	1978	. : : .	3.60	3.5393	.0580	.5318	.5246	.0852	.0094
30	1980	. : : .	3.66	3.6922	-.0286	-.2674	-.2628	.1181	.0032
31	1981	. : : .	3.74	3.5415	.1696	1.5521	1.5960	.0823	.0760
Case #	YEAR	0 : : : 0	LAGEF	*PRED	*RESID	*SRESID	*SDRESID	*LEVER	*COOK D
		-3.0 0.0 3.0							

Source: Regression analysis.

Appendix 8 Casewise Residual Plot of Male Cancer Force, Initial Model, Canada, 1950-1981

Casewise Plot of Standardized Residual

*: Selected X: Missing

Case #	YEAR	-3.0	0.0	3.0	*ZRESID	*SRESID	*SDRESID	*LEVER	*COOK D
1	1950				-.5771	-.6180	-.6065	.0881	.0256
2	1951				-1.0822	-1.1458	-1.1521	.0711	.0798
3	1952				.8556	.8852	.9847	.0668	.0438
4	1953				.1827	.2018	.1986	.0573	.0020
5	1954				.1637	.1707	.1679	.0485	.0013
6	1955				.9815	1.0280	1.0001	.0404	.0408
7	1956				.9149	.8458	.9441	.0331	.0304
8	1957				.2672	.2452	.2709	.0265	.0023
9	1958				.3330	.3420	.3369	.0206	.0032
10	1959				-2.2182	-2.1720	-2.4549	.0155	.1265
11	1960				-.6288	-.6427	-.6363	.0111	.0091
12	1961				.4844	.4841	.4877	.0074	.0049
13	1962				-.1633	-.1663	-.1636	.0045	.0005
14	1963				-.8111	-.8250	-.8205	.0023	.0118
15	1964				.6497	.6603	.6540	.0008	.0072
16	1965				-1.7258	-1.7535	-1.8198	.0001	.0497
17	1966				.0063	.0064	.0063	.0001	.0000
18	1967				.6437	.6543	.6479	.0008	.0071
19	1968				.0912	.0927	.0914	.0023	.0001
20	1969				.8665	.8843	.8837	.0045	.0180
21	1970				1.5563	1.5873	1.6306	.0074	.0507
22	1971				1.0989	1.1230	1.1281	.0111	.0278
23	1972				1.4983	1.5346	1.5718	.0155	.0577
24	1973				1.5645	1.6068	1.6525	.0206	.0706
25	1974				.6312	.6503	.6438	.0265	.0130
26	1975				.0165	.0171	.0168	.0331	.0000
27	1976				-.5620	-.5806	-.5841	.0404	.0135
28	1977				-.4076	-.4249	-.4191	.0485	.0078
29	1978				-.2938	-.3078	-.3031	.0573	.0046
30	1979				-.0372	-.0382	-.0385	.0668	.0001
31	1980				.2090	.2213	.2178	.0771	.0030
32	1981				-2.4345	-2.4345	-2.6720	.0881	.4015
Case #	YEAR	-3.0	0.0	3.0	*ZRESID	*SRESID	*SDRESID	*LEVER	*COOK D

Source: Regression analysis

Appendix 9 Casewise Residual Plot of Male Cancer Force, Final Model, Canada, 1950-1981

Case #	YEAR	FORCENC	*PRED	*RESID	*SRESID	*SDRESID	*LEVER	*COOK D
1	1952	-5.40	-5.7651	.3681	.4960	.4873	.2139	.0138
2	1953	-5.60	-4.6343	-.9657	-1.2688	-1.0877	.1820	.0800
3	1954	-4.30	-3.5036	-.7964	-1.0109	-1.0208	.1197	.0387
4	1955	-1.20	-2.3729	1.1729	1.5406	1.5167	.0870	.0615
5	1956	.00	-1.2422	1.2422	1.6314	1.5836	.0838	.0533
6	1957	.10	-.1115	.1115	.1372	.1341	.0503	.0004
7	1958	2.20	1.0192	1.1808	1.5506	1.5366	.0463	.0232
8	1959	5.80	3.2807	2.5193	3.3087	3.3681	.0671	.0418
9	1960	5.90	4.4114	1.4886	1.9554	1.9883	.0818	.0419
10	1961	5.90	5.3421	.5579	.7328	.4513	.1261	.0083
11	1962	7.50	7.8035	-.3035	-.3986	-1.0216	.1700	.0044
12	1963	11.70	11.8917	-.2017	-.2649	-.2724	.2234	.0055
13	1964	14.40	13.8226	.5774	.7585	.4408	.3706	.0288
14	1965	14.60	15.0636	-1.0636	-1.3970	-1.3962	.2368	.0479
15	1966	17.80	17.4815	.3185	.4184	.3902	.1415	.0048
16	1967	20.40	19.3955	1.0045	1.3194	1.3874	.0939	.0564
17	1968	20.80	21.1464	-.3464	-.4550	-.4412	.0920	.0056
18	1969	23.00	22.9774	.0226	.0297	.0305	.1415	.0001
19	1970	24.50	24.5043	-.0043	-.0056	-.0062	.2368	.0022
20	1971	23.90	22.9036	.9964	1.3089	1.3227	.3786	.0638
21	1972	23.80	24.1243	-.3243	-.4260	-.3820	.1488	.0032
22	1973	24.10	25.2550	-1.1550	-1.5172	-1.5138	.1250	.0750
23	1974	25.80	26.3857	-.5857	-.7692	-.7362	.1056	.0185
24	1975	27.40	27.5164	-.1164	-.1529	-.1450	.1106	.0008
25	1976	28.30	28.6471	-.3471	-.4559	-.4328	.4250	.0272
26	1977	30.30	29.7778	.5221	.6858	.6720	.1489	.0212
Case #	YEAR	FORCENC	*PRED	*RESID	*SRESID	*SDRESID	*LEVER	*COOK D

Casewise Plot of Standardized Residual

* Selected M: Missing

```
Case #  YEAR      -3.0 ..... 0.0 ..... 3.0
     1  1952        0:                   :
     2  1953                             :
     3  1954                             :
     4  1955                             :
     5  1956                             :
     6  1957                             :
     7  1958                             :
     8  1959                             :
     9  1960                             :
    10  1961                             :
    11  1962                             :
    12  1963                             :
    13  1964                             :
    14  1965                             :
    15  1966                             :
    16  1967                             :
    17  1968                             :
    18  1969                             :
    19  1970                             :
    20  1971                             :
    21  1972                             :
    22  1973                             :
    23  1974                             :
    24  1975                             :
    25  1976                             :
    26  1977                             :
    27  1978                             :
    28  1979                             :
    29  1980       -3.0 ..... 0.0 ..... 3.0
Case #  YEAR
```

Source: Regression analysis.

Appendix 10 Casewise Residual Plot of Female Cancer Force, Initial Model, Canada, 1950-1981

Casewise Plot of Standardized Residual

*: Selected M: Missing

Case #	YEAR	-3.0 0.0 3.0	FORCEFC	*PRED	*RESID	*SRESID	*LEVER	*COOK D
1	1950	0 : : : 0	4.30	2.5375	1.7625	1.2823	.0818	.1166
2	1951		3.50	2.0963	1.4037	1.0153	.0606	.0635
3	1952		1.30	1.6551	-.3551	-.2510	.0700	.0033
4	1953		2.40	1.2139	1.1861	.8435	.0602	.0366
5	1954		1.30	.7727	.5272	.3684	.0511	.0064
6	1955		.10	.3315	-.4315	-.3007	.0427	.0038
7	1956		.00	-.1097	.1097	.0760	.0351	.0002
8	1957		.00	-.5509	.5509	.3812	.0283	.0048
9	1958		.00	-.9921	.3079	.2156	.0221	.0013
10	1959		-1.30	-1.4333	.1333	.0931	.0171	.0002
11	1960		-1.30	-1.8745	.5745	.4001	.0133	.0037
12	1961		-6.30	-2.3157	-3.9843	-2.7896	.0083	.1620
13	1962		-2.70	-2.7569	.0569	.0395	.0051	.0000
14	1963		-2.70	-3.1980	.4980	.3452	.0027	.0022
15	1964		-4.00	-3.6392	-.3608	-.2498	.0011	.0011
16	1965		-4.80	-4.0804	-.8196	-.5673	.0002	.0054
17	1967		-7.60	-4.5216	-3.0784	-2.1307	.0000	.0757
18	1968		-4.90	-4.9628	.0628	.0435	.0006	.0000
19	1969		-5.70	-5.4040	-.2960	-.2051	.0020	.0007
20	1970		-6.20	-5.8452	-.3548	-.2461	.0040	.0011
21	1971		-6.60	-6.2864	-.3136	-.2178	.0069	.0010
22	1972		-8.30	-6.7276	-1.5724	-1.0942	.0104	.0267
23	1973		-5.50	-7.1688	1.6688	1.1640	.0147	.0334
24	1974		-8.50	-7.6100	1.1100	.7765	.0198	.0165
25	1975		-6.30	-8.0512	1.7512	1.2285	.0256	.0463
26	1976		-10.00	-8.4924	-1.5076	-1.0613	.0322	.0388
27	1977		-11.30	-8.9336	-2.3664	-1.6725	.0394	.1060
28	1978		-9.90	-9.3748	-.5252	-.3728	.0475	.0060
29	1979		-10.20	-9.8160	-.3840	-.2739	.0563	.0038
30	1980		-8.00	-10.2572	2.2572	1.6183	.0658	.1424
31	1981		-9.40	-10.6984	1.2984	.9362	.0761	.0532
			-9.30	-11.1396	1.8396	1.3347	.0871	.1207
Case #	YEAR	0 : : : 0	FORCEFC	*PRED	*RESID	*SRESID	*LEVER	*COOK D
		-3.0 0.0 3.0						

Source: Regression analysis.

Appendix 11 Casewise Residual Plot of Female Cancer Force, Final Model, Canada, 1950-1981

Selected M: Missing

Case #	YEAR	-3.0 0.0 3.0	FORCEFC	*PRED	*RESID	*SRESID	*SDRESID	*LEVER	*COOK D
1	1950		4.20	3.4434	.8566	1.4843	1.5229	.1433	.1181
2	1951		3.50	2.8341	.6659	.9660	.9647	.1184	.0421
3	1952		1.30	2.4249	-1.1249	-1.8957	-2.0073	.0963	.1362
4	1953		2.40	1.9456	.4544	.8073	.8015	.0770	.0204
5	1954		1.30	1.4064	-.1064	-.1757	-.1723	.0603	.0008
6	1955		-.10	.8972	-.9972	-1.6342	-1.6943	.0464	.0588
7	1956		.00	.3878	-.3878	-.6318	-.6242	.0353	.0075
8	1957		-1.30	.1213	-1.4213	-.1966	-.1929	.0268	.0006
9	1958		-1.30	.6306	-1.9306	-1.0423	-1.0862	.0211	.0172
10	1959		-.10	-1.1398	1.0398	1.6785	1.7459	.0181	.0391
11	1960		-1.30	-1.6491	.3491	.5634	.5555	.0178	.0044
12	1962		-2.70	-2.6675	-.0325	-.0526	-.0515	.0255	.0000
13	1963		-2.70	-3.1766	.4766	.7759	.7696	.0334	.0110
14	1964		-4.00	-3.6860	-.3140	-.5139	-.5062	.0441	.0056
15	1965		-4.90	-4.1953	-.7047	-1.1620	-1.1706	.0575	.0342
16	1967		-4.90	-5.2137	.3137	.5276	.5198	.0924	.0101
17	1968		-5.70	-5.7230	.0230	.0391	.0383	.1140	.0001
18	1970		-6.20	-6.2322	.0322	.0557	.0546	.1383	.0002
19	1972		-6.60	-6.7415	.1415	.2485	.2438	.1653	.0039
20	1973		-5.50	-5.7788	.2788	.4626	.4551	.0681	.0061
21	1973		-6.50	-6.1643	-.3357	-.5604	-.5525	.0794	.0101
22	1974		-6.30	-6.5497	.2497	.4197	.4127	.0917	.0064
23	1825		-10.00	-9.5303	-.4697	-.8327	-.8275	.1800	.0473
24	1976		-11.30	-10.0523	-1.2477	-2.2447	-2.4614	.2028	.3918
25	1977		-9.90	-10.5743	.6743	1.2339	1.2465	.2271	.1346
26	1978		-10.20	-10.0963	-.1037	-.1863	-.1839	.2529	.0806
27	1979		-8.00	-8.4768	.4768	.8397	.8346	.1696	.0452
28	1980		-8.40	-8.8622	.4622	.8378	.8317	.1884	.0659
29	1981		-9.30	-9.2477	-.0523	-.0945	-.0926	.2083	.0007
Case #	YEAR		FORCEFC	*PRED	*RESID	*SRESID	*SDRESID	*LEVER	*COOK D

Source: Regression analysis.

Appendix 12 Predicted Male Crude and Standardized cancer Death Rates, Canada, 1982-2003

Year	Age Effect	Cancer Force	Crude Rate	Standardized Rate	95% Interval (Crude Rate)
1982	20.6	32.0	192.1	171.5	190.2-194.1
1983	18.5	33.2	191.2	172.7	189.3-193.1
1984	15.5	34.3	189.3	173.8	187.4-191.2
1985	19.6	35.4	194.5	174.9	192.6-196.4
1986	17.1	36.6	193.1	176.1	191.2-195.0
1987	15.6	37.7	192.8	177.2	190.9-194.7
1988	11.7	38.8	190.0	178.3	188.1-191.9
1989	11.3	40.0	190.8	179.5	188.9-192.7
1990	10.4	41.1	191.0	180.4	189.1-192.9
1991	7.4	42.2	189.1	181.7	187.1-190.9
1992	3.3	43.3	186.2	182.8	184.3-188.1
1993	3.7	44.5	187.7	184.0	185.1-189.6
1994	4.0	45.6	189.1	185.2	187.2-191.0
1995	4.4	46.7	190.6	186.2	188.7-192.5
1996	4.2	47.9	191.6	187.4	189.7-193.5
1997	1.6	49.0	190.1	188.5	188.2-192.0
1998	3.5	50.1	193.1	189.6	191.2-195.0
1999	2.8	51.3	193.6	190.7	191.7-195.5
2000	2.8	52.4	194.7	191.9	192.8-196.6
2001	3.0	53.5	196.0	193.0	194.1-197.9
2002	2.6	54.6	196.7	194.2	194.8-198.6
2003	2.6	55.8	197.9	195.4	196.0-199.8

Appendix 13 Predicted Female Crude and Standardized Cancer Death Rates, Canada, 1982-2003

Year	Age Effect	Cancer Force	Crude Rate	Standardized Rate	95% Interval (Crude Rate)
1982	39.8	-9.6	150.0	110.2	148.6-151.4
1983	37.1	-10.0	146.9	109.8	145.5-148.3
1984	27.9	-10.4	137.3	109.4	135.9-138.7
1985	36.5	-10.8	145.5	109.0	144.1-146.9
1986	36.2	-11.2	144.8	108.6	143.4-146.2
1987	34.5	-11.6	142.7	108.2	141.3-144.1
1988	29.1	-12.0	136.9	107.8	135.5-138.3
1989	27.3	-12.3	136.8	107.5	135.4-138.2
1990	24.6	-12.7	131.7	107.1	130.3-133.1
1991	20.2	-13.1	126.9	106.7	125.5-128.3
1992	14.5	-13.5	120.8	106.3	119.4-122.2
1993	13.6	-13.9	119.7	105.9	118.3-121.1
1994	12.8	-14.3	118.3	105.5	116.9-119.7
1995	12.7	-14.6	117.9	105.2	116.5-119.3
1996	12.2	-15.0	117.0	104.8	115.6-118.4
1997	10.7	-15.4	115.1	104.4	113.7-116.5
1998	9.6	-15.8	13.61	104.0	112.2-115.0
1999	7.9	-16.2	111.5	103.6	110.1-112.9
2000	7.9	-16.6	111.1	103.2	109.7-112.5
2001	7.8	-17.0	110.6	102.8	109.2-112.0
2002	7.8	-17.3	109.5	102.5	108.1-110.9
2003	6.8	-17.7	108.9	102.1	107.5-110.3

Note: All figures are number of deaths per 100,000 population

Source: From multiple and ridge regression equations

Appendix 14 Correlation Matrix of Variables Used in the Socioeconomic Analysis of Male Cancer Mortality

	e25p 31	e65p 33	mfp 35	lnumi 38	spsco 40	lnsnop 49	no45p 51	finat 54	hincom 57	hincfr 58	hilled 59	hedfre 60	loman 61
e25p 31	1.000												
e65p 33	-0.724	1.000											
mfp 35	0.275	-0.310	1.000										
lnumi 38	-0.103	-0.155	-0.172	1.000									
spsco 40	-0.044	0.084	-0.028	-0.025	1.000								
lnsnop 49	-0.192	0.288	-0.665	0.078	-0.072	1.000							
no45p 51	-0.502	0.802	-0.232	-0.200	-0.070	0.346	1.000						
finat 54	0.225	0.022	0.031	-0.339	-0.132	-0.087	0.114	1.000					
hincom 57	-0.602	-0.475	-0.118	-0.002	-0.109	0.254	-0.220	0.168	1.000				
hincfr 58	0.168	-0.195	0.673	-0.082	-0.072	-0.538	-0.141	0.064	-0.078	1.000			
hilled 59	0.488	-0.331	-0.183	-0.068	-0.005	-0.193	-0.080	0.271	0.537	-0.164	1.000		
hedfre 60	-0.199	-0.175	-0.666	-0.167	-0.062	-0.486	-0.146	0.002	-0.113	-0.724	-0.131	1.000	
loman 61	-0.367	-0.228	-0.318	-0.051	-0.083	0.201	-0.090	-0.232	-0.242	-0.077	-0.132	-0.054	1.000
himan 62	-0.328	-0.194	0.236	-0.247	-0.171	-0.091	-0.002	-0.124	0.251	0.121	-0.155	0.136	-0.498
himan25 69	0.588	-0.398	0.142	-0.172	-0.118	-0.011	-0.217	0.287	0.362	0.221	0.300	0.203	-0.230
loman25 70	0.688	-0.522	0.301	-0.102	0.093	-0.311	-0.420	0.139	0.384	0.023	0.328	0.078	-0.351
hield25 71	0.797	-0.547	0.165	-0.129	-0.084	-0.048	-0.336	0.169	0.484	0.175	0.396	0.313	-0.184
hinco25 72	0.830	-0.620	0.146	-0.063	-0.147	-0.113	-0.448	0.173	0.478	0.266	0.394	0.215	-0.180
hinco45 73	-0.403	0.508	-0.140	-0.235	0.061	-0.165	-0.713	0.138	-0.085	-0.218	0.033	-0.175	-0.147
quemin 74	-0.141	0.029	-0.239	0.503	-0.041	0.133	-0.042	-0.187	-0.007	-0.182	0.085	-0.237	-0.046
ontain 75	-0.114	-0.060	0.108	0.448	0.002	-0.121	-0.062	-0.176	-0.046	-0.148	-0.135	-0.131	-0.022
incomm 82	0.204	-0.184	0.253	-0.165	0.080	-0.317	-0.168	0.284	0.117	0.154	0.088	-0.164	-0.168
westain 83	0.027	-0.219	-0.164	-0.642	-0.012	0.089	-0.242	-0.214	0.034	-0.130	0.063	-0.137	-0.131
hin65 85	-0.627	-0.725	-0.204	-0.200	-0.199	0.192	-0.577	0.086	-0.322	-0.338	-0.201	-0.228	-0.083
fre25 87	0.180	-0.172	0.782	-0.115	-0.024	-0.545	-0.110	0.052	-0.135	0.787	-0.163	0.680	-0.071

	himan 62	himan25 69	loman25 70	hield25 71	hinco25 72	hinco45 73	quemin 74	ontain 75	incomm 82	westain 83	hin65 85	fre25 87
himan 62	1.000											
himan25 69	0.461	1.000										
loman25 70	0.175	0.081	1.000									
hield25 71	0.222	0.620	0.412	1.000								
hinco25 72	0.226	0.634	0.397	0.862	1.000							
hinco45 73	0.043	-0.224	-0.213	-0.348	-0.536	1.000						
quemin 74	-0.295	-0.218	-0.115	-0.182	-0.129	-0.062	1.000					
ontain 75	-0.157	-0.118	-0.070	-0.059	-0.124	-0.067	-0.018	1.000				
incomm 82	0.123	0.046	0.275	0.096	0.077	0.071	-0.090	-0.062	1.000			
westain 83	-0.092	-0.022	-0.028	-0.014	0.089	-0.289	0.034	0.025	-0.156	1.000		
hin65 85	-0.114	-0.432	-0.315	-0.628	-0.770	0.756	0.006	-0.113	0.032	-0.254	1.000	
fre25 87	0.145	0.161	0.091	0.088	0.080	-0.100	-0.213	0.136	0.236	-0.131	-0.136	1.000

Source: Regression analysis. See p.293 for symbols

Appendix 15 Correlation Matrix of Variables Used in the Socioeconomic Analysis of Female Cancer Mortality

	f25p 34	f65p 36	inffp 53	inchil 56	commun 58	hincom 62	hiled 63	hiedf 67	hincf 68	hisa 69	ioma 70	hisaf 71	iomaf 72
f25p 34	1.000												
f65p 36	-0.642	1.000											
inffp 53	0.268	-0.280	1.000										
inchil 56	0.022	-0.575	0.439	1.000									
commun 58	-0.081	-0.146	0.257	0.407	1.000								
hincom 62	0.584	-0.345	-0.082	-0.268	-0.123	1.000							
hiled 63	0.224	-0.030	-0.409	-0.340	-0.186	0.344	1.000						
hiedf 67	0.205	-0.177	0.683	0.302	0.180	-0.049	-0.287	1.000					
hincf 68	0.196	-0.202	0.678	0.308	0.184	-0.035	-0.280	0.652	1.000				
hisa 69	0.273	0.091	0.232	0.148	0.283	0.179	0.093	0.081	0.137	1.000			
ioma 70	-0.328	0.069	-0.280	0.083	0.030	-0.273	0.117	0.116	0.071	0.491	1.000		
hisaf 71	0.173	0.198	0.653	0.215	0.029	0.081	0.321	0.426	0.566	0.265	0.132	1.000	
iomaf 72	0.274	0.139	0.520	0.128	0.205	0.129	0.135	0.371	0.170	0.180	0.362	0.048	1.000
hinc25 73	0.851	0.562	0.167	0.045	0.105	0.409	0.233	0.222	0.281	0.187	0.140	0.182	0.132
hied25 74	0.809	0.560	0.148	0.074	0.058	0.453	0.153	0.287	0.085	0.160	0.243	0.056	0.217
hisa25 75	0.628	0.321	0.181	0.027	0.125	0.324	0.103	0.187	0.272	0.322	0.160	0.253	0.058
ioma25 76	0.630	0.450	0.227	0.095	0.063	0.429	0.088	0.111	0.015	0.177	0.356	0.047	0.383
hinchil 77	0.030	0.407	0.348	0.645	0.213	0.264	0.269	0.386	0.493	0.133	0.075	0.228	0.023
hedchil 78	0.088	0.429	0.316	0.688	0.233	0.204	0.303	0.404	0.309	0.053	0.084	0.206	0.038

	hinc25 73	hied25 74	hisa25 75	ioma25 76	hinchil 77	hedchil 78
hinc25 73	1.000					
hied25 74	0.798	1.000				
hisa25 75	0.672	0.495	1.000			
ioma25 76	0.366	0.542	0.057	1.000		
hinchil 77	0.143	0.034	-0.001	-0.011	1.000	
hedchil 78	0.121	0.150	0.035	0.091	0.670	1.000

Source: Regression analysis.

Notations In Correlation Matrix

Notation	Interpretation
Female	
F25P	% of 25-44
FM45P	% of 45-64
F65P	% of 65+
FFP	% of French
LNCHIL	Child-Woman Ratio
COMMUN	% of Communication Labor Force
HINCOM	High Income Group
HINCF	High Income and % of French
HILED	High Education
HIEDF	High Education and % of French
HIMA	High Manufacturing
LOMA	Low Manufacturing
HIMAF	High Manufacturing and French
LOMAF	Low Manufacturing and French
HIMA25	High Manufacturing and Male 25-44
LOMA25	Low Manufacturing and Male 25-44
HIED25	High Education and Male 25-44
HINC25	High Income and Male 25-44
HINCHIL	High Income and Child-Woman Ratio
HIEDCHIL	High Education and Child-Woman Ratio
Male	
M25P	% of 25-44
NM45P	% of 45-64
M65P	% of 65+
MFP	% of French
LNMMI	% of Mining Labour Force
SOMCO	% of Construction Labor Force
MNOP	% of Norwegians
FINAT	% of Finance Labour Force
HINCOM	High Income Group
HINCFR	High Income and % of French
HILED	High Education
HEDFRE	High Education and % of French
HIMAN	High Manufacturing
LOMAN	Low Manufacturing
HIMAN25	High Manufacturing and Male 25-44
LOMAN25	Low Manufacturing and Male 25-44
HIELD25	High Education and Male 25-44
HINCO25	High Income and Male 25-44
HINCO45	High Income and Male 45-64
HIN65	High Income and Male 65+
QUEMIN	% of Mining In Quebec
ONTMIN	% of Mining In Ontario
WESTMIN	% of Mining In Western Canada
LNCOMM	% of Communication Labour Force
FRE25	% of French 25-44

Note: Variable that are not selected for any subset are excluded.