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Acknowledgements—We thank Nicoletta Susini and Donatella Tanzini for interviewing and coding; Eva Buiatti and Enzo Merler for their comments on the manuscript; and the Italian Association for Research on Cancer (AIRC) and the Italian League for the Fight Against Cancer, Florence branch, for financial support.

Eur J Cancer, Vol. 27, No. 7, pp. 917-921, 1991. Printed in Great Britain 0277-5379/91 \$3.00 + 0.00 © 1991 Pergamon Press plc

# Socioeconomic Status and Cancer Mortality and Incidence in Melbourne

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Data were obtained for all deaths registered between 1979–1983, and for all new cancers recorded at the Victorian Cancer Registry between 1982–1983, in residents of Melbourne. A socioeconomic status (SES) measure had been produced for each local government area (LGA) by principal components analysis of sociodemographic variables recorded at the 1981 census. A SES score from 1 to 10 was assigned to each death and cancer. Population data from the census were similarly scored. Age standardised rates for all cause mortality, for mortality from all causes other than cancer and for both incidence and mortality of total cancers, cancer of the stomach, colon, rectum, lung, female breast, cervix, uterus, prostate and bladder, and for melanoma, lymphoma and leukaemia were analysed as a function of SES decile using weighted linear regression. Despite the limited number of years of data and the misclassification of the SES score, analyses showed there were inequitable distributions of mortality, and of some major cancers, across social strata in Melbourne during the early 1980s. The incidences of cancer of the breast, colon, prostate and melanoma were all positively associated with SES, while the incidences of cancer of the stomach, lung and cervix demonstrated negative SES gradients. For cancers where incidence showed a significant SES gradient there was a similar SES gradient with mortality. These patterns are consistent with the literature and implicate SES differences in education and access to services. Implications for health policy are discussed.

Eur J Cancer, Vol. 27, No. 7, pp. 917–921, 1991

# INTRODUCTION

SINCE THE late nineteenth century, much has been written about the typically negative associations between socioeconomic status (SES) and measures of both health status and mortality [1–5]. This is particularly true of malignant disease, and many associations between specific cancers and SES have been observed in studies from several countries using various measures of SES. The consistency of this evidence has lent credence to the idea that SES has a real association with cancer occurrence and mortality. It is the mechanisms that have remained subject to debate

A review of 28 studies from 11 countries published prior to 1981 [5] showed that all cause mortality and all cancer mortality decreased with increasing SES and that mortality from cancer of the oesophagus, stomach, liver, lung and cervix also decreased with increasing SES, whereas mortality from cancer of the breast, ovary and brain increased with decreasing SES. In the

same review, mortality from cancer of the colon, rectum, skin, uterus, prostate, bladder and kidney, leukaemia and lymphoma varied in its relationship with SES across populations.

Analysis of reports of the Registrar General's Office of England and Wales from 1851 to 1971 on cancer mortality by occupation and social class [5] showed that standard mortality ratios decreased with increasing SES for cancers of the oesophagus, stomach, rectum, lung and skin (other than melanoma) in both men and women, and for prostate cancer in men and for cervical cancer in women. Conversely, standard mortality ratios which increased with increasing SES included cancer of the colon, pancreas, melanoma and brain, leukaemia, lymphoma and myeloma in both men and women and breast and ovary in women.

A relationship between SES and cancer has also been reported in Australian populations. McMichael [6] demonstrated a strong negative correlation between SES and deaths from lung and stomach cancer in Australian males. Siskind et al. [7] investigated all cause mortality in Brisbane and found higher overall mortality rates in lower ranking SES strata. Lung cancer, however, was the only malignancy for which this association was evident. Bonnet et al. [8] examined cancer survival and SES in South Australia. Significantly lower survival in low SES groups was observed for both cancer of the colon and breast cancer.

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Received 2 Apr. 1991; accepted 3 Apr. 1991.

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Although not statistically significant, a similar trend was observed for cancer of the cervix.

The patterns of association between cancer and SES that have been observed in Australian studies are similar to those identified in Europe and the United States. Positive relationships between SES and mortality and incidence from cancers of the breast and colon contrast with the inverse associations between SES and mortality from cancers of the lung, stomach and cervix. Given the possibility that associations between specific malignancies and SES might point to opportunities for cancer control and to priorities in addressing issues of equity in health, it was decided to examine contemporary relationships between SES and cancer mortality and cancer incidence within Melbourne.

## **DATA AND METHODS**

Death certificate data for the Melbourne Statistical Division for the 5 year period 1979–1983 were obtained from the Registrar of Births, Deaths and Marriages. The 1979–1983 quinquennium was chosen because the 1981 census was held at its midpoint. Data from this census were used both for describing the population base and for constructing the SES scale (see below). Each death record included sex, date of death, local government area (LGA) of usual residence within the Melbourne Statistical Division, cause of death coded to the International Classification of Diseases Ninth Revision [9] (ICD-9), and age at death (date of birth is not recorded on death certificates in Victoria). Cancer incidence data for the Melbourne Statistical Division were obtained from the Victorian Cancer Registry for a shorter period 1982–1983 as the registry had only been population based since 1982. Each incidence record contained sex, date of birth, date of diagnosis, LGA of usual residence and site of cancer coded to ICD-9. In addition to mortality from all causes and total cancer the malignancies selected for analysis included stomach, colon, rectum, lung, melanoma, female breast, cervix, body of the uterus and prostate cancers, lymphoma and leukaemia. Age in 5 year groupings was restricted to 40-74 years.

The instrument chosen to measure SES was that of Ross [10]. It summarised a network of interrelated social features which depict the social landscape of a given area. Regions of low SES (or disadvantaged areas, in Ross' terms) typically exhibited high rates of low-status occupations, low income, low educational attainment, high rates of family instability and high concentrations of persons living in low standard housing who were likely to have difficulties with English. Originally, the scale had been developed at the census collector's district level using principal components analysis of variables taken from the 1981 census [11]. Subsequently, LGA values were calculated as weighted averages of collector's district values. The 56 LGAs which comprised the Melbourne Statistical Division were ranked in order of their SES values and were divided into ten groups so that each group contained an approximately equal number of deaths from all causes over the 5 year period. These deciles formed a SES score which took the values of 1 (low) to 10 (high). The same SES cut point values were also used to divide both the base population and the incidence data so that direct comparisons could be made of incidence and mortality rates specific for SES decile.

Age-adjusted incidence and mortality rates were obtained for each cancer and each sex by calculating age-specific rates by 5 year age groups and weighting these by the world standard population [12]. These were then analysed by linear regression using S [13], weighting by the inverse of the within group variance applied over the 10 SES deciles. Similar analyses were

also conducted for mortality from all causes and all causes other than cancer

### RESULTS

The mean age standardised incidence and mortality rates, for all causes, for all causes other than cancer, for total cancer, and for cancers of the stomach, colon, rectum, lung, melanoma, female breast, cervix, body of the uterus and prostate and lymphoma and leukaemia are given in Table 1. In addition, the predicted rates in the first and tenth SES deciles obtained from weighted linear regressions are shown.

A measure of the gradient in risk across the SES deciles is calculated by taking the difference between the predicted rates in the tenth and first SES deciles, dividing by the mean rate and multiplying by 100. The direction, magnitude and statistical significance of these gradients are given.

Of the 48 regressions of SES decile specific rates by cancer and sex and four additional regressions of all-cause mortality and causes other than cancer, 21 are significant at the 5% level and 8 at the 1% level. It is unlikely that all these associations are due to chance.

All cause mortality was shown to be negatively associated with SES (P < 0.001), and the gradient was independent of sex. This negative gradient is more evident for all causes other than cancer. For total cancer the gradient was negative in males (P < 0.01) and was not significant in females. The sex difference in cancer mortality was a reflection of differences in the cancer mix. Lung cancer, the most common cancer in males, had a strong negative gradient. Breast cancer, the most common cancer in females, had a positive gradient.

Negative gradients for both mortality and incidence were evident for cancers of the lung, stomach and cervix. This pattern is illustrated in Fig. 1, using male lung cancer as an example. Positive gradients for incidence but not mortality were observed for cancers of the breast, colon, prostate and melanoma, as illustrated in Fig. 2 for female breast cancer. For none of the cancers analysed were both incidence and mortality strongly and positively associated with SES. Other cancers gave non-significant trends, or inconsistent associations with SES.

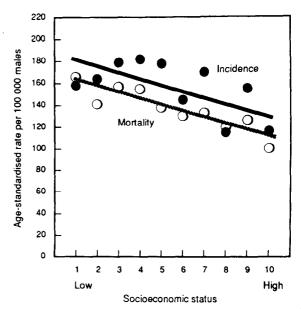


Fig. 1. Regression of SES with lung cancer incidence and mortality in males aged 40-74.

Table 1. Predicted mortality and incidence rates for the lowest and highest deciles of socioeconomic status in people aged 40-74

	Sex	Mortality 1979-1983				Incidence 1982-1983			
		Decile one	Mean	Decile ten	Change	Decile one	Mean	Decile ten	Change
All causes	M F	1137 510	973 436	809 365	-34‡ -33‡				
All causes other than cancer	M F	674 225	541 299	422 97	-47‡ -43‡				
Total cancer	M	453	420	387	−16†	670	654	642	-4
	F	285	277	268	−6	517	547	574	10
Stomach	M	29	27	25	-15	47	42	37	-24
	F	16	12	8	-67‡	21	17	11	-59*
Colon	M	40	40	40	0	57	70	85	40*
	F	31	31	31	0	53	64	73	31*
Rectum	M	22	20	19	-15	50	52	53	6
	F	10	11	11	9	27	32	34	22
Lung	M	163	136	110	-39‡	182	155	126	-36*
	F	38	33	27	-33*	56	48	37	-40*
Melanoma	M	5	<b>9</b>	12	78*	15	35	54	111‡
	F	6	5	3	-60	20	33	44	73‡
Breast	F	57	63	68	17	146	176	203	32‡
Cervix	F	13	10	7	-60*	28	24	20	-33
Uterus	F	5	5	4	-20	29	29	29	0
Prostate	M	19	22	25	27*	67	87	102	40*
Bladder	M F	8	10 3	11 3	30* 0	52 13	58 15	<b>62</b> 17	17 27*
Lymphoma	M	13	16	17	25	30	33	35	15
	F	10	10	10	0	22	22	22	0
Leukaemia	M	9	11	12	27	12	16	20	50
	F	6	7	7	14	10	11	11	9
Other and unspecified	M	20	17	14	-35†	31	31	31	0
	F	12	12	12	0	27	23	16	-48*

Significance levels: \*P < 0.05, †P < 0.01, ‡P < 0.001.

# **DISCUSSION**

The findings here are consistent with the SES associations described in the literature, i.e. that cancers of the lung, stomach and cervix are negatively associated with SES, and cancers of the breast, colon, prostate and melanoma are positively associated with SES [1-8]. The cancer/SES gradients within Melbourne mimicked international cancer differences between western and developing nations; cancer of the stomach and cervix are common in Africa and Asia and cancer of the bowel, breast and prostate are common in developed countries [14]. There are also secular trends in cancer that have been observed over the last century which are consistent with trends in technological development, e.g. the decline in mortality from cancer of the stomach and cancer of the cervix [15]. To these observations can be added the evidence from cancer trends in migrants to western countries from countries of lower economic development. Typically, stomach cancer declines in migrants with increasing duration of residence in a more developed country while cancers of the bowel, breast and prostate increase [16]. Differences in international cancer incidence rates (and in migrants) implicate environmental differences that are amenable

to change [17]. From this it follows that the cancer/SES differentials in Melbourne may not be immutable.

Most lung cancers are caused by tobacco smoking. Both tobacco consumption and lung cancer mortality are declining in Australian men but not in women. As there is no practical method to detect lung cancer early, the sole method of prevention is to reduce smoking, a habit which varies by sex and social stratum [18]. The findings emphasise the need for future preventive strategies in Australia to be effectively targetted at women and low SES populations.

A large proportion (29%) of Melbourne's population aged 40-74 comprises migrants from countries with higher stomach cancer rates than Australia's, and who suffer higher rates of stomach cancer when living in Australia than do the Australian-born population [19]. It is considered that the negative SES gradients with stomach cancer incidence and mortality in this study may be underestimated because of the contributions of certain migrant groups who are at increased risk of stomach cancer and who may live in high SES areas. Declining mortality from stomach cancer in developed parts of the world has been linked to advances in the preservation of fruit and vegetables by

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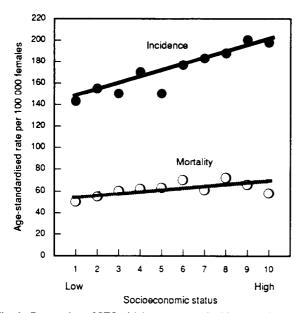


Fig. 2. Regression of SES with breast cancer incidence and mortality in females aged 40-74.

refrigeration rather than by drying, salting and pickling, and the increased consumption of fresh fruit and vegetables that contain protective micronutrients, such as vitamins A and C and carotenoids [20]. Stomach cancer is likely to continue to decline in Australia as dietary intakes and practices change and as first generation migrants, and older cohorts of Australian-born, eventually die. There is no specific public health action required.

Cancer of the cervix is preventable in that treatment following early detection by Pap smears virtually removes the risk of invasive disease [21]. The negative gradients with SES of the incidence and the mortality of cancer of the cervix may relate both to differences with respect to exposure to risk factors, and to a lack of knowledge and use of screening services [22, 23]. Although the risk of cervical cancer increases with the number of a woman's sexual partners, and with the number of her partners' sexual partners [24], from a public health perspective any increase in cervical cancer with lower SES is only important to the extent to which it may be due to a differential utilisation of regular Pap smear tests. Primary prevention via the modification of sexual behaviour is a strategy that is unlikely to be adopted for an uncommon cancer for which there exists a reasonable method of early detection. For cancer of the cervix, the suggestion of a stronger gradient with SES of mortality compared to incidence may indicate that women of low SES delay seeking medical advice and present with more advanced disease. This finding highlights the need for screening programmes to be focused appropriately on women of low SES and on women who have other reasons for diminished access to services. Migrant women from non-English speaking backgrounds and different health cultures may require special attention.

The increase in the incidence of breast cancer with increasing SES may be related to reproductive practices associated with SES, such as delaying first pregnancy, fewer pregnancies and diminished duration of lactation [25]. It may also be related to other lifestyle factors associated with SES, such as overnutrition, through its effect on age at menarche, adiposity and postmeno-

pausal aromatisation of peripheral fat to oestrogens. Colon cancer shares some risk factors with breast cancer. The increased colon cancer rates in high SES groups could be related to lifestyle factors, especially overnutrition and sedentary occupation. Prostate cancer rates are susceptible to the level of diagnostic zeal. As they are strongly age dependent, in ageing western societies these cancers are being increasingly detected [26]. Increased life expectancy and access to regular medical scrutiny are consistent with the observed SES gradients. Melanoma has been linked with SES elsewhere [27]. Unlike non-melanocytic skin cancer, it is increased in office workers and in those who are intermittently rather than chronically exposed to strong sunshine. Melanoma is amenable to early detection by self or medical examination, and early treatment is associated with decreased mortality. These features are consistent with the SES gradients shown in this study.

The salient finding for cancer of the breast, colon, prostate and melanoma is illustrated in Fig. 2—i.e. the strong positive gradient with incidence is not reflected in the mortality gradient. This apparent mortality benefit enjoyed by people of high SES could be due to better treatment. Alternatively, it might be explained by earlier detection, earlier treatment and subsequently improved prognosis. If this latter explanation is the case, SES gradients would be expected to be stronger when analysed by disease stage. An analysis by stage and size of tumour, however, will require appropriately detailed and more data.

Weak and inconsistent SES gradients for some cancer sites in this analysis may be a consequence of misclassification error. Although Najman [28] suggests that in Australia, income variations adjusted for the number of persons in a household, total years of previous education, and occupational status are the most useful measures in assigning SES to individuals, these variables are not available from routine Australian statistical collections. We have used the Ross scale [10] because a value can be assigned to any person on the basis of usual place of residence alone, and this information is available from the Cancer Registry, death certificates and the census. All individuals in the same LGA were given the same SES score because mortality data were only available at LGA level. However, substantial variation in SES across collectors' districts within LGAs indicates considerable heterogeneity [10]. Ascribing a single value to all residents of any one LGA will result in a substantial proportion of individuals being misclassified. Any inaccuracy of addresses recorded on death certificates will compound misclassification. On average, the errors of misclassification will have a conservative effect that attenuates the apparent strength of any real associations with SES.

Problems also exist when assigning SES to migrants because the scale was designed to identify areas of disadvantage with respect to educational needs. As a result, LGAs are given a lower SES score if they have a high proportion of migrants. It is known that migrant groups differ in their cancer mortality patterns from the Australian born population [19] and also that migrants from different countries differ considerably in their overall mortality [29]. This will affect all cause mortality gradients with SES as certain migrants of putative low SES enjoy greater life expectancy on average than some of the Australian-born [29].

The significant SES gradients of incidence and mortality of certain cancers in Melbourne are consistent with other studies. Cancers that demonstrate strong SES gradients within Melbourne are those which vary greatly in incidence between

countries and which demonstrate similar gradients across nations ranked in terms of economic development. Strong international cancer variation is considered to be linked to national differences in lifestyle and medical services. Cancers that show little international variation would not be expected to show intranational variation with SES as these cancers are probably not related to factors amenable to prevention or early detection.

Further work is necessary to clarify the way(s) in which SES in Australia reflects cancer aetiology, early detection or treatment. However, the existing differentials have policy implications for programmes oriented to both prevention and early detection of cancer. Inequities in health and death appear to stem from two related phenomena: health education and access to medical services. Although Medicare provides a basic level of care for all people, medicine is largely a free enterprise system in Australia. In this context, it is unlikely that education alone will improve SES differentials without increasing access to services of Australians with low SES.

The gradients for cancer of the breast and melanoma are consistent with the more educated and higher SES groups being better informed about health, able to act to prevent ill health and able to benefit from early detection. The gradients for cancer of the lung highlight the need for public education programmes to address adequately the smoking problem in lower SES groups. The gradients for cervical cancer emphasise the need for health education and screening delivery programmes to be linked to appropriate social and community networks.

Despite the limited number of years of data and the misclassification of the SES measure, these analyses have shown that there were inequitable distributions of mortality, and of some major cancers, across social strata in Melbourne during the early 1980s. Future analyses will address the persistence or diminution of these inequalities, the role of early detection in explaining the positive gradients with SES, and the success of recent cancer control programmes (mammography, Pap smear registry, smoking cessation and sun protection) which were initiated in Victoria in the late 1980s.

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