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Socio-economic patterning in the incidence and survival of boys and young men diagnosed with testicular cancer in northern England

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Abstract

Purpose

Previous research from developed countries has shown a marked increase in the incidence of testicular cancer in the past fifty years. This has also been demonstrated in northern England, along with improving five-year survival. The aims of the present study were to determine if socio-economic factors may play a role in both aetiology and survival from testicular cancer.

Methods

We extracted all 292 cases of testicular cancer diagnosed in males aged 0-24 years during 1968-2003 from a population-based specialist regional registry. Negative binomial regression was used to examine the relationship between incidence and both the Townsend deprivation score (and component variables) and small-area population density. Cox regression was used to analyse the relationship between survival and both deprivation and population density.

Results

Decreased risk was associated with living in areas of higher household overcrowding (relative risk [RR] per 1% increase in household overcrowding = 0.87; 95% confidence interval [CI] 0.81-0.93). Household overcrowding was also associated with worse survival (hazard ratio per 1% increase in household overcrowding = 1.33; 95% CI 1.19-1.48).

Conclusions

This study has shown that increased risk of testicular cancer is associated with an aspect of more advantaged living. In contrast, greater deprivation confers worse survival prospects.

Introduction

Testicular cancer is relatively rare, accounting for less than two percent of all malignancies in males [1,2]. It mainly affects younger males and is most common in those aged 20–34 years [3]. Since the 1960's, the incidence of testicular cancer has risen markedly in developed countries [4-8]. However, one of these studies suggested that the incidence of non-seminomas, which tend to affect a younger age group, has reached a plateau [7]. The magnitude and uniformity of the observed increases, together with the finding of space-time clustering, suggests a role for environmental or lifestyle factors in aetiology [9,10].

Despite the rise in incidence, survival for boys and young men diagnosed with testicular cancer has greatly improved in recent years and far exceeds survival from other carcinomas [4,10]. However, the possible roles that socioeconomic factors may play in determining survival have not been hitherto explored. In general, survival for most adult cancers has been found to be significantly lower in more deprived areas [11].

In view of previous findings, the aim of this study was to assess geographical variation in incidence and survival of cases of testicular cancer that might arise as a result of environmental or lifestyle factors related to area-level population density and area-level socio-economic deprivation. The following *a priori* hypotheses were tested: a primary factor influencing geographical heterogeneity of incidence of testicular cancer is modulated by differences occurring in (i) less and more densely populated areas of residence; and (ii) less and more socio-economically deprived areas of residence; and survival from testicular cancer is modulated by differences occurring in (iii) less and more densely populated areas of residence; and (iv) less and more socio-economically deprived areas of residence. These were tested using

data from the Northern Region Young Persons' Malignant Disease Registry (NRYPMDR).

Subjects and methods

Cases

Data were included for all patients, aged 0–24 years, registered during the period 1968 to 2003 by the Northern Region Young Persons' Malignant Disease Registry (NRYPMDR). This is a specialist registry, which has recorded all cases of cancer in children and young adults, since its establishment in 1968. It covers the former Northern Region of England, with the exclusion of Barrow-in-Furness (Cumbria) [12]. The registry currently holds details on over 7000 cases of cancer and is housed within the regional specialist centre for this age-group at the Newcastle upon Tyne Hospitals NHS Foundation Trust [13]. Data on children (aged 0 – 14 years) have been obtained prospectively since 1968. Data on teenagers and young adults (aged 15 – 24 years) have been collected retrospectively for the years 1968 – 1985 and prospectively since then [14]. Although registration is not mandatory, cases are identified from a number of sources, including consultants, death certificates and hospital admissions records. Registry data are regularly cross-checked with regional and national cancer registries, thus ensuring a high level of accuracy and completeness, with over 98% ascertainment. Data held include demographic details as well as diagnosis and treatment.

Population data

In this study, analyses were performed at the small-area census ward level. The populations of wards, aged 0-24 years, ranged from 134 to 6142 (median = 1159). During the study period there were four censuses [15-18]. There were also widespread boundary changes throughout this time, especially at small-area level. To derive population estimates, allowing for these perturbations, the data were

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apportioned from the original boundary systems to using the small-area boundaries that applied at the time of the 2001 census [19].

Demographic data

Census ward demographic characteristics were derived from the censuses [15-18]. These characteristics were population density (persons resident per hectare) and the Townsend score for area-based level of deprivation [20], which is a combination of four census measures: unemployment as a percentage of those aged 16 years and over who are economically active, non-car ownership as a percentage of all households, non-home ownership as a percentage of all households and household overcrowding. A time series of Townsend deprivation scores was constructed by allocating these four constituent measures from the 1971, 1981, 1991 and 2001 censuses to the time periods for cancer diagnosis that were closest, i.e. 1968-1975, 1976-1985, 1986-1995 and 1996-2003 respectively, for the 2001 census geography [21]. Increasingly negative Townsend scores represent lower area deprivation (better). Increasingly positive scores represent higher deprivation (worse). Population density was derived using the apportioned populations and then dividing by the areal extent of the 2001 wards.

Statistical analysis

Age-specific incidence rates per million person years were calculated based on mid-year population estimates for males only from the study region obtained from ONS. Age-standardised incidence rates (ASR) were calculated based on the standard world population [22]. Temporal trends for incidence were assessed using Poisson regression with the logarithm of population as an offset. Models considered were:

models linear in time, non-linear in time and models with sinusoidal cyclical variation over time. An assumption of a linear trend was tested by inclusion of a non-linear (categorical) term for time in the model.

There was evidence of extra-Poisson variation: 93.3% of age group specific ward cells had zero counts. Therefore, incidence was modelled at census ward level using negative binomial regression in STATA [23]. The number of cases observed in each census ward was the dependent variable and the logarithm of the underlying population was used as the offset. The ecological (independent) variables were the census-derived ward characteristics, which were allocated to the 2001 census geography [21]. Analysis of survival was performed using Cox regression modelling

[24].

For both incidence and survival, a series of multivariable models were fitted including the following independent variables: age (categorized in two groups as: 0-14 and 15-24 years), sub-type (seminoma, non-seminoma), population density and the Townsend score (as a composite). The following components of the Townsend score were included in separate models that did not include the composite score: unemployment as a percentage of those aged 16 years and over who are economically active, non-car ownership as a percentage of all households, non-home ownership as a percentage of all households and household overcrowding. The interactions between each of age and sub-type and the Townsend score (and its components) and sub-type were also considered for inclusion in the models. Each variable in turn was removed and compared using a likelihood ratio test. Thus, the effect of each variable was assessed by calculating differences in residual deviances and comparing with a chi-square distribution with degrees of freedom (df) equal to the difference in residual degrees of freedom. Model fit was assessed using the

residual deviance for incidence models and minus twice log-likelihood for survival models together with the Akaike information criterion (AIC). Linearity assumptions were tested by including quintiles of significant continuous variables as ordinal variables in the models.

For the analysis of incidence, relative risks (RRs) and associated 95% confidence intervals (CIs) are reported. For the analysis of survival, hazard ratios (HRs) and associated 95% CIs are reported. All *P* values were two-sided and statistical significance was taken as $P < 0.05$ throughout the analyses.

Results

The study included 292 cases of testicular cancer diagnosed aged 0 – 24 years

(comprising 54 cases of seminoma and 238 cases of non-seminoma). There were 29

cases aged 0 – 14 years, 71 cases aged 15 – 19 years and 192 cases aged 20 – 24

years. The ASR over the study period was 13.50 per million persons per year (95%

CI 11.93 to 15.08) for all males aged 0 – 24 years. Case numbers, crude rates and

ASRs by age-group, period and sub-type (dichotomized into seminoma/non-

seminoma) are given in Table 1. Poisson regression showed that there was a

significant annual increase in incidence of 2.8% (95% CI 1.6% to 3.9%) over the

study period, together with a seven year cyclical variation ($P = 0.020$, Figure 1).

As none of the seminoma cases were aged less than 15 years at diagnosis,

this prompted us to use a modified age-sub-type variable comprising the following

three categories: non-seminoma aged 0-14, non-seminoma aged 15-24 and

seminoma. The analysis of deviance and AIC showed that the model fit for all

testicular cancer incidence was significantly improved using the modified variable (P

< 0.001) with higher rates for non-seminoma aged 15-24 and seminoma. Townsend

score (as a composite; $P = 0.007$), and then in separate models with three of its

component variables, was statistically significant, compared with the model

containing age-sub-type (Household overcrowding: $P < 0.001$; Non-home ownership:

$P = 0.031$; and Unemployment: $P = 0.045$). Non-car ownership ($P = 0.106$) and area-

level population density ($P = 0.597$) were not significant compared with the model

containing age and sub-type. The best fitting model contained: modified age-sub-

type and household overcrowding. Additional analysis by quintile of household

overcrowding did not improve model fit. Furthermore interactions of overcrowding

with modified age-sub-type were not significant ($P = 0.575$). Table 2 gives the RRs

for the best fitting model containing age, sub-type and household overcrowding. A statistically significant decreased risk was associated with higher levels of overcrowding (RR for one percent increase in level of household overcrowding = 0.87; 95% CI 0.81 to 0.93).

The analysis of log-likelihood and AIC showed that survival was not related to age (P = 0.226). Survival was related to sub-type however (P = 0.024). After adjustment for sub-type, area-level population density was not significant (P = 0.174). Townsend score (as a composite), and then in separate models with all of its component variables, was statistically significant (Townsend: P < 0.001; Unemployment: P < 0.001; Non-home ownership: P = 0.018; Non-car ownership: P = 0.006; Household overcrowding: P < 0.001). The best fitting model contained sub-type and household overcrowding only. Table 3 gives a comparison of the goodness-of-fit of the different models, assessed using AIC with model 9 denoting the best fitting model. Additional analysis by quintile of household overcrowding (models 10 and 11) did not improve model fit. Furthermore the interaction of overcrowding with type was not significant (P = 0.684). Table 4 gives the HRs for household overcrowding both as a continuous variable and as non-linear quintile (models 9 and 10, respectively). There was a statistically lower risk of death for the seminoma sub-type (HR = 0.36; 95% CI 0.15 to 0.84; P < 0.001). A statistically significant increased risk of death was associated with higher levels of household overcrowding (HR for one percent increase in level of household overcrowding = 1.33; 95% CI 1.19 to 1.48; P < 0.001). The most deprived quintile has a HR of 5.60 (95% CI 1.69 to 18.60), when compared with the least deprived quintile.

Discussion

This is the first analyses of socio-demographic patterning in incidence and survival from testicular cancer. The study has been made possible by the availability of highly accurate and complete cancer registration data from a specialist population-based registry (the NRYPMDR), together with corresponding census population and socio-demographic data. This study has two novel findings: (a) decreased risk of testicular cancer was associated with living in areas of greater household overcrowding; and (b) worse survival from testicular cancer was associated with living in areas of greater household overcrowding.

The results suggest that geographical heterogeneity of incidence is modulated by differences occurring in areas with less and more household overcrowding (reflecting a component of area-level socio-economic deprivation). Thus, there was support for prior hypothesis (ii), but not prior hypothesis (i), since incidence was not related to area-level population density. The results also suggest that geographical heterogeneity of survival is modulated by differences occurring in areas with less and more household overcrowding (again reflecting area-level socio-economic deprivation). Thus, there was support for prior hypothesis (iv), but not prior hypothesis (iii), since survival was not related to area-level population density.

Three methodological caveats must be noted. First, census ward population density and Townsend deprivation scores may not be related to characteristics of individual cases and must only be seen as ecological proxies. These area-level measurements have been allocated to individuals. Caution should be used when extrapolating from grouped data to make inferences about individuals. It is conceivable that there could be unmeasured confounders that exhibit similar patterns of spatial variation [25]. Secondly, case, population and socio-demographic

data were analyzed using 2001 census boundaries. The possible effects of migration were not taken into account. This could have weakened the results. Thirdly, there is at least a theoretical possibility that delays in diagnosis may be related to the demographic variables that have been analysed. Hence, it is possible that there has been a differential loss of some cases related to socio-demographic factors.

A recent cohort study of migrants to Israel found that there were lower rates of testicular cancer among men who were born in North Africa and Asia compared to those born in Europe, but a marked increase in second-generation migrants. This suggests that recent lifestyle and environment are important determinants of risk [26]. Another cohort study, from Denmark, found that risk in first-generation immigrants was lower than native-born Danes, reflecting countries of origin [27]. Environmental factors that have been suggested to be involved in aetiology include occupational exposures to oestrogenic chemicals and maternal exposures to chemicals including polychlorinated biphenyls, hexachlorobenzene and chlordanes [28-31]. Some studies have suggested a link with infections, including HIV, CMV, EBV and SV-40 [32-36]. Exposure to environmental factors is likely to be socially determined.

Recent initiatives in UK by the National Cancer Research Institute (NCRI), the National Cancer Intelligence Network (NCIN) and the National Awareness and Early Diagnosis Initiative (NAEDI) have highlighted the need for early diagnosis of cancer to improve survival [37]. Delays in diagnosis may have an adverse effect on outcomes for patients diagnosed with testicular cancer although with high overall survival impact will be limited. Teenage and young adult males present a particularly neglected group of patients, with low use of health-care resources and late presentation [38]. Delays may be 'patient' or 'professional'. Our findings of worse

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survival linked with social deprivation suggests that either patients from these areas are delaying seeking health advice or general practitioners are slow to refer to a diagnostic centre. Alternatively patients from more deprived areas may be less willing to adhere to treatment protocols.

In conclusion, we have found that lower incidence of testicular cancer was observed in areas associated with higher levels of household overcrowding, indicating that increased risk is linked to some aspect of greater affluence. This suggests that the aetiology may be related to lifestyle factors in early life. We also found that worse survival was seen in areas with higher levels of household overcrowding, indicating that survival is linked with some aspect of social deprivation. This suggests that patients from more deprived areas are less likely to seek early diagnosis or are less likely to adhere to treatment regimens.

Acknowledgements

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TABLE 1

Rates of testicular cancer incidence in northern England by age, period and sub-type during 1968-2003

	N[1]	Male Population years at risk	Crude Rate /	ASR[2]
		(000's)	million	(95% CI[3])
Age				
Ages 0 to 14	29	10911.4	2.66	3.23 (2.16, 4.65)
Ages 15 to 24	263	7986.0	32.93	32.24 (28.34, 36.14)
Period				
1968 – 1976	55	5078.7	10.83	9.73 (7.08, 12.39)
1977 – 1985	62	5024.6	12.34	10.89 (8.09, 13.68)
1986 – 1994	86	4584.0	18.76	15.88 (12.46, 19.31)
1995 – 2003	89	4210.1	21.14	18.95 (15.00, 22.90)
Sub-Type				
Seminoma	54	18897.4	2.86	2.31 (1.69, 2.92)
Non-seminoma	238	18897.4	12.59	11.20 (9.75, 12.65)
Total	292	18897.4	15.45	13.50 (11.93, 15.08)

[1]*N = number of cases*

[2]*ASR = Age-standardised rate*

[3]*CI = Confidence Interval*

TABLE 2

Effect of age and household overcrowding on testicular cancer incidence

Factor	RR[1]	P value
<u>Modelling overcrowding as continuous variable</u>		
Age-sub-type[2]		
Non-seminoma aged 15-24	10.66 (7.09,16.05)	<0.001
Seminoma	2.79 (1.75,4.46)	<0.001
Overcrowding	0.87 (0.81,0.93)	<0.001
<u>Modelling quintile of overcrowding</u>		
Age-sub-type[2]		
Non-seminoma aged 15-24	10.67 (7.09,16.06)	<0.001
Seminoma	2.79 (1.74,4.45)	<0.001
Overcrowding		
Quintile 2	1.30 (0.87,1.94)	0.205
Quintile 3	0.83 (0.55,1.27)	0.402
Quintile 4	0.72 (0.48,1.09)	0.117
Quintile 5	0.61 (0.41,0.91)	0.016

[1]*RR = Relative Risk with 95% confidence interval in brackets*

[2]*Reference group is non-seminoma aged 0-14*

TABLE 3
Comparison of Cox regression models[1]

Model	Factor	AIC[2]
1	Null	
2	Age	645.061
3	Sub-type	641.419
4	Sub-type, Pop density	641.574
5	Sub-type, Townsend	626.852
6	Sub-type, Unemployed	624.563
7	Sub-type, Home not owned	637.850
8	Sub-type, No cars	635.751
9	Sub-type, Overcrowding	620.875
10	Sub-type, Overcrowding Quintiles (non linear)	629.596
11	Sub-type, Overcrowding Quintiles (continuous)	624.685
12	Sub-type, Overcrowding, Overcrowding*Seminoma	622.709

[1]Sub-type = Non Seminoma, Seminoma

[2]AIC = Akaike Information Criterion

TABLE 4

Effect of household overcrowding on testicular cancer survival, modelled as a continuous variable (model 9) and by non-linear quintile (model 10)

Model		HR[1]	P value
9	Seminoma	0.36 (0.15,0.84)	0.018
	% overcrowding	1.33 (1.19,1.48)	<0.001
10	Seminoma	0.41 (0.17,0.95)	0.037
	Overcrowding		
	Quintile 2	1.32 (0.31,5.52)	0.707
	Quintile 3	2.98 (0.81,11.02)	0.102
	Quintile 4	4.62 (1.35,15.81)	0.015
	Quintile 5	5.60 (1.69,18.60)	0.005

[1]HR = hazard ratio with 95% confidence intervals in parentheses

FIGURE 1

Variation over time in crude rates for all cases of testicular cancer aged 0 – 24 years[1]
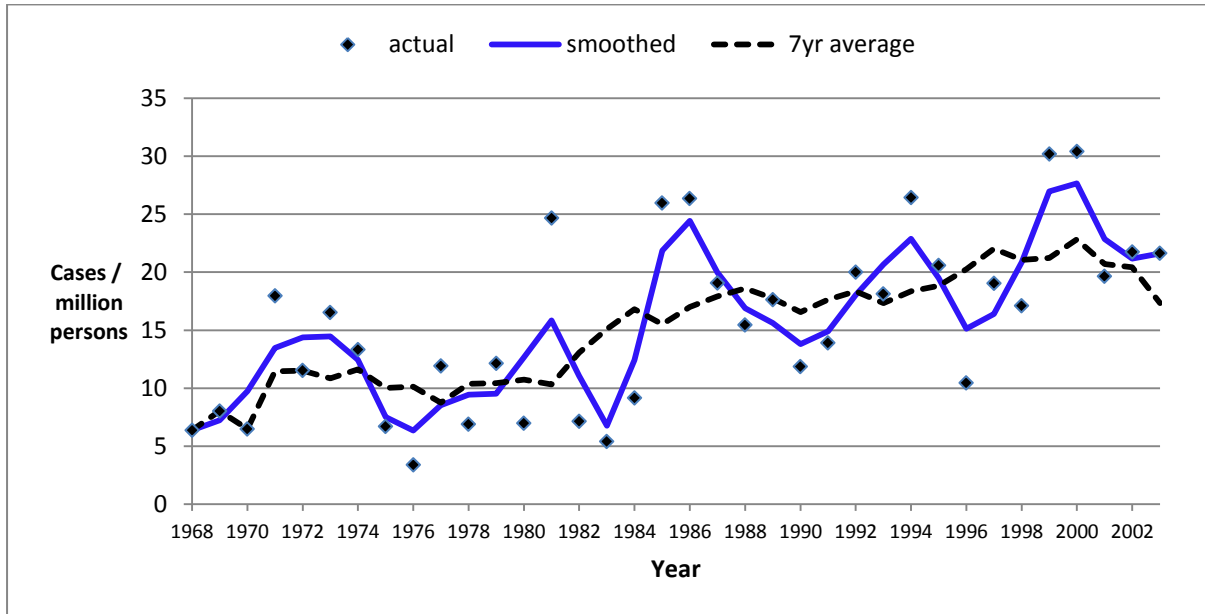
1. Smoothed values based on moving average with weights in the ratio 1:2:1.

FIGURE 2
Survival of testicular cancer cases by quintile of overcrowding

Survival by quintile of overcrowding