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Original article

Socioeconomic patterning in the incidence and survival of teenage and young adult men aged between 15 and 24 years diagnosed with non–seminoma 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 50 years. This has also been demonstrated in northern England, along with improving 5-year survival. The present study aims to determine if socioeconomic factors may play a role in both etiology and survival from non–seminoma testicular cancer.

Materials and methods: We extracted all 214 cases of non–seminoma testicular cancer diagnosed in teenage and young adult men aged between 15 and 24 years during 1968 to 2006 from the Northern Region Young Persons' Malignant Disease Registry, which is 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 analyze the relationship between survival and both deprivation and population density.

Results: Decreased incidence was associated with living in areas of higher household overcrowding for young adults aged between 20 and 24 years (relative risk per 1% increase in household overcrowding = 0.79; 95% CI: 0.66–0.94) but no association was detected for young people aged between 15 and 19 years. Community-level household unemployment was associated with worse survival (hazard ratio per 1% increase in household unemployment = 1.04; 95% CI: 1.00–1.08).

Conclusions: This study has shown that increased risk of non–seminoma testicular cancer in teenage and young adult men may be associated with some aspect of more advantaged living. In contrast, greater deprivation is linked with worse survival prospects. The study was ecological by design and so these area-based results may not necessarily apply to individuals.

Keywords: Socioeconomic factors; Epidemiology; Incidence; Survival; Testicular neoplasms

1. Introduction

Testicular cancer is relatively rare, accounting for less than 2% of all malignancies in men, mainly affecting younger men [1,2]. Since the 1960s, the incidence of testicular cancer has risen markedly in developed countries. However, more recently the incidence of non–seminoma testicular cancer, which tends to affect a younger age group, has reached a plateau [3–5]. The magnitude and uniformity of the observed increases, together with the finding of space-time clustering [6,7], suggests a role for environmental or lifestyle factors in etiology.

Despite the rise in incidence, survival from testicular cancer has greatly improved in recent years and far exceeds survival from other carcinomas [1,7–9]. In general, survival for most adult cancers has been found to be significantly lower in more deprived areas [10]. A previously published review considered 63 studies that examined the role of socioeconomic status on the incidence and survival from

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testicular cancer; overall more advantaged socioeconomic status was associated with greater incidence and better survival [11]. However, 1 case-control study from the UK found no association between socioeconomic status and risk of testicular cancer [12]. Another study from the UK of all testicular cancer (diagnosed up to 2001) found worse survival associated with greater deprivation [13]. However, the possible roles that socioeconomic factors may play in determining survival have not been hitherto explored for teenage and young adult men (aged 15–24 y) with non–seminoma testicular cancer in the UK.

In view of previous findings, the aim of this study was to assess geographical variation in incidence and survival of cases of non–seminoma testicular cancer that might arise as a result of environmental or lifestyle factors related to area-level population density and area-level socioeconomic deprivation. The following a priori hypotheses were tested: a primary factor in uencing geographical heterogeneity of incidence of non–seminoma testicular cancer is modulated by differences occurring in (i) less and more densely populated areas of residence; (ii) less and more socio-economically deprived areas of residence; survival from non–seminoma testicular cancer is modulated by differences occurring in (iii) less and more densely populated areas of residence; and (iv) less and more socioeconomically deprived areas of residence. These were tested using data from the Northern Region Young Persons' Malignant Disease Registry (NRYPMDR).

2. Materials and methods

2.1. Case data

Data were included for all patients with non–seminoma testicular cancer, aged between 15 and 24 years at time of diagnosis, registered during the period 1968 to 2006 by the 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). The region is ethnically homogeneous with fewer than 2% from minorities [14–16]. There are low rates of migration into or out of the region [17–19]. The registry currently holds details on over 7,000 cases of cancer and is housed within the regional specialist center for this age group at the Newcastle upon Tyne Hospitals National Health Service (NHS) Foundation Trust. Data on children (aged 0–14 y) have been obtained prospectively since 1968. Data on teenagers and young adults (aged 15–24 y) have been collected retrospectively for the years 1968 to 1985 and prospectively since then [20]. 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. Data held include demographic details as well as diagnosis and treatment. The registry is exempted (originally under Section 60 of the UK Health and Social Care Act 2001, which has now been superseded by Section 251 of the NHS Act 2006) from the need to obtain patient consent for recording and analysis of data.

2.2. Population data

In this study, analyses were performed at the small-area census ward level. The populations of wards, aged between 15 and 24 years, ranged from 45 to 4,396 (median = 463). During the study period there were 4 censuses. 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 apportioned from the original boundary systems to using the small-area boundaries that applied at the time of the 2001 census [21].

2.3. Demographic data

Census ward demographic characteristics were derived from the censuses. These characteristics were population density (persons resident per hectare) and the Townsend score for area-based level of deprivation [22], which is a combination of 4 census measures: unemployment (as a percentage of those aged 16 y and over who are economically active) and noncar ownership, nonhome ownership, and household overcrowding (each as a percentage of all households). A time series of Townsend deprivation scores was constructed by allocating these 4 constituent measures from the 1971, 1981, 1991, and 2001 censuses to the time periods for cancer diagnosis that were closest, i.e., 1968 to 1975, 1976 to 1985, 1986 to 1995, and 1996 to 2006, respectively, for the 2001 census geography [23]. 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.

2.4. Statistical analysis

Age-specific incidence rates per million person years were calculated based on mid-year population estimates for men only from the study region obtained from the Office for National Statistics. Age-standardized incidence rates (ASR) were calculated based on the standard world population [24]. Temporal trends for incidence were assessed using Poisson regression with the logarithm of population as an offset.

There was evidence of extra-Poisson variation: 95.0% of age group specific ward cells had zero counts. Therefore, incidence was modeled at census ward level using negative