Cancer Among New Zealand Adolescents and Young People 1988–2002

An occasional paper
The image on the front cover is *Farbstudie Quadrate mit konzentrischen Ringen* ('Colour study of squares with concentric rings'), 1913, oil on canvas, by Wassily Kandinsky (1866–1944), held by the Städtische Galerie im Lenbachhaus, Munich.

This image was chosen as a metaphor for adolescence, a time of multiple and changing roles and spheres of influence. It also reflects the great diversity of adolescents and young people.

The image was also chosen to represent the multidisciplinary approach, with many interconnected and co-ordinated parts, required to deliver effective adolescent health care.

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Foreword

Cancer is a leading cause of morbidity, disability and death for New Zealanders, and is a priority issue for the New Zealand health system. In 2003 the New Zealand Cancer Control Strategy was launched and in 2005 the Action Plan for 2005–2010 was published, setting objectives for implementation of the Strategy.

This paper describes the pattern of cancer incidence and survival among New Zealand adolescents and young people. It is an important paper, not only because of its comprehensive overview, but also because its scholarship and timeliness enables it to inform the work of the Paediatric/Adolescent subgroup of the New Zealand Cancer treatment working party. The paper utilises the International Classification of Childhood Cancer which permits direct comparison between New Zealand and published overseas data.

Dr Greg Williams undertook this work, as part of his advanced training in paediatrics, during a six-month attachment to the Ministry of Health. The content of this paper does not necessarily represent the Ministry of Health’s viewpoint, but is intended to guide policy regarding adolescent cancer treatment and prevention. The intended audience for this paper includes policy makers within the Ministry of Health and other relevant government departments, District Health Board funders and planners, and clinicians working in the field of cancer control.

Dr Pat Tuohy
Chief Advisor Child and Youth Health
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Abstract

Objectives

This study aims to describe the pattern of cancer incidence and survival among New Zealand adolescents and young people, discuss the importance of adolescent development, and discuss the future of adolescent cancer management in light of current literature.

Methods

Data were extracted from the New Zealand Cancer Registry, along with matched mortality data, for new cases of cancer diagnosed among those aged between 10 and 24 years over a 15-year period from 1988–2002. These cases were analysed and grouped using the International Classification of Childhood Cancer (ICCC), with further analysis using an adolescent-specific classification scheme. Cases were also analysed based on gender, ethnicity and deprivation. Five-year survival was calculated. International comparisons were made where possible.

Results

The study included 2917 cases of cancer and a total of 12,452,820 person-years at risk, giving a crude incidence rate for 10–24-year-olds of 234 per million person-years. The patterns of cancer were broadly similar to those reported internationally in this age group, except for a higher incidence of malignant melanoma than most countries with which comparisons were made, apart from Australia. Females had a slightly lower incidence of cancer than males. Overall incidence in Māori was lower than that in non-Māori. At older ages this difference was partly due to lower rates of melanoma among Māori. The study was not able to demonstrate differences in incidence based on deprivation.

Conclusions and recommendations

The high incidence of melanoma emphasises the importance of primary preventative measures to reduce ultraviolet radiation exposure.

The absence of an adolescent-focused cancer service in New Zealand has led to current work to develop such a service. Ideally this service will take account of both the medical and developmental needs of adolescents and young people. A service of this nature will include access to appropriately supported and networked clinicians able to deliver adolescent-specific care, facilities appropriate for adolescents, developmentally appropriate psychosocial support, assistance with care co-ordination, access to adolescent-appropriate clinical trials, culturally supportive services, and long-term follow-up services. In addition, moves to improve access to primary health services may assist in early diagnosis, with subsequently improved outcomes.

Monitoring progress is also vital. An extension of the Children’s Cancer Registry to include older adolescents and young people would be a pragmatic way to achieve this.

Finally, the legislation could be amended to include surveillance of benign central nervous system tumours. These tumours are not currently included under the legislation, although some cases probably do get entered into the Registry. Due to their location, their effect can be as devastating as that of a malignant tumour.
Introduction

New Zealand context

The New Zealand Cancer Control Strategy was launched in 2003 for the purposes of reducing the incidence and impact of cancer and reducing inequalities with respect to cancer (Minister of Health 2003). In 2005 the Action Plan for 2005–2010 was published, setting objectives for implementation of the Strategy (Cancer Control Taskforce 2005). Goal 3, Objective 4 of the Action Plan is to ‘Improve the quality of care delivered to adolescents with cancer and their family and whanau’. To gain a picture of adolescent cancer incidence and survival in New Zealand so that planning for the implementation of this objective could proceed, this epidemiological study was commissioned.

There is no comprehensive population-wide epidemiological description of adolescent cancer in New Zealand. Cancer incidence and survival have been studied in the paediatric population in New Zealand (Becroft et al 1999; Dockerty et al 1996, 1997; Douglas and Dockerty 2005), and in the adolescent age group internationally (Birch et al 2002, 2003; Bleyer 2002; Cotterill et al 2000; Desandes et al 2004; Gatta et al 2003; Ries et al 1999; Smith et al 1999; Stiller 2002; Wu et al 2003). The New Zealand Health Information Service (NZHIS) publishes summary results across all age groups annually (NZHIS 2004); however these analyses are presented by the anatomical site of the cancer, which is generally more suited to cancers that affect adults, rather than those that affect children and young people. In addition, given that cancer is relatively uncommon at younger ages, a single year’s data provide only a limited picture.

International context

Adolescence has been defined in numerous ways, without any one of them being universally accepted. Its definition may be based on age or developmental stage, and often differs depending on the reason for the distinction. The World Health Organization defines early adolescence as 10–14 years of age and late adolescence as 15–19 years. Other definitions include the teenage years, or the period from onset to completion of puberty, or later. One suggested definition encompasses many of these principles (Santrock 2005):

Adolescence: the developmental period of transition from childhood to early adulthood; it involves biological, cognitive, and socioemotional changes.

For the purposes of this study, people aged between 10 and 24 years at diagnosis were included. This allowed analysis in three five-year age ranges (10–14 years, 15–19 years, and 20–24 years). These years span the transition period when the incidence of cancer steadily increases from a lifetime low at around 7–8 years of age. The types of cancer move from those more common in children (such as embryonal tumours) to those more common in adults (such as malignant melanoma). In addition there is a group of cancers (such as testicular cancer) that reach close to peak lifetime incidence in the older part of this age range.
Cancer registries worldwide store cancer information using the International Classification of Diseases coding schemes (ICD-10, ICD-O), which use a combination of anatomical site and histology to categorise cancers. With cancer being relatively uncommon in the young, a method of logically grouping similar types of cancer can provide a more useful overall representation. The approach used in this paper is similar to that adopted elsewhere, involving the use of the International Classification of Childhood Cancer (Steliarova-Foucher et al 2005) combined with a scheme specific to adolescents and young adults (Birch et al 2002) for further analysis of a subset of cancer types more common in adolescents. The use of this method allows comparison between New Zealand results and those reported in other countries.
Defining Features of Cancer in Adolescents and Young Adults

A number of characteristics help to define the experience of adolescents with cancer. These include the biological aspects of cancer that are unique to adolescents. Of equal importance is the developmental transition that adolescents make from child to adult. While these factors are relevant individually, the biology and treatment of cancer in adolescents often interact with development in complex and fluctuating ways. To provide some context for the discussion of adolescent development that follows, some elements of this interaction are outlined below.

Disease factors

A distinct range of cancers affects adolescents and young adults. Classic paediatric cancers such as acute lymphoblastic leukaemia and brain tumours still feature prominently, while bone cancers peak in the teenage years. Incidence of cancers with a strong environmental influence, such as malignant melanoma and cervical carcinoma, starts to rise sharply. The earliest cases of cancers associated with later adulthood, such as breast and bowel cancer, appear. Thyroid cancer becomes increasingly common, especially in females, while testicular cancer is at close to peak lifetime incidence by the early 20s.

Overall, cancer incidence in 15–19-year-olds appears to be slowly increasing in Western populations, by about 1 percent per year (Cotterill et al 2000; Smith et al 1999). Incidence rates in some regions are heavily influenced by local factors, such as the burgeoning numbers of cases of Kaposi sarcoma in sub-Saharan Africa associated with the AIDS epidemic, or the increase in thyroid carcinoma in parts of eastern Europe that were exposed to high levels of radioactive fallout from the Chernobyl nuclear reactor disaster in 1986. The highest rates of malignant melanoma in the world have previously been noted to occur in Australasia, presumably as a result of high levels of ultraviolet radiation exposure acting on a population that includes a large number of fair-skinned individuals due to historical immigration patterns (Gandini et al 2005; Jones et al 1999). The incidence of some other cancers is also affected by ethnic differences within a population. For example, US black adolescents have a lower incidence of Ewing’s sarcoma, testicular germ cell tumours, melanoma, acute lymphoblastic leukaemia, and thyroid cancer compared with US whites (Smith et al 1999).

A minority of cancers affecting adolescents have established aetiologies. Apart from those cancers with established environmental risk factors (such as malignant melanoma and cervical carcinoma), and a small number of cancers affecting individuals with genetic or chromosomal abnormalities, the remainder of cancers do not have a clear environmental cause (National Cancer Institute 2005). Adolescent behaviour (such as smoking) does have a bearing on cancer incidence later in life, however.

Outcomes for adolescents and young adults with cancer have improved over time. Five-year survival of patients diagnosed with cancer at 15–19 years of age in the US has gone from 69 percent in 1975–1984 to 77 percent in 1985–1994. Despite this, survival improvements for adolescents with some types of cancer (eg, leukaemia) have not matched the survival improvements for younger children (Albritton and Bleyer 2003; Bleyer 2002; Haase and Phillips 2004).
The differences in outcomes are undoubtedly due to multiple factors (Albritton and Bleyer 2003). Cancer in this age group may demonstrate biological differences, and treatments may have a different impact on adolescents. Poorer outcomes have also been attributed to a failure of specific recognition of this section of the population with regard to cancer, and low rates of clinical trial enrolment (Bleyer et al. 1997; Cole 2004; McTiernan 2003). Enrolment in clinical trials appears to improve outcome. Because of the relatively low numbers of adolescents with cancer, trial access requires a large degree of co-ordination both nationally and between paediatric and adult oncology services (McTiernan 2003).

Ethnicity can affect outcome as well. For example, in the US, black and Hispanic young people have significantly worse five-year survival than white and Asian children do (Kadan-Lottick et al. 2003). Again, the reasons are likely to be multifactorial, but probably part of the explanation is that black and Hispanic young people tend to have poorer access to cancer services as the difference in survival rates is seen in relation to other diseases too (Easterbrook et al. 1991; Shi and Stevens 2005). In New Zealand, ethnic disparities in health outcomes have been well described (Blakely et al. 2005); they include worse survival for Māori patients with cancer across all ages above 14 years (Jeffreys et al. 2005).

Adolescent development

Adolescence encompasses a wide range of physical, psychological, cognitive and social developments. The timing and pace of these developments are highly variable between individuals. As young people define their identity and future role in society, they move from a position of relative dependence to relative independence. The independence is by no means absolute, as relationships with family, peers and others take on a new form.

Understanding the many facets of adolescent development is essential to providing appropriate health services to this population. The processes described below are an explanation of normal development; however, the experience and outcome for an individual may be very different.

Physical development

Adolescence is characterised by marked physical growth and pubertal maturation. Girls tend to start and finish this process earlier than boys. Ethnicity appears to affect timing of puberty as well; for example, the onset is earlier in US black and Hispanic young people than it is for US white young people (Sun et al. 2002; Wu et al. 2002).

For girls, breast development starts from around 8 years of age, followed by pubic hair growth, and rapid growth in height. Menstruation usually starts about two years after initial pubertal changes, which can be anywhere from 10–15 years. The rate of height gain usually peaks at about 12, and is complete by 14–15 years. Pubertal changes are usually complete at a similar time.

For boys, the earliest changes include scrotal and testicular enlargement from 9 years of age, followed closely by lengthening of the penis. Rapid growth in height starts at around 10–11 years, with pubic hair developing from around 12 years. Growth velocity peaks at about 14, and tapers off at 16–18 years. Pubertal changes are usually complete by that age (Greene 2005).
Psychosocial and cognitive development

During adolescence, young people establish a sense of identity, increasingly exercise autonomy, form intimate relationships with peers, and reach sexual maturity. An integral part of this process involves cognitive changes – changes in the way adolescents think and conceptualise themselves and others. This period also marks a change in responsibilities. Adolescents make decisions about future education and career, and some exercise their reproductive potential and produce dependants of their own. The process of change tends to be haphazard rather than smooth; it is common for adolescents to test and re-test new constructs before a series of equilibria is reached.

Moving from an established identity within a family context, the peer group becomes increasingly important as the young person defines their own identity. Much of this happens without conscious deliberation, and does not occur in isolation. Young people integrate their peer group’s opinions as they mould their own likes and dislikes. They do not cut all ties with their family, but redefine the nature of the relationships. Adolescents form a clearer view of their cultural and spiritual identity, and develop their own moral code.

Adolescents start to form intimate relationships outside their family. These start as close friendships within the peer environment, allowing them to develop their social skills. Eventually their intimate relationships expand to include romantic relationships. As they sexually mature, and form a sexual identity, adolescents may engage in sexual relationships. Around a quarter of New Zealand 14-year-olds report that they have had sexual intercourse at some time; this proportion rises to half by age 17 (Adolescent Health Research Group 2003).

With the physical and pubertal changes, adolescents become acutely aware of their own appearance. Their intense self-scrutiny is accompanied by an assumption that everyone else is equally scrutinising them, leading to some of the self-awareness, detachment and embarrassment in adolescents that others often observe.

A key cognitive transformation is the move from concrete to abstract thought. Children tend to think in a fixed, concrete framework. As they age, their cognitive capacity increases, and adolescents become able to consider more complex concepts, hypothesise, and start questioning established norms. As young people reach adulthood they become able to see perspectives other than their own. These cognitive changes allow the adolescent to become increasingly autonomous (Huebner 2000; Ozretich and Bowman 2001).

Effect of adolescent development on cancer diagnosis and treatment

An adolescent negotiating their way through this multitude of changes may find it difficult to incorporate a life-threatening diagnosis into their worldview. A limited capacity for abstract thought may explain the late presentation and poor compliance demonstrated by some in this age group. A sense of indestructibility (‘it won’t happen to me’) can lead to a lack of concern about the future consequences of decisions made now.

Effect of cancer on adolescent development

The range of implications that a diagnosis of cancer has for adolescents and young people differs from the implications for children or older adults. The cancer itself or associated treatment can
affect physical growth and pubertal development. The impact of cancer and treatment can also interrupt psychosocial and cognitive development. In some adolescents, there may be regression to an earlier level of dependence; in others, there may be accelerated emotional development as they come to terms with such an unexpected life-changing event.

Many young people will be living away from family. An increasing proportion will be engaged in employment or tertiary education. As they get older, some will have families of their own, and some may be the main income-earner for themselves and their family. Their role in life is becoming established, but is threatened by the implications of cancer.

Treatment may have significant effects on both body and body image at a time when peer acceptance is vital. The short-term physical effects may be clearly visible, such as hair loss, weight gain, or loss of a limb. There may also be psychological effects that are less visible. A young person may feel distanced from their healthy, ‘normal’ peers at a stage in life where they are seeking their place in the world, and their niche amongst their peers.

Long-term physical effects are also significant. Certain types of cancer and treatment can have considerable impact on growth, puberty, and reproductive potential, either through altered hormonal function or the direct effects of malignancy, radiotherapy or chemotherapy.

**Implications for adolescent cancer services**

Adolescence presents a unique set of developmental circumstances that influence and are influenced by cancer diagnosis and treatment. An adolescent-oriented service should allow for the developmental implications of adolescence and, where possible, enable and support developmental progression across all domains: physical, pubertal, psychological and social.

These unique factors are the rationale for many to advocate for the establishment of cancer services specifically for adolescents and young people, with a particular focus on:

- provision of developmentally appropriate and ongoing psychosocial support (Carr-Gregg and Hampson 1986; Sawyer et al 1986; Searle et al 2003)
- improved access to and enrolment in clinical trials (Bleyer 1997, 2002; Bleyer et al 1997; Hammond et al 1993; McTiernan 2003).
Objectives

This study aims to describe the pattern of cancer incidence and survival among New Zealand adolescents and young people, discuss the importance of adolescent development, and discuss the future of adolescent cancer management in light of current literature.
Materials and Methods

Ethical approval

The New Zealand Multi-Region Ethics Committee approved the project.

Data sources

Cancer data were extracted from the New Zealand Cancer Registry (NZCR) with the assistance of NZHIS. The NZCR was set up in 1948 primarily using information sent by public hospitals. The Cancer Registry Act 1993 and Cancer Registry Regulations 1994 were introduced to improve reporting of cancers in New Zealand. Prior to 1994 cancers that did not require public hospital treatment tended not to be registered. For younger age groups, most primary cancer diagnoses will have been made in the public system. For older age groups, the incidence of some cancers – for example, malignant melanoma – was probably understated (Jones et al 1999). The exact effect of this change in reporting is unknown, but is likely to underestimate total incidence of less serious cancers for the older age group for the pre-1994 period of this study.

NZHIS frequently checks and audits the cancer registry to maintain data quality. The validity of data held by the NZCR has been examined externally in a study of childhood cancer for the period 1990–1993 (Dockerty et al 1997). Notably the Cancer Registry Act was not yet in force in this period. The study compared multiple sources of data including the NZCR to confirm the accuracy and completeness of registration, as well as pathology review of all available specimens to check the accuracy of histological diagnosis. The study found that the NZCR captured 97 percent of confirmed cases. However, it also reported an extra 10 percent of registrations that were included due to coding error, duplication or incorrect pathological diagnosis; the majority of the overcount occurred because of miscoding of benign tumours as malignant.

Data validity and completeness across all cancers for this age group have not been checked externally in a similar fashion since the Cancer Registry Act came into effect. It is likely that data quality and completeness have improved due to the legislative requirements. However, without the facility to undertake the multiple source data check and pathology review of the nature undertaken by Dockerty et al, the present study may still include some degree of overcount.

To reduce error and overcounting, data were checked using a variety of methods. In addition, trends over the period of the study were analysed to see if there were any significant changes to reporting patterns for common types of cancer. These checks are discussed in detail in Appendix 1. In brief, they involved excluding in situ/non-invasive cancers, checking and removing duplicate registrations, checking and validating dates of birth, and applying a range of validity checks to diagnostic data using specialist software. This process revealed a small number of apparent errors, which were communicated back to NZHIS.
Benign central nervous system tumours are generally not registered with the New Zealand Cancer Registry as they fall outside of the definition of cancer within the Cancer Registry Act. While these tumours may have significant consequences for an individual, they are generally not included in this study. Some tumours with borderline malignant/benign behaviour (such as juvenile pilocytic astrocytoma) may be entered into the NZCR if they are coded as malignant. If entered, they are included in the study.

All cancer registrations recorded in the NZCR for the period 1 January 1988 to 31 December 2002 were extracted. In addition, data held in the public hospital discharge database (National Minimum Dataset – NMDS) and the Mortality collection were extracted for all individuals with cancer registrations.

The cancer registrations may include some non-New Zealand residents, for example, children from Pacific Island nations transferred to New Zealand for treatment. It was not possible to identify these cases separately, and so incidence and survival data will include them. The effect is likely to be small, given that Dockerty et al discovered one case in this category in their population-based study of 409 children with cancer in New Zealand (Dockerty et al 1997). Anecdotally, numbers in this category have increased more recently, and patients are often referred with advanced disease (Scott MacFarlane, personal communication, 2005). Outcomes for these individuals if they return to their home country are generally not known.

**Coding**

The NZCR codes cancer information using an Australian modification of the International Classification of Diseases (ICD). The current version used in the NZCR is ICD-10-AM-II (NCCH 2000). Historical cancer registrations coded in older revisions of ICD have been mapped to ICD-10 codes by NZHIS. There is a separate but related version of ICD for oncology diagnoses, called ICD-O. For the purposes of analysing data in this study, the codes were converted from ICD-10-AM-II to ICD-O version 2 (ICD-O-2), then to version 3 (ICD-O-3) using the International Agency for Research on Cancer conversion program (Ferlay et al 2005). The ICD-O-2 codes were used to classify cancers using the Birch scheme for adolescents (Birch et al 2002). The ICD-O-3 codes were used to classify cancers using the International Classification for Childhood Cancer, 3rd edition (ICCC-3). ICCC-3 has only recently been published, and is recommended as the standard for classification of childhood cancer (Steliarova-Foucher et al 2005). A few cases did not clearly fit into the ICCC-3 scheme. These were clarified with one of the ICCC-3 authors and allocated accordingly (Eva Steliarova-Foucher, 2005, personal communication).

Population data for calculation of incidence rates were obtained from Statistics New Zealand (1989–2003, 2005c, 2005d). Estimated mean annual population figures based on census data were obtained for each age group, for each year of the study, by gender and by Māori/non-Māori ethnicity. These are presented in Appendix 3.
Statistical analysis

The data were analysed using Epi Info, Microsoft Access and Excel, and SURV3 (CDC 2005; Microsoft Corporation 1999a, 1999b; Voutilainen et al 2002). As this study used population data, incidence rates represent the true population rates for the period of the study. However, the data can be considered as a sample in time, and incidence rates therefore as an estimate of the incidence over a longer period. On this basis, confidence intervals for incidence rates were calculated assuming the cases were drawn from a Poisson distribution. Comparison of incidence rates between groups was done using a version of the Mantel-Haenszel test (Pearce 2003).

Survival was calculated using actuarial methods. Comparison of survival between groups was done using Cox proportional hazard modelling, and the Logrank test (Campbell and Machin 1999). Death notifications are likely to be almost complete, due to automatic notification of deaths by the Office of Births, Deaths and Marriages to NZHIS. Deaths that occur outside New Zealand may be missed; in addition, for survivorship, calculations were censored (assumed alive) at 30 June 2005. The size of these effects is unknown, but is likely to be small.

Relative survival was calculated based on New Zealand Life Tables (Statistics New Zealand 1998, 2004). Given the survivorship of the general population for this age range (>99 percent five-year survival for 10–24-year-olds), survival rates of those diagnosed with cancer differ from the crude all-cause survival figures by less than 1 percent (see Appendix 3 for the actuarial five-year survival for the population aged 10–24 in 2000–2002).

Calculating survival based on cancer-specific mortality is also possible, using the cause of death as reported in the death certificate. This information is held by NZHIS for deaths up to 31 December 2002. The validity of this alternative is debatable, mainly because of concerns about the accuracy of death certificate reporting. A second reason is that some apparent non-cancer causes of death may actually be cancer related – for example, suicide following a diagnosis of cancer. For both these reasons the relative survival approach was used.

For further details about statistical methods, see Appendix 5.

Ethnicity

Ethnicity of individuals within this study was obtained using the ethnicity as recorded in the National Health Index. A prioritisation algorithm (known as the ‘ever-Māori’ approach) classed individuals as: ‘Māori’ if they had identified as Māori during any hospital inpatient episode (either solely or along with other ethnicities); ‘Pacific’ if they had ever identified with one of the Pacific ethnicities unless they were already classed as Māori; or ‘Other’ for all others. Due to small numbers of Pacific individuals, Other and Pacific were grouped together as ‘non-Māori’ for analysis and comparison of incidence rates and survival.

The reliability of New Zealand ethnicity data has been discussed elsewhere (Jeffreys et al 2005), and the New Zealand Census-Mortality Study has noted the significant undercounting of Māori ethnicity based on death notification forms (Ajwani et al 2003; Blakely et al 2002a, 2002b). Using the ever-Māori approach reduces the undercounting effect.

For statistical purposes, the definition of Māori ethnicity based on census questionnaires has altered over time. For this reason, following the 2001 census, Statistics New Zealand (2005b)
retrospectively recalculated estimated annual mean Māori population as far back as 1991. The approach taken (to include anyone who self-identifies as Māori either singly or among other ethnicities) is broadly similar to the prioritisation approach described above, allowing incidence rates to be calculated for Māori.

Prior to 1991 the mean annual population figures available for Māori ethnicity are based entirely on a ‘sole Māori’ definition – that is, they include only those people identifying with Māori ethnicity in the Census. For this reason, where Māori incidence rates are calculated and compared, only cases from 1991 are included.

**Deprivation**

To estimate the influence of deprivation, a New Zealand-specific deprivation indicator (NZDep) was assigned to each cancer registration based on where the person lived at time of diagnosis. This approach has been used in other studies accessing similar source data (McFadden et al 2004).

The NZDep2001 Index of Deprivation (Salmond and Crampton 2002b) combines dimensions of deprivation from New Zealand census data, and assigns a score to small geographically defined groups of the population known as meshblocks. Meshblocks vary in size from zero to over 300 persons, with a median of 90. A similar process produced NZDep96 and NZDep91 (Crampton et al 1997; Salmond et al 1998).

The NZCR uses census area unit, an aggregation of meshblocks with 3000–5000 persons in each, to code geographical location. The population-weighted average deprivation of the meshblocks within each census area unit can be used to determine an NZDep score.

The use of geographical location as a marker of deprivation is liable to error and misinterpretation, as discussed elsewhere (McLoone 2001; Salmond and Crampton 2001, 2002a), particularly because an individual living within an area of deprivation may not be deprived themselves, and vice versa. The aggregation of meshblocks into larger census area units, as done in this study, compounds this effect. Therefore the analyses using deprivation scores in this report should be interpreted with caution.

The deprivation score of a census area may change over time as the sociodemographic characteristics of that area change. To account for these changes, the deprivation scale in use closest to the time of diagnosis was assigned. Scores for census area units using NZDep91 were not available. For this reason, comparisons involving deprivation were limited to cases occurring from 1994 to 2002. Deprivation scores were therefore assigned using NZDep96 for 1994 to 1998, and NZDep2001 for 1999 to 2002.

To avoid the interpretation problems identified above, a new deprivation indicator, the NZiDep, has recently been developed and validated for individuals within the New Zealand population (Salmond et al 2005). Unfortunately, the New Zealand Cancer Registry does not hold the degree of personal detail required to assign an NZiDep score. The Children’s Cancer Registry, if modified and expanded to include adolescents, may hold sufficient detail to assign an NZiDep score.
Basis of diagnosis

The majority of cancers notified to the NZCR are diagnosed pathologically. In the period under study, a small number were notified only via death certificate, as shown in Table 1. These were included in analyses due to the importance of capturing all cancers, including those presenting at death.

Table 1: Basis of diagnosis

<table>
<thead>
<tr>
<th>Basis of diagnosis</th>
<th>Number of cases</th>
<th>Percentage of total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Histology of primary tumour</td>
<td>2440</td>
<td>83.6</td>
</tr>
<tr>
<td>Cytology or haematology</td>
<td>365</td>
<td>12.5</td>
</tr>
<tr>
<td>Histology of metastases</td>
<td>52</td>
<td>1.7</td>
</tr>
<tr>
<td>Clinical investigation</td>
<td>29</td>
<td>1.0</td>
</tr>
<tr>
<td>Death certificate only</td>
<td>13</td>
<td>0.4</td>
</tr>
<tr>
<td>Clinical only</td>
<td>9</td>
<td>0.3</td>
</tr>
<tr>
<td>Autopsy with concurrent or previous histology</td>
<td>9</td>
<td>0.3</td>
</tr>
<tr>
<td>Total</td>
<td>2917</td>
<td>100</td>
</tr>
</tbody>
</table>
Results: Cancer Incidence

Key findings

For young people aged 10–14 years at diagnosis:

- there were 530 cases of cancer for the 15-year period of this study, producing an overall incidence of 129 per million person-years (95 percent confidence interval [CI] 119–141)
- the most common groups of cancer in descending order are leukaemias, CNS tumours, lymphomas and bone tumours, together comprising approximately 70 percent of all cancer cases in this age group
- lymphoid leukaemia is the most common single type of cancer, followed by astrocytoma and osteosarcoma.

For young people aged 15–19 years at diagnosis:

- there were 875 cases of cancer for the 15-year period, producing an overall incidence of 208 per million person-years (95 percent CI 195–222)
- the most common groups of cancer in descending order are melanomas, lymphomas, leukaemias, germ cell tumours, and bone tumours, together comprising 70 percent of all cancer cases in this age group
- malignant melanoma is the most common single type of cancer, followed by Hodgkin lymphoma, lymphoid leukaemia and gonadal germ cell tumours.

For young people aged 20–24 years at diagnosis:

- there were 1512 cases of cancer for the 15-year period, producing an overall incidence of 366 per million person-years (95 percent CI 347–384)
- the most common groups of cancer in descending order are melanomas, germ cell tumours, lymphomas and leukaemias, together comprising 65 percent of all cancer cases in this age group
- malignant melanoma is the most common single type of cancer, followed by gonadal germ cell tumours, Hodgkin lymphomas and thyroid carcinomas.

Compared with other developed countries, the overall rate of cancer is slightly higher (for example, rates for 15–19-year-olds in Western populations have been reported in the range of 150–200 per million person-years, compared with 208 in New Zealand). This difference can be explained entirely by the excess burden of malignant melanoma in the New Zealand population.

Overall age-specific incidence rates are shown in Figure 1. Incidence rates categorised using child and adolescent classification schemes are shown in Tables 2 and 3.
Figure 1: Age-specific incidence rates for all cancers by age at diagnosis, 1988–2002, with 95 percent confidence intervals.

Table 2: Case numbers and age-specific incidence rates categorised using the ICCC-3

<table>
<thead>
<tr>
<th>ICCC category</th>
<th>Age at diagnosis (years)</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>10–14 Cases</td>
<td>10–14 IR*</td>
<td>15–19 Cases</td>
</tr>
<tr>
<td>I Leukaemias, myeloproliferative diseases, and myelodysplastic diseases</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>a Lymphoid leukaemias</td>
<td>120</td>
<td>29.3</td>
<td>132</td>
<td>31.4</td>
</tr>
<tr>
<td>b Acute myeloid leukaemias</td>
<td>83</td>
<td>20.2</td>
<td>76</td>
<td>18.1</td>
</tr>
<tr>
<td>c Chronic myeloproliferative diseases</td>
<td>30</td>
<td>7.3</td>
<td>42</td>
<td>10.0</td>
</tr>
<tr>
<td>e Unspecified and other specified leukaemias</td>
<td>4</td>
<td>1.0</td>
<td>2</td>
<td>0.5</td>
</tr>
<tr>
<td>II Lymphomas and reticuloendothelial neoplasms</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>a Hodgkin lymphomas</td>
<td>32</td>
<td>7.8</td>
<td>86</td>
<td>20.4</td>
</tr>
<tr>
<td>b Non Hodgkin lymphomas (except Burkitt lymphoma)</td>
<td>28</td>
<td>6.8</td>
<td>33</td>
<td>7.8</td>
</tr>
<tr>
<td>c Burkitt lymphoma</td>
<td>10</td>
<td>2.4</td>
<td>8</td>
<td>1.9</td>
</tr>
<tr>
<td>d Miscellaneous lymphoreticular neoplasms</td>
<td>1</td>
<td>0.2</td>
<td>1</td>
<td>0.2</td>
</tr>
<tr>
<td>e Unspecified lymphomas</td>
<td>2</td>
<td>0.5</td>
<td>5</td>
<td>1.2</td>
</tr>
<tr>
<td>III CNS and miscellaneous intracranial and intraspinal neoplasms</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>a Ependymomas and choroid plexus tumour</td>
<td>2</td>
<td>0.5</td>
<td>5</td>
<td>1.2</td>
</tr>
<tr>
<td>b Astrocytomas</td>
<td>58</td>
<td>14.1</td>
<td>38</td>
<td>9.0</td>
</tr>
<tr>
<td>c Intracranial and intraspinal embryonal tumours</td>
<td>19</td>
<td>4.6</td>
<td>5</td>
<td>1.2</td>
</tr>
<tr>
<td>d Other gliomas</td>
<td>13</td>
<td>3.2</td>
<td>10</td>
<td>2.4</td>
</tr>
<tr>
<td>e Other specified intracranial and intraspinal neoplasms</td>
<td>4</td>
<td>1.0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>f Unspecified intracranial and intraspinal neoplasms</td>
<td>10</td>
<td>2.4</td>
<td>8</td>
<td>1.9</td>
</tr>
<tr>
<td>ICCC category</td>
<td>Age at diagnosis (years)</td>
<td>10–14 Cases</td>
<td>IR*</td>
<td>15–19 Cases</td>
</tr>
<tr>
<td>---------------</td>
<td>--------------------------</td>
<td>-------------</td>
<td>-----</td>
<td>-------------</td>
</tr>
<tr>
<td>IV Neuroblastoma and other peripheral nervous cell tumours</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>a Neuroblastoma and ganglioneuroblastoma</td>
<td>14</td>
<td>3.4</td>
<td>6</td>
<td>1.4</td>
</tr>
<tr>
<td>b Other peripheral nervous cell tumours</td>
<td>3</td>
<td>0.7</td>
<td>2</td>
<td>0.5</td>
</tr>
<tr>
<td>V Retinoblastoma</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>VI Renal tumours</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>a Nephroblastoma and other non-epithelial renal tumours</td>
<td>4</td>
<td>1.0</td>
<td>8</td>
<td>1.9</td>
</tr>
<tr>
<td>b Renal carcinomas</td>
<td>1</td>
<td>0.2</td>
<td>5</td>
<td>1.2</td>
</tr>
<tr>
<td>c Unspecified malignant renal tumours</td>
<td>2</td>
<td>0.5</td>
<td>2</td>
<td>0.5</td>
</tr>
<tr>
<td>VII Hepatic tumours</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>a Hepatoblastoma</td>
<td>5</td>
<td>1.2</td>
<td>12</td>
<td>2.9</td>
</tr>
<tr>
<td>b Hepatic carcinomas</td>
<td>1</td>
<td>0.2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>c Unspecified malignant hepatic tumours</td>
<td>4</td>
<td>1.0</td>
<td>12</td>
<td>2.9</td>
</tr>
<tr>
<td>VIII Malignant bone tumours</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>a Osteosarcomas</td>
<td>66</td>
<td>16.1</td>
<td>93</td>
<td>22.1</td>
</tr>
<tr>
<td>b Chondrosarcomas</td>
<td>40</td>
<td>9.8</td>
<td>54</td>
<td>12.8</td>
</tr>
<tr>
<td>c Ewing tumour and related sarcomas of bone</td>
<td>2</td>
<td>0.5</td>
<td>2</td>
<td>0.5</td>
</tr>
<tr>
<td>d Other specified malignant bone tumours</td>
<td>23</td>
<td>5.6</td>
<td>27</td>
<td>6.4</td>
</tr>
<tr>
<td>e Unspecified malignant bone tumours</td>
<td>1</td>
<td>0.2</td>
<td>1</td>
<td>0.2</td>
</tr>
<tr>
<td>IX Soft tissue and other extraosseous sarcomas</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>a Rhabdomyosarcomas</td>
<td>40</td>
<td>9.8</td>
<td>58</td>
<td>13.8</td>
</tr>
<tr>
<td>b Fibrosarcomas, peripheral nerve sheath tumours, and other fibrous neoplasms</td>
<td>12</td>
<td>2.9</td>
<td>24</td>
<td>5.7</td>
</tr>
<tr>
<td>c Kaposi sarcoma</td>
<td>2</td>
<td>0.5</td>
<td>6</td>
<td>1.4</td>
</tr>
<tr>
<td>d Other specified soft tissue sarcomas</td>
<td>1</td>
<td>0.2</td>
<td>1</td>
<td>0.2</td>
</tr>
<tr>
<td>e Unspecified soft tissue sarcomas</td>
<td>19</td>
<td>4.6</td>
<td>17</td>
<td>4.0</td>
</tr>
<tr>
<td>X Germ cell tumours, trophoblastic tumours, and neoplasms of gonads</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>a Intracranial and intraspinal germ cell tumours</td>
<td>42</td>
<td>10.2</td>
<td>111</td>
<td>26.4</td>
</tr>
<tr>
<td>b Malignant extracranial and extragonadal germ cell tumours</td>
<td>15</td>
<td>3.7</td>
<td>10</td>
<td>2.4</td>
</tr>
<tr>
<td>c Malignant gonadal germ cell tumours</td>
<td>1</td>
<td>0.2</td>
<td>9</td>
<td>2.1</td>
</tr>
<tr>
<td>d Gonadal carcinomas</td>
<td>23</td>
<td>5.6</td>
<td>69</td>
<td>16.4</td>
</tr>
<tr>
<td>e Other and unspecified malignant gonadal tumours</td>
<td>3</td>
<td>0.7</td>
<td>18</td>
<td>4.3</td>
</tr>
<tr>
<td>XI Other malignant epithelial neoplasms and malignant melanomas</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>a Adrenocortical carcinomas</td>
<td>57</td>
<td>13.9</td>
<td>232</td>
<td>55.1</td>
</tr>
<tr>
<td>b Thyroid carcinomas</td>
<td>4</td>
<td>1.0</td>
<td>29</td>
<td>6.9</td>
</tr>
<tr>
<td>c Nasopharyngeal carcinomas</td>
<td>32</td>
<td>7.8</td>
<td>160</td>
<td>38.0</td>
</tr>
<tr>
<td>d Malignant melanomas</td>
<td>2</td>
<td>0.5</td>
<td>5</td>
<td>1.2</td>
</tr>
<tr>
<td>e Other and unspecified carcinomas</td>
<td>1</td>
<td>0.2</td>
<td>2</td>
<td>0.5</td>
</tr>
<tr>
<td>XII Other and unspecified malignant neoplasms</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>a Other specified malignant tumours</td>
<td>4</td>
<td>1.0</td>
<td>24</td>
<td>5.7</td>
</tr>
<tr>
<td>b Other unspecified malignant tumours</td>
<td>3</td>
<td>0.7</td>
<td>24</td>
<td>5.7</td>
</tr>
<tr>
<td>Total for all cancers</td>
<td>530</td>
<td>129.2</td>
<td>875</td>
<td>207.8</td>
</tr>
</tbody>
</table>

Note: * IR = incidence rate per million person-years.
Table 3: Cases and age-specific incidence rates for ICCC category XI using the Birch classification scheme for adolescents

<table>
<thead>
<tr>
<th>Birch category</th>
<th>Age at diagnosis (years)</th>
<th>10–14</th>
<th>15–19</th>
<th>20–24</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Cases</td>
<td>IR*</td>
<td>Cases</td>
<td>IR*</td>
</tr>
<tr>
<td>7.1 Melanoma</td>
<td>32</td>
<td>7.8</td>
<td>160</td>
<td>38.0</td>
</tr>
<tr>
<td>8.1 Thyroid carcinoma</td>
<td>4</td>
<td>1.0</td>
<td>29</td>
<td>6.9</td>
</tr>
<tr>
<td>8.2 Other carcinoma of head and neck</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8.2.1 Nasopharyngeal carcinoma</td>
<td>2</td>
<td>0.5</td>
<td>5</td>
<td>1.2</td>
</tr>
<tr>
<td>8.2.2 Other sites in lip, oral cavity and pharynx</td>
<td>5</td>
<td>1.2</td>
<td>11</td>
<td>2.6</td>
</tr>
<tr>
<td>8.2.3 Nasal cavity, middle ear, sinuses, larynx and other and ill defined head and neck</td>
<td>1</td>
<td>0.2</td>
<td>4</td>
<td>1.0</td>
</tr>
<tr>
<td>8.3 Carcinomas of trachea, bronchus and lung</td>
<td>2</td>
<td>0.5</td>
<td>2</td>
<td>0.5</td>
</tr>
<tr>
<td>8.4 Carcinoma of breast*</td>
<td>1</td>
<td>0.5*</td>
<td>28</td>
<td>13.6*</td>
</tr>
<tr>
<td>8.5 Carcinoma of genitourinary (GU) tract</td>
<td>3</td>
<td>0.7</td>
<td>2</td>
<td>0.5</td>
</tr>
<tr>
<td>8.5.2 Carcinoma bladder</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8.5.4 Carcinoma of cervix and uterus*</td>
<td>4</td>
<td>1.9*</td>
<td>38</td>
<td>18.4*</td>
</tr>
<tr>
<td>8.5.5 Carcinoma of other and ill-defined sites in GU tract</td>
<td>3</td>
<td>0.7</td>
<td>3</td>
<td>0.7</td>
</tr>
<tr>
<td>8.6 Carcinoma of gastro-intestinal (GI) tract</td>
<td>8</td>
<td>2.0</td>
<td>7</td>
<td>1.7</td>
</tr>
<tr>
<td>8.6.1 Carcinoma of colon and rectum</td>
<td>1</td>
<td>0.2</td>
<td>4</td>
<td>1.0</td>
</tr>
<tr>
<td>8.6.4 Carcinoma pancreas</td>
<td>1</td>
<td>0.2</td>
<td>3</td>
<td>0.7</td>
</tr>
<tr>
<td>8.6.5 Carcinoma of other and ill-defined sites in GI tract</td>
<td>1</td>
<td>0.2</td>
<td>1</td>
<td>0.2</td>
</tr>
<tr>
<td>8.7 Carcinomas of other and ill-defined sites not elsewhere classified (NEC)</td>
<td></td>
<td>1</td>
<td>0.2</td>
<td>2</td>
</tr>
<tr>
<td>8.7.1 Adrenocortical carcinoma</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8.7.2 Carcinoma of other and ill-defined sites</td>
<td>1</td>
<td>0.2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total for ICCC category XI</td>
<td>57</td>
<td>13.9</td>
<td>232</td>
<td>55.1</td>
</tr>
</tbody>
</table>

Notes:
* IR = incidence rate per million person-years.
# Female only rates.
Source: Birch et al 2002.

Time trends for incidence

Key time trends found in this study are that:

- the relatively small number of cases of cancer per year in these age groups is reflected in the degree of fluctuation year to year
- over the period studied, overall incidence appears to be stable.

Figure 2 shows the annual incidence rates for all types of cancer. This graph demonstrates how the rates can fluctuate widely from year to year when there are relatively small numbers of cases of cancer in each age group annually. Despite reports elsewhere that cancer incidence is increasing over time in the 15–19-year age group (Bleyer 2002; Stiller 2002), the results from this study did not demonstrate this trend. There are a number of possible explanations for this
including that: there was no change; there were not enough cases to show a trend; or the period covered by the study may not have been long enough.

**Figure 2:** Age-specific incidence rates for all cancers by age and year of diagnosis, 1988–2002

![Incidence rate graph](image)

**Demographics**

Key demographic findings are that:

- more males than females were diagnosed with cancer for each age group over the period of the study
- the incidence of cancer rises markedly with age at diagnosis
- cancer is more common in non-Māori than in Māori for each age group.

**Gender differences in incidence**

Figure 3 shows the overall cancer incidence for each age group. While male rates exceeded female rates at each age, the difference was not significant for all diagnoses combined. Table 4 shows male and female incidence rates for ICCC categories, and rate ratios where there are more than 10 cases in each comparison group. The incidence of some types of cancer at some ages occurred at significantly different rates in males and females; these are also shown in Table 4.
Figure 3: Age-specific incidence rates for all cancers by age at diagnosis and gender, 1988–2002, with 95 percent confidence intervals.
Table 4: Comparisons between gender age-specific incidence rates

<table>
<thead>
<tr>
<th>ICCC category</th>
<th>10–14</th>
<th>15–19</th>
<th>20–24</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Female</td>
<td>Male</td>
<td>IRR</td>
</tr>
<tr>
<td>I Leukaemias, myeloproliferative diseases, and myelodysplastic diseases</td>
<td>23.0</td>
<td>35.2</td>
<td>1.5</td>
</tr>
<tr>
<td>II Lymphomas and reticuloendothelial neoplasms</td>
<td>13.5</td>
<td>21.4</td>
<td>1.6</td>
</tr>
<tr>
<td>III CNS and miscellaneous intracranial and intraspinal neoplasms</td>
<td>23.0</td>
<td>28.6</td>
<td>1.2</td>
</tr>
<tr>
<td>IV Neuroblastoma and other peripheral nervous cell tumours</td>
<td>2.0</td>
<td>4.8</td>
<td>1.4</td>
</tr>
<tr>
<td>V Retinoblastoma</td>
<td>0.5</td>
<td></td>
<td></td>
</tr>
<tr>
<td>VI Renal tumours</td>
<td>1.0</td>
<td>1.0</td>
<td></td>
</tr>
<tr>
<td>VII Hepatic tumours</td>
<td>1.0</td>
<td>1.4</td>
<td></td>
</tr>
<tr>
<td>VIII Malignant bone tumours</td>
<td>16.0</td>
<td>16.2</td>
<td>1.0</td>
</tr>
<tr>
<td>IX Soft tissue and other extraosseous sarcomas</td>
<td>11.5</td>
<td>8.1</td>
<td>0.7</td>
</tr>
<tr>
<td>X Germ cell tumours, trophoblastic tumours, and neoplasms of gonads</td>
<td>15.0</td>
<td>5.7</td>
<td>0.4</td>
</tr>
<tr>
<td>XI Other malignant epithelial neoplasms and malignant melanomas</td>
<td>16.0</td>
<td>11.9</td>
<td>0.7</td>
</tr>
<tr>
<td>XII Other and unspecified malignant neoplasms</td>
<td>1.0</td>
<td>1.0</td>
<td></td>
</tr>
<tr>
<td>All cancers</td>
<td>122.9</td>
<td>135.2</td>
<td>1.1</td>
</tr>
</tbody>
</table>

Notes

* IR = incidence rate per million person-years.
** IRR = incidence rate ratio, expressed as rate of male IR to female IR.
† = p < 0.05.
# = p < 0.01; all other differences non-significant (p > 0.05).

The higher rate of category I (leukaemia, etc) diagnoses in 15–19-year-old males is due to excess lymphoblastic leukaemia in this group (M:F ratio = 4.0). The higher rate of category VIII (bone) tumours among 20–24-year-old males is because both osteosarcoma and Ewing sarcoma are more common in males in this group. The higher rate of category X (germ cell and gonadal) tumours for 10–14-year-old females is due to the rate of ovarian germ cell tumours (10.0 per million person-years), and the higher rate for 20–24-year-old males is due to the rate of testicular cancer in this group (90.6 per million person-years). The higher rate of category XI (Other malignant epithelial neoplasms and malignant melanomas) among 20–24-year-old females is because of greater rates of melanoma (M:F ratio = 0.6) and thyroid carcinoma (M:F ratio = 0.3), and because breast and cervical cancer (13.6 and 18.4 per million person-years respectively) are also included in this group.

Location at time of diagnosis

The local District Health Board (DHB), or its predecessor, where each individual was resident at the time of diagnosis was assigned by NZHIS according to address information that it held.
Total cancers across all ages from 10–24 years at diagnosis were summed and the relative proportions are shown in Figure 4, with comparison to the 10–24-year-old population within each DHB taken from the mean of the 1991, 1996 and 2001 censuses (Statistics New Zealand 2005a). This graph shows the geographic spread of cases generally matches the population spread. In other words, overall incidence does not appear to vary by region. Incidence for certain types of cancer may vary by region; however, due to small numbers of cases of each type in each region, this analysis was not done.

**Figure 4:** Resident DHB at time of diagnosis, 10–24-year-olds, 1988–2002

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**Treatment location and treating service**

One of the original intentions of this study was to describe where adolescents with cancer were treated, and by whom. Fulfilling this purpose was not possible for a number of reasons. First, the National Minimum Dataset of hospital discharge data captures only inpatient visits, and therefore misses a large proportion of interactions between patients and clinicians. Secondly, while diagnoses and certain treatments are coded within the NMDS using the ICD, it is not always clear if a specific visit is related to the cancer diagnosis. Thirdly, there is great variation in how the treating service is coded, so that attempts to determine the type of service on a particular visit (adult or paediatric, oncology or non-oncology) are fraught with inaccuracies and produce unreliable results.

**Ethnicity differences in incidence**

Ethnicity within the NZCR data was assigned as Māori, Pacific and non-Māori/non-Pacific as discussed in the ‘Materials and methods’ section. The total number of cases of cancer among each ethnic group is shown in Figure 5. The actual number of cases split between Māori and non-Māori for the whole period is shown in Table 5.
Figure 5: Total number of cases by age at diagnosis and ethnicity, 1988–2002

Table 5: Total number of cases by Māori/non-Māori ethnicity, 1988–2002

<table>
<thead>
<tr>
<th>Age at diagnosis (years)</th>
<th>Māori total</th>
<th>Non-Māori total</th>
<th>Grand total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Female</td>
<td>Male</td>
<td>Female</td>
</tr>
<tr>
<td>10–14</td>
<td>40</td>
<td>33</td>
<td>73</td>
</tr>
<tr>
<td>15–19</td>
<td>42</td>
<td>68</td>
<td>110</td>
</tr>
<tr>
<td>20–24</td>
<td>67</td>
<td>113</td>
<td>180</td>
</tr>
<tr>
<td>Grand total</td>
<td>149</td>
<td>214</td>
<td>363</td>
</tr>
</tbody>
</table>

The overall incidence rate for each age group including and excluding melanoma is shown in Table 6 (using data from 1991–2002 only, as discussed in the ‘Materials and methods’ section). Rates are significantly lower for Māori than for non-Māori, although this difference disappears in 20–24-year-olds when melanoma is excluded.

Table 6: Age-specific incidence rates for Māori and non-Māori including and excluding melanoma, 1991–2002

<table>
<thead>
<tr>
<th>Age at diagnosis (years)</th>
<th>Māori</th>
<th>Non-Māori</th>
<th>IRR**</th>
<th>p'</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Cases</td>
<td>IR*</td>
<td>Cases</td>
<td>IR</td>
</tr>
<tr>
<td>10–14</td>
<td>58</td>
<td>80.4</td>
<td>363</td>
<td>139.8</td>
</tr>
<tr>
<td>All cancer</td>
<td>Excluding melanoma</td>
<td>58</td>
<td>80.4</td>
<td>337</td>
</tr>
<tr>
<td>15–19</td>
<td>88</td>
<td>134.2</td>
<td>592</td>
<td>223.7</td>
</tr>
<tr>
<td>Age</td>
<td>Type</td>
<td>Excluding melanoma</td>
<td></td>
<td></td>
</tr>
<tr>
<td>------</td>
<td>------</td>
<td>--------------------</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td></td>
<td></td>
<td>83</td>
<td>126.6</td>
<td>470</td>
</tr>
<tr>
<td>20–24</td>
<td>All cancer</td>
<td>146</td>
<td>255.4</td>
<td>1065</td>
</tr>
<tr>
<td></td>
<td>Excluding melanoma</td>
<td>145</td>
<td>253.7</td>
<td>714</td>
</tr>
</tbody>
</table>

Notes:
* IR = incidence rate per million person-years
** IRR = incidence rate ratio (expressed as ratio of Māori IR to non-Māori IR)
† p = two-tailed test of significance

The rates of some types of cancer seem to differ between Māori and non-Māori populations, as shown in Table 7 (again, for 1991–2002 data only).
### Table 7: Age-specific incidence rates for Māori and non-Māori, 1991–2002

<table>
<thead>
<tr>
<th>Cancer type (ICCC classification)</th>
<th>10–14 years</th>
<th></th>
<th>15–19 years</th>
<th></th>
<th>20–24 years</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Māori</td>
<td>Non-Māori</td>
<td>IRR**</td>
<td>p</td>
<td>Māori</td>
<td>Non-Māori</td>
</tr>
<tr>
<td></td>
<td>Cases</td>
<td>IR*</td>
<td>Cases</td>
<td>IR</td>
<td>Cases</td>
<td>IR</td>
</tr>
<tr>
<td>I Leukaemias, myeloproliferative diseases, and myelodysplastic diseases</td>
<td>14</td>
<td>19.4</td>
<td>86</td>
<td>33.1</td>
<td>0.59</td>
<td>0.064</td>
</tr>
<tr>
<td>a Lymphoid leukaemias</td>
<td>10</td>
<td>13.9</td>
<td>61</td>
<td>23.5</td>
<td>0.59</td>
<td>0.121</td>
</tr>
<tr>
<td>b Acute myeloid leukaemias</td>
<td>2</td>
<td>2.8</td>
<td>20</td>
<td>7.7</td>
<td>0.36</td>
<td>0.168</td>
</tr>
<tr>
<td>c Chronic myeloproliferative diseases</td>
<td>3</td>
<td>4.6</td>
<td>4</td>
<td>1.5</td>
<td>3.03</td>
<td>0.147</td>
</tr>
<tr>
<td>e Unspecified and other specified leukaemias</td>
<td>2</td>
<td>2.8</td>
<td>2</td>
<td>0.8</td>
<td>3.60</td>
<td>0.201</td>
</tr>
<tr>
<td>II Lymphomas and reticuloendothelial neoplasms</td>
<td>7</td>
<td>9.7</td>
<td>49</td>
<td>18.9</td>
<td>0.51</td>
<td>0.099</td>
</tr>
<tr>
<td>a Hodgkin lymphomas</td>
<td>26</td>
<td>10.0</td>
<td>6</td>
<td>9.2</td>
<td>0.697</td>
<td>0.36</td>
</tr>
<tr>
<td>b Non Hodgkin lymphomas (except Burkitt lymphoma)</td>
<td>6</td>
<td>8.3</td>
<td>18</td>
<td>6.9</td>
<td>1.20</td>
<td>0.697</td>
</tr>
<tr>
<td>c Burkitt lymphoma</td>
<td>1</td>
<td>1.4</td>
<td>5</td>
<td>1.9</td>
<td>0.72</td>
<td>0.764</td>
</tr>
<tr>
<td>d Miscellaneous lymphoreticular neoplasms</td>
<td>1</td>
<td>1.4</td>
<td>1</td>
<td>0.4</td>
<td>1</td>
<td>0.4</td>
</tr>
<tr>
<td>e Unspecified lymphomas</td>
<td>1</td>
<td>1.7</td>
<td>3</td>
<td>1.2</td>
<td>1</td>
<td>0.4</td>
</tr>
<tr>
<td>III CNS and miscellaneous intracranial and intraspinal neoplasms</td>
<td>9</td>
<td>12.5</td>
<td>75</td>
<td>28.9</td>
<td>0.43</td>
<td>0.017**</td>
</tr>
<tr>
<td>a Ependymomas and choroid plexus tumour</td>
<td>2</td>
<td>0.8</td>
<td>1</td>
<td>1.5</td>
<td>3</td>
<td>1.1</td>
</tr>
<tr>
<td>b Astrocytomas</td>
<td>6</td>
<td>8.3</td>
<td>42</td>
<td>16.2</td>
<td>0.51</td>
<td>0.129</td>
</tr>
<tr>
<td>c Intracranial and intraspinal embryonal tumours</td>
<td>2</td>
<td>2.8</td>
<td>13</td>
<td>5.0</td>
<td>0.55</td>
<td>0.436</td>
</tr>
<tr>
<td>d Other gliomas</td>
<td>9</td>
<td>3.5</td>
<td>3</td>
<td>1.2</td>
<td>1</td>
<td>0.4</td>
</tr>
<tr>
<td>e Other specified intracranial and intraspinal neoplasms</td>
<td>1</td>
<td>1.4</td>
<td>6</td>
<td>2.3</td>
<td>0.60</td>
<td>0.638</td>
</tr>
<tr>
<td>Cancer type (ICCC classification)</td>
<td>10–14 years</td>
<td></td>
<td>15–19 years</td>
<td></td>
<td>20–24 years</td>
<td></td>
</tr>
<tr>
<td>---</td>
<td>---</td>
<td>---</td>
<td>---</td>
<td>---</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td></td>
<td>Māori</td>
<td>Non-Māori</td>
<td>IRR**</td>
<td>p</td>
<td>IRR</td>
<td>p</td>
</tr>
<tr>
<td>Cases</td>
<td>IR*</td>
<td>Cases</td>
<td>IR</td>
<td>Cases</td>
<td>IR</td>
<td>Cases</td>
</tr>
<tr>
<td>IV Neuroblastoma and other peripheral nervous cell tumours</td>
<td>2 2.8</td>
<td>7 2.7</td>
<td>1.03 0.976</td>
<td></td>
<td>4 1.5</td>
<td></td>
</tr>
<tr>
<td>a Neuroblastoma and ganglioneuroblastoma</td>
<td>6 2.3</td>
<td></td>
<td></td>
<td>4 1.5</td>
<td></td>
<td></td>
</tr>
<tr>
<td>b Other peripheral nervous cell tumours</td>
<td>2 2.8</td>
<td>1 0.4</td>
<td>7.20 0.107</td>
<td></td>
<td>2 3.5</td>
<td>2 0.7</td>
</tr>
<tr>
<td>V Retinoblastoma</td>
<td></td>
<td></td>
<td>1 0.4</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>VI Renal tumours</td>
<td>3 1.2</td>
<td></td>
<td>2 3.1</td>
<td>5 1.9</td>
<td>1.61 0.569</td>
<td></td>
</tr>
<tr>
<td>a Nephroblastoma and other non-epithelial renal tumours</td>
<td>1 0.4</td>
<td></td>
<td>1 1.5</td>
<td>3 1.1</td>
<td>1.35 0.795</td>
<td></td>
</tr>
<tr>
<td>b Renal carcinomas</td>
<td>2 0.8</td>
<td></td>
<td>1 1.5</td>
<td>1 0.4</td>
<td>4.04 0.322</td>
<td></td>
</tr>
<tr>
<td>c Unspecified malignant renal tumours</td>
<td></td>
<td></td>
<td>1 0.4</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>VII Hepatic tumours</td>
<td>4 1.5</td>
<td></td>
<td>1 1.5</td>
<td>8 3.0</td>
<td>0.50 0.516</td>
<td></td>
</tr>
<tr>
<td>a Hepatoblastoma</td>
<td>1 0.4</td>
<td></td>
<td>1 1.5</td>
<td>8 3.0</td>
<td>0.50 0.516</td>
<td></td>
</tr>
<tr>
<td>b Hepatic carcinomas</td>
<td>3 1.2</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>VIII Malignant bone tumours</td>
<td>9 12.5</td>
<td>44 16.9</td>
<td>0.74 0.401</td>
<td></td>
<td>9 13.7</td>
<td>57 21.5</td>
</tr>
<tr>
<td>a Osteosarcomas</td>
<td>6 8.3</td>
<td>24 9.2</td>
<td>0.90 0.818</td>
<td></td>
<td>4 6.1</td>
<td>38 14.4</td>
</tr>
<tr>
<td>b Chondrosarcomas</td>
<td>2 0.8</td>
<td></td>
<td>2 0.8</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>c Ewing tumour and related sarcomas of bone</td>
<td>3 4.2</td>
<td>17 6.5</td>
<td>0.64 0.472</td>
<td></td>
<td>4 6.1</td>
<td>11 4.2</td>
</tr>
<tr>
<td>d Other specified malignant bone tumours</td>
<td>1 1.5</td>
<td>1 1.5</td>
<td>5 1.9</td>
<td>0.81 0.841</td>
<td></td>
<td>1 0.4</td>
</tr>
<tr>
<td>e Unspecified malignant bone tumours</td>
<td>1 0.4</td>
<td></td>
<td>1 0.4</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>IX Soft tissue and other extraosseous sarcomas</td>
<td>9 12.5</td>
<td>23 8.9</td>
<td>1.41 0.384</td>
<td></td>
<td>9 13.7</td>
<td>33 12.5</td>
</tr>
<tr>
<td>a Rhabdomyosarcomas</td>
<td>1 1.4</td>
<td>8 3.1</td>
<td>0.45 0.453</td>
<td></td>
<td>4 6.1</td>
<td>14 5.3</td>
</tr>
<tr>
<td>b Fibrosarcomas, peripheral nerve sheath tumours, and other fibrous neoplasms</td>
<td>1 0.4</td>
<td></td>
<td>1 0.4</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>c Kaposi sarcoma</td>
<td>1 0.4</td>
<td></td>
<td>1 0.4</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>d Other specified soft tissue sarcomas</td>
<td>7 9.7</td>
<td>10 3.9</td>
<td>2.52 0.060</td>
<td></td>
<td>3 4.6</td>
<td>12 4.5</td>
</tr>
<tr>
<td>e Unspecified soft tissue sarcomas</td>
<td>1 1.4</td>
<td>3 1.2</td>
<td>1.20 0.873</td>
<td></td>
<td>2 3.1</td>
<td>5 1.9</td>
</tr>
<tr>
<td>Cancer type (ICCC classification)</td>
<td>10–14 years</td>
<td>15–19 years</td>
<td>20–24 years</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>----------------------------------</td>
<td>-------------</td>
<td>-------------</td>
<td>-------------</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Māori Cases</td>
<td>Non-Māori Cases</td>
<td>Māori Cases</td>
<td>Non-Māori Cases</td>
<td>Māori Cases</td>
<td>Non-Māori Cases</td>
</tr>
<tr>
<td>X Germ cell tumours, trophoblastic tumours, and neoplasms of gonads</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>a Intracranial and intraspinal germ cell tumours</td>
<td>5</td>
<td>31</td>
<td>6.9</td>
<td>31</td>
<td>11.9</td>
<td>0.58</td>
</tr>
<tr>
<td>b Malignant extracranial and extragonadal germ cell tumours</td>
<td>1</td>
<td>14</td>
<td>5.4</td>
<td>0.26</td>
<td>0.190</td>
<td>3</td>
</tr>
<tr>
<td>c Malignant gonadal germ cell tumours</td>
<td>2</td>
<td>15</td>
<td>5.8</td>
<td>0.48</td>
<td>0.327</td>
<td>14</td>
</tr>
<tr>
<td>d Gonadal carcinomas</td>
<td>1</td>
<td>2</td>
<td>0.8</td>
<td>1.80</td>
<td>0.631</td>
<td>3</td>
</tr>
<tr>
<td>e Other and unspecified malignant gonadal tumours</td>
<td>4</td>
<td>1.5</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>XI Other malignant epithelial neoplasms and malignant melanomas</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>a Adrenocortical carcinomas</td>
<td>3</td>
<td>4.2</td>
<td>38</td>
<td>14.6</td>
<td>0.28</td>
<td>0.036††</td>
</tr>
<tr>
<td>b Thyroid carcinomas</td>
<td>2</td>
<td>0.8</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>c Nasopharyngeal carcinomas</td>
<td>1</td>
<td>1.5</td>
<td>25</td>
<td>9.4</td>
<td>0.16</td>
<td>0.073</td>
</tr>
<tr>
<td>d Malignant melanomas</td>
<td>26</td>
<td>10.0</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>e Other and unspecified carcinomas</td>
<td>3</td>
<td>4.2</td>
<td>10</td>
<td>3.9</td>
<td>1.08</td>
<td>0.904</td>
</tr>
<tr>
<td>XII Other and unspecified malignant neoplasms</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>a Other specified malignant tumours</td>
<td>3</td>
<td>1.2</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>b Other unspecified malignant tumours</td>
<td>3</td>
<td>1.2</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total all cancers</td>
<td>58</td>
<td>80.4</td>
<td>363</td>
<td>139.8</td>
<td>0.58</td>
<td>&lt;0.001††</td>
</tr>
</tbody>
</table>

Notes:
* IR = incidence rate per million person-years.
** IRR = incidence rate ratio (expressed as ratio of Māori IR to non-Māori IR).
† p = two-tailed test of significance.
†† Significant findings (p<0.05).
International reports suggest that some ethnic groups have a lower incidence of cancer (Smith et al 1999). This study demonstrated similar results for Māori, who had a significantly lower incidence of cancer than non-Māori at all ages. The lower rate of melanoma contributes a large proportion of this difference, especially at older ages. Other possible explanations include undercounting of ethnicity data (as discussed in the ‘Materials and methods’ section), environmental influences, and biological differences between Māori and non-Māori. The aetiology of many of the cancers in this age group is not known, meaning that although potential environmental influences (such as poverty or childhood infections) have been proposed, their impact has not been proven (McNally and Eden 2004; Villar and Menck 1994). Explanations for differences between Māori and non-Māori incidence rates for many cancers are therefore still speculative. Biological differences in the cancers affecting Māori, or differences in host susceptibility are other possible explanations.

Deprivation differences

Geographic deprivation scores were assigned based on where each individual was living at the time of diagnosis, for cases diagnosed from 1994 on (as discussed in the ‘Materials and methods’ section). Location data were not available for 85 cases. The distribution of cases across deciles of deprivation is shown in Table 8. While the distribution appears slightly skewed towards greater numbers of cases in more deprived regions, the actual age distribution within each decile is not equal. Appendix 4 shows the actual age distribution of this section of the population across deciles. When this is taken into account, as also shown in Table 8, there is no significant difference in incidence based on regional deprivation. Results must be interpreted cautiously given the imprecision of deprivation data, as discussed in the ‘Materials and methods’ section.

Table 8: Distribution of cancers by deprivation decile based on census area unit at time of diagnosis, 10–24-year-olds, 1994–2002

<table>
<thead>
<tr>
<th>Decile</th>
<th>Actual cases</th>
<th>Expected cases†</th>
</tr>
</thead>
<tbody>
<tr>
<td>1st*</td>
<td>178</td>
<td>143.8</td>
</tr>
<tr>
<td>2nd</td>
<td>200</td>
<td>155.8</td>
</tr>
<tr>
<td>3rd</td>
<td>182</td>
<td>148.0</td>
</tr>
<tr>
<td>4th</td>
<td>168</td>
<td>159.3</td>
</tr>
<tr>
<td>5th</td>
<td>202</td>
<td>156.4</td>
</tr>
<tr>
<td>6th</td>
<td>141</td>
<td>170.7</td>
</tr>
<tr>
<td>7th</td>
<td>158</td>
<td>176.8</td>
</tr>
<tr>
<td>8th</td>
<td>147</td>
<td>187.7</td>
</tr>
<tr>
<td>9th</td>
<td>188</td>
<td>211.5</td>
</tr>
<tr>
<td>10th#</td>
<td>134</td>
<td>188.0</td>
</tr>
</tbody>
</table>

Chi² test for independence: p = 0.3 (not significant)

Notes:
* Least deprived decile.
# Most deprived decile.
† Calculated based on what the distribution across deciles would be if there was no deprivation effect (allowing for age and period of diagnosis).
**Malignant melanoma**

Melanoma has been recognised as a particular problem in Australasia. Rising rates are associated with increased ultraviolet exposure due to ozone depletion and individual behaviour, which in combination have produced the highest incidence rates in the world. More recently rates have levelled off, possibly due to increased sun-protective behaviour brought about by extensive public health campaigns (Bulliard and Cox 2000).

Melanoma is rare in children, but incidence steadily increases after 15 years of age.

This study showed a relatively stable incidence rate over the 15-year period. Small numbers of actual cases per year (between 6 and 16 cases per year for 15–19-year-olds, for example) produce the annual fluctuations shown in Figure 6.

There was a gender difference in melanoma incidence: 20–24-year-old males had a significantly lower rate of melanoma than females in this age group over the 15-year period (M:F ratio = 0.6, p < 0.05).

Incidence of melanoma in Māori is much lower than in non-Māori (p < 0.01). For example, for 15–19-year-olds at diagnosis, Māori incidence was 6.5 per million person-years, compared with 45.0 in non-Māori. This difference is understandable, given the protective effects of skin melanin (pigmentation). This is an individual protective factor, however, so that the risk of melanoma for someone who identifies as Māori but has relatively fair skin may be just as great as for someone who identifies as non-Māori and who has fair skin.

**Figure 6**: Malignant melanoma incidence rates by age and year of diagnosis, 1988–2002
The overall incidence rate of malignant melanoma in New Zealand remains higher than in many other populations; for example, for 15–19-year-olds at diagnosis, the incidence rate in New Zealand is 38.0 per million person-years compared with 14.1 in the US, 8.1 in the UK and 15.6 in France (Albritton and Bleyer 2003; Birch et al 2003; Desandes et al 2004). However, the rate in New Zealand is lower than in Australia (incidence rate of 57 in 2001) (AIHW 2003).
Results: Cancer Outcome – Survival

Key points about survival overall found in this study are that:

- out of the 2917 cases of cancer diagnosed from 1988–2002 in 10–24-year-olds, there were 754 deaths notified up to 30 June 2005
- overall, survival is similar to that in European countries, but slightly worse than in Australia and the US
- female survival is better than male survival at older ages
- survival overall is better in 20–24-year-olds at age of diagnosis than in 15–19-year-olds, whose survival in turn is better than for 10–14-year-olds; the differences are mainly due to the different types of cancer affecting each age group, particularly higher rates of non-metastatic melanoma at older ages
- survival varies greatly depending on type of cancer, gender and age at diagnosis
- lymphoid leukaemia survivorship ranges from 70 percent for 10–14-year-olds to 43 percent for 20–24-year-olds
- for those aged over 15 years at diagnosis with a central nervous system (brain) tumour or non-Hodgkin lymphoma, the chance of surviving five years was less than 70 percent
- survival has improved over the period of the study for lymphoid leukaemia, myeloid leukaemia, Hodgkin lymphoma; there has been no significant change for non-Hodgkin lymphoma or bone tumours
- Māori survival, after adjustment for age, gender and cancer group, was worse than that of non-Māori, although other confounding factors may have affected this result.

Improvement in outcomes for young people with cancer is a key objective of the Cancer Control Strategy (Cancer Control Taskforce 2005). Detailed outcome data are collected locally in some regions, and are collected nationwide for children with cancer via the Children’s Cancer Registry. They are not routinely collected for adolescents and young people outside of these domains. NZHIS does not collect detailed treatment or outcome data, apart from death. This practice allows accurate calculation of survivorship but does not provide a broader picture of the morbidity incurred by patients. Such data are crucial in monitoring the effectiveness of cancer services. A pragmatic solution would be to expand the Children’s Cancer Registry to include adolescents and young adults. With those limitations, mortality and survival data provide one measure of effectiveness of cancer detection and treatment.

There are two common approaches to calculating cancer survival: the cause-specific model and the relative survival model. Researchers in the field of cancer survival have used one of these two approaches, while the strengths and weaknesses of both continue to be debated (Teuff et al 2005).
The cause-specific survival model calculates survival based on deaths due to cancer-related causes alone. The appeal of this approach is that the excess mortality due to cancer itself is separated out from unrelated deaths that may affect anyone. This is particularly important at older ages, when other causes of death become significant. In the adolescent age group, death is rare (occurring to less than 0.5 percent per year), and so the difference between deaths from all causes and deaths from cancer alone is small. In addition, at the time of this study, NZHIS held information about cause of death extracted from death certificates for deaths up to 31 December 2002, but held only date of death for deaths after this date up to 30 June 2005. Finally, using the cause-specific model requires accurate cause of death to be entered, extracted and coded from death certificates, a process known to be inaccurate (Brown and Frankovich 1998). For all of these reasons, the relative survival model was used.

The relative survival model compares the survival of the group being studied with the survival of the general population, matched for gender and age. A relative survival rate of 0.8 (or 80 percent) means that the group being studied has 80 percent of the survival of the general population from which they are drawn.

Table 9 shows overall age-specific five-year relative survival rates for the New Zealand group in this study, along with a comparison with other countries. As noted above, the all-cause (or crude) survival is very similar given the low overall mortality in this age group. Table 10 shows five-year relative survival rates for the more common types of cancer for the group in this study.

Survival is similar to that reported for young people in European countries but slightly worse than that for Australia and the US (Ries et al 1999; Smith et al 1999; Strouse et al 2005). The period covered by each study is pertinent, as survival has generally improved over time due to improved cancer management. More recent results should therefore be expected to show better survival than older results do, all else being equal. To allow for this effect, analysis of New Zealand data for comparable periods was undertaken; however the differences remained. Part of the difference between New Zealand and Australia may be due to Australia’s higher incidence of melanoma, which has a higher survival rate than most cancers.

For individual types of cancer, survival was broadly similar in international comparisons, except for astrocytoma, where US survival was better. New Zealand survival for acute myeloid leukaemia appears better than US survival; however, this difference disappears when similar periods are analysed. The comparisons do not take into account differences in tumour subtypes, which may also explain some of the differences.
Table 9: Age-specific five-year relative survival for all types of cancer, 1988–2002, with international comparisons

<table>
<thead>
<tr>
<th>Age at diagnosis (years)</th>
<th>10–14</th>
<th>15–19</th>
<th>20–24</th>
</tr>
</thead>
<tbody>
<tr>
<td>New Zealand 1988–2002</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Females</td>
<td>72%</td>
<td>79%</td>
<td>83%</td>
</tr>
<tr>
<td>(66–77)†</td>
<td>(75–83)</td>
<td>(81–86)</td>
<td></td>
</tr>
<tr>
<td>Males</td>
<td>70%</td>
<td>69%</td>
<td>77%</td>
</tr>
<tr>
<td>(65–75)</td>
<td>(65–74)</td>
<td>(74–80)</td>
<td></td>
</tr>
<tr>
<td>Females and males</td>
<td>71%</td>
<td>74%</td>
<td>80%</td>
</tr>
<tr>
<td>(67–75)</td>
<td>(71–77)</td>
<td>(78–82)</td>
<td></td>
</tr>
<tr>
<td>Comparisons with other countries</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Australia 1992–1997&amp;a</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Females</td>
<td>78%</td>
<td>83%</td>
<td>88%</td>
</tr>
<tr>
<td>Males</td>
<td>77%</td>
<td>78%</td>
<td>84%</td>
</tr>
<tr>
<td>SEER (US) 1985–1994*</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>77%</td>
<td></td>
</tr>
<tr>
<td>North of England 1988–1995#</td>
<td>73%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Northern Europe 1990–1994%</td>
<td>81%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>UK 1990–1994%</td>
<td>75%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Central Europe 1990–1994%</td>
<td>78%</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Notes:
† Ranges in brackets represent 95 percent confidence intervals.
&a AIHW (2003).
* Smith et al (1999); SEER = Surveillance, Epidemiology and End Results service of the National Cancer Institute (US data).
## Table 10: Age-specific five-year relative survival for common types of cancer, 1988–2002, with international comparisons

<table>
<thead>
<tr>
<th>ICCC category</th>
<th>Age at diagnosis (years)</th>
<th>SEER data* 15–19</th>
<th>Australian data* 10–24</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>10–14</td>
<td>15–19</td>
<td>20–24</td>
</tr>
<tr>
<td>I Leukaemias, myeloproliferative diseases, and myelodysplastic diseases</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>I.a Lymphoid leukaemias</td>
<td>64% (55–72)†</td>
<td>55% (46–64)</td>
<td>49% (39–58)</td>
</tr>
<tr>
<td>I.b Acute myeloid leukaemias</td>
<td>67% (57–77)</td>
<td>53% (41–64)</td>
<td>41% (26–55)</td>
</tr>
<tr>
<td>II Lymphomas and reticuloendothelial neoplasms</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>II.a Hodgkin lymphomas</td>
<td>88% (80–95)</td>
<td>83% (76–89)</td>
<td>77% (71–84)</td>
</tr>
<tr>
<td>II.b Non Hodgkin lymphomas (except Burkitt lymphoma)</td>
<td>61% (52–71)</td>
<td>63% (51–75)</td>
<td>63% (52–74)</td>
</tr>
<tr>
<td>III CNS and miscellaneous intracranial and intraspinal neoplasms</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>III.b Astrocytomas</td>
<td>67% (55–79)</td>
<td>58% (41–74)</td>
<td>56% (41–70)</td>
</tr>
<tr>
<td>VIII Malignant bone tumours</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>55% (43–68)</td>
<td>57% (47–67)</td>
<td>65% (51–79)</td>
</tr>
<tr>
<td>X Germ cell tumours, trophoblastic tumours, and neoplasms of gonads</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>X.c Malignant gonadal germ cell tumours</td>
<td>89% (82–95)</td>
<td>93% (90–96)</td>
<td>94% (90–97)</td>
</tr>
<tr>
<td>XI.d Malignant melanomas</td>
<td>84% (72–97)</td>
<td>95% (91–98)</td>
<td>95% (92–97)</td>
</tr>
</tbody>
</table>

### Notes

† Ranges in brackets represent 95 percent confidence intervals.
Survival trends over time

To assess the trends over the period of the study, cases were divided into those diagnosed before 1 January 1995 and those diagnosed after, then the two groups were compared. Taking all cancers together for each age group, survival has improved slightly but not significantly between 1988–1994 and 1995–2002. Looking at individual types of cancer, however, survival has improved markedly for lymphoid leukaemia, myeloid leukaemia and Hodgkin lymphoma, as shown in Table 11. There has been no significant change for non-Hodgkin lymphoma, astrocytoma or bone tumours. Survival for gonadal germ cell tumours and melanoma was high throughout.

Overall survival for a given period will depend on the mix of different cancer types, and the age and gender distribution of individuals. To take these potentially confounding factors into account, the size and significance of the change over time for common cancer types were assessed using Cox modelling including deaths from all causes, allowing for age and gender; the results of this analysis are shown in Table 12.


<table>
<thead>
<tr>
<th>ICCC category</th>
<th>Period of diagnosis</th>
<th>Age at diagnosis (years)</th>
<th>10–14</th>
<th>15–19</th>
<th>20–24</th>
</tr>
</thead>
<tbody>
<tr>
<td>I.a Lymphoid leukaemias</td>
<td>47% (29–65)</td>
<td>83% (72–94)</td>
<td>47% (31–64)</td>
<td>53% (35–71)</td>
<td>26% (6–44)</td>
</tr>
<tr>
<td>I.b Acute myeloid leukaemias</td>
<td>50% (24–76)</td>
<td>64% (39–90)</td>
<td>44% (20–68)</td>
<td>63% (43–84)</td>
<td>48% (26–69)</td>
</tr>
<tr>
<td>II.a Hodgkin lymphomas</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>II.b Non Hodgkin lymphomas (except Burkitt lymphoma)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>III.b Astrocytomas</td>
<td>68% (51–85)</td>
<td>68% (50–85)</td>
<td>58% (36–81)</td>
<td>63% (39–87)</td>
<td>55% (34–76)</td>
</tr>
<tr>
<td>VIII.a Osteosarcomas</td>
<td>71% (53–89)</td>
<td>63% (33–92)</td>
<td>65% (47–82)</td>
<td>52% (31–73)</td>
<td>84% (63–105)</td>
</tr>
<tr>
<td>VIII.c Ewing tumour and related sarcomas of bone</td>
<td>45% (12–77)</td>
<td>23% (4–50)</td>
<td>41% (18–65)</td>
<td>40% (10–71)</td>
<td>67% (29–105)</td>
</tr>
<tr>
<td>X.c Malignant gonadal germ cell tumours</td>
<td>97% (91–104)</td>
<td>92% (82–101)</td>
<td>93% (88–99)</td>
<td>93% (88–99)</td>
<td>93% (88–99)</td>
</tr>
<tr>
<td>XI.d Malignant melanomas</td>
<td>94% (88–99)</td>
<td>96% (92–101)</td>
<td>94% (91–98)</td>
<td>94% (91–98)</td>
<td>95% (92–98)</td>
</tr>
<tr>
<td>All cancers</td>
<td>70% (64–76)</td>
<td>72% (66–77)</td>
<td>72% (67–76)</td>
<td>76% (72–80)</td>
<td>80% (77–83)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>ICCC category</th>
<th>Age at diagnosis (years)</th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>10–14</td>
<td>15–19</td>
<td>20–24</td>
<td>All ages (10–24)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>HR**</td>
<td>p†</td>
<td>HR</td>
<td>p</td>
<td>HR</td>
<td>p</td>
</tr>
<tr>
<td>I.a Lymphoid leukaemias</td>
<td>0.86</td>
<td>0.002††</td>
<td>0.97</td>
<td>0.37</td>
<td>0.91</td>
<td>0.08</td>
</tr>
<tr>
<td>I.b Acute myeloid leukaemias</td>
<td>0.95</td>
<td>0.46</td>
<td>0.90</td>
<td>0.09</td>
<td>0.93</td>
<td>0.25</td>
</tr>
<tr>
<td>II.a Hodgkin lymphomas</td>
<td>0.88</td>
<td>0.36</td>
<td>0.97</td>
<td>0.79</td>
<td>0.83</td>
<td>0.03†</td>
</tr>
<tr>
<td>II.b Non Hodgkin lymphomas (except Burkitt)</td>
<td>0.98</td>
<td>0.83</td>
<td>0.95</td>
<td>0.48</td>
<td>0.99</td>
<td>0.77</td>
</tr>
<tr>
<td>III.b Astrocytomas</td>
<td>1.01</td>
<td>0.85</td>
<td>1.03</td>
<td>0.65</td>
<td>1.06</td>
<td>0.25</td>
</tr>
<tr>
<td>VIII.a Osteosarcomas</td>
<td>1.12</td>
<td>0.08</td>
<td>1.02</td>
<td>0.74</td>
<td>1.18</td>
<td>0.14</td>
</tr>
<tr>
<td>VIII.c Ewing tumour and related sarcomas of bone</td>
<td>1.02</td>
<td>0.81</td>
<td>0.99</td>
<td>0.86</td>
<td>1.15</td>
<td>0.18</td>
</tr>
<tr>
<td>X.c Malignant gonadal germ cell tumours</td>
<td>‡</td>
<td></td>
<td>1.06</td>
<td>0.63</td>
<td>1.01</td>
<td>0.86</td>
</tr>
</tbody>
</table>

Notes:
* Ranges in brackets represent 95 percent confidence intervals.
** Cox model includes age and gender (stratification variables).
† HR = hazard ratio, using 1988–1994 as baseline, using all-cause mortality.
‡ Significant findings (p<0.05).
†† No deaths in this group.

Ethnic differences in survival

This study set out to determine whether there was a significant difference in cancer survival between Māori and non-Māori. After adjusting for gender, age and cancer group, Māori experienced significantly worse survival than non-Māori. When divided into five-year age groups, the significant difference remained for 10–14-year-olds and 20–24-year-olds, but disappeared for 15–19-year-olds at diagnosis. The comparisons were also made excluding melanoma, in case this numerically dominant cancer was skewing results, but the findings were similar. The results of both analyses are shown in Table 13.

Survival following a diagnosis of cancer depends on many factors including age at diagnosis, gender, era of treatment, type of cancer, and cancer grade or stage at diagnosis. In the context of comparing one group with another, these factors are known as confounders. Adjusting for age, gender and cancer group (using the 12 ICCC categories) reduces potential confounding; however, the chance of confounding remains. In particular, the numbers in this study were insufficient to allow statistical comparison between individual types of cancer affecting both Māori and non-Māori. For this reason, the differences in survival found in this study should be interpreted cautiously.
Table 13: Survival hazard ratios for Māori versus non-Māori, including and excluding melanoma, 1988–2002

<table>
<thead>
<tr>
<th>Age at diagnosis (years)</th>
<th>Māori/non-Māori HR* (95% CI**)</th>
<th>p†</th>
</tr>
</thead>
<tbody>
<tr>
<td>10–14 All cancer</td>
<td>1.75 (1.18–2.59)</td>
<td>0.005††</td>
</tr>
<tr>
<td>Excluding melanoma</td>
<td>1.76 (1.19–2.61)</td>
<td>0.005††</td>
</tr>
<tr>
<td>15–19 All cancer</td>
<td>1.31 (0.91–1.86)</td>
<td>0.14</td>
</tr>
<tr>
<td>Excluding melanoma</td>
<td>1.28 (0.90–1.83)</td>
<td>0.17</td>
</tr>
<tr>
<td>20–24 All cancer</td>
<td>2.15 (1.62–2.85)</td>
<td>&lt;0.0001††</td>
</tr>
<tr>
<td>Excluding melanoma</td>
<td>1.85 (1.39–2.44)</td>
<td>&lt;0.0001††</td>
</tr>
<tr>
<td>10–24 All cancer</td>
<td>1.75 (1.44–2.12)</td>
<td>&lt;0.0001††</td>
</tr>
<tr>
<td>Excluding melanoma</td>
<td>1.63 (1.35–1.98)</td>
<td>&lt;0.0001††</td>
</tr>
</tbody>
</table>

Notes:
* HR = hazard ratio derived from Cox proportional hazard modelling, adjusted for age (individual year), gender and ICCC category – Māori/non-Māori. For example, an HR of 1.7 means Māori survival is 1.7 times worse than non-Māori survival.
** 95% CI = 95% confidence interval for hazard ratio.
† p = two-tailed test of significance.
†† Significant findings (p<0.05).

Apart from confounding, other possible explanations for the differences in survival between Māori and non-Māori are that:

- this is a chance finding (and there is no true difference)
- adolescents identify multiple factors that limit their access to health services (Adolescent Health Research Group 2003). Māori young people may be more affected by these factors than non-Māori, which would contribute to later diagnosis; however, this idea is speculative.

Other cancer research has shown worse survival among Māori at older ages (Jeffreys et al 2005), but not in childhood (Douglas and Dockerty, paper in preparation).

To further assess ethnic differences in outcome amongst adolescents, a larger study would be required, along with more detailed data on diagnosis and outcome. In addition, analysis of other ethnic groups (such as Pacific peoples) in a larger study would be beneficial.
Conclusions and Recommendations

Cancer incidence and survival among New Zealand adolescents

This study has confirmed that cancer among New Zealand adolescents generally follows a similar pattern to that in other developed countries. Namely, the incidence of cancer increases with age, the types of cancer that are most common change with age, and some types of cancer have relatively poor outcomes. Incidence was stable over time. Females had better survival than males. Māori had lower incidence (overall) than non-Māori.

The study also shows that there are some aspects unique to New Zealand. First, the high rate of melanoma is well demonstrated, confirming the need for ongoing preventative measures aimed at reducing sun exposure. In addition, after adjusting for age, gender and cancer group, Māori survival was worse than non-Māori survival. This finding requires further investigation.

Improving management of cancer among adolescents

Management of cancer among adolescents has improved over the past decades. To improve the situation for adolescents further, a systemic approach is required. Some cancer is amenable to public health interventions aimed at primary prevention, such as the development and implementation of a vaccine that may prevent cervical cancer, and the avoidance of ultraviolet radiation to reduce melanoma.

For young people who develop cancer, moves to improve access to health services generally may assist in early diagnosis. Adolescents are infrequent users of health services, not because of a lack of need but because of multiple factors limiting access. Among these limiting factors are that adolescents do not want to make a fuss, cannot be bothered, find the services too expensive, are afraid and/or are uncomfortable with the health provider (Adolescent Health Research Group 2003).

Once diagnosed, adolescents should have access to cancer services designed to take account of their medical and developmental needs so that the outcome for survivors is optimal. This level of access includes access to:
- appropriately trained clinicians able to deliver adolescent-specific care
- facilities appropriate for adolescents
- developmentally appropriate psychosocial support
- assistance with care co-ordination
- adolescent-appropriate clinical trials
- culturally supportive services
- long-term follow-up services.

Monitoring progress is also vital. This includes the ongoing surveillance of population incidence of cancer, analysed using classification schemes designed for adolescents. In addition, detailed demographic, treatment and outcome data are needed. This goal could be achieved by extending the Children’s Cancer Registry, which already collects these data for children, to include adolescents and young people.
Finally, there is a good argument for amending the Act governing the New Zealand Cancer Registry to include benign central nervous system tumours. These tumours are not currently included under the Registry legislation, even though due to their location their effect can be as devastating as a malignant tumour.
Appendix 1: Data Validity Checks

The process for checking and converting data was as follows:

1. In situ/non-invasive cancers were excluded (totalling 5190 registrations).
2. Date of birth was checked against NMDS and mortality datasets, leading to exclusion of two registrations.
3. ICD codes were converted from ICD-10-AM-v2 (Australian modification, version 2) to ICD-O-2, then to ICD-O-3 using IARC conversion software (Ferlay 2005).
4. ICD-O-3 topography and morphology codes (as converted) were checked for validity using IARC check software (Ferlay 2005).
5. Rules for registration of multiple primary cancers were applied (IARC 2004), leading to exclusion of four registrations.

Exclusion of non-invasive/in situ cancers

In situ cancers were excluded from the data extracted on the basis of an ICD-10 site (topography) code denoting an in situ diagnosis (ie, a code beginning with ‘D’). These totalled 4844 registrations. In addition, some registrations prior to 1994 had an ‘extent of disease’ code translating to in situ, at variance with the invasive characteristics implied by the ICD site code. These comprised mainly cervical carcinoma-in-situ, totalled 346 registrations, and were also excluded.

Age and date of birth checks

Dates of birth as recorded in the NZCR were compared with hospital discharge data and mortality data. These sources disagreed on dates of birth for 111 individuals. The majority appeared to be due to transpositions of digits or other errors during data entry. The 111 discrepancies were dealt with in the following ways:

- Two individuals were excluded from further analysis. These two individuals were recorded in the NMDS as being much older, and the nature of their cancer diagnoses and co-morbidities (for example, prostatic carcinoma and ischaemic heart disease) were in keeping with the older ages.
- Fourteen individuals had a date of birth recorded in the NZCR that was different to that recorded during hospital admissions. The differences did not affect their age group; therefore for the purposes of analysis, the dates of birth recorded in the NZCR were used.
- Two individuals had a different date of birth recorded consistently in a number of hospital admissions and/or the mortality dataset. For the purposes of analysis, the date of birth was altered to reflect the date that was consistently used. This alteration changed the age groups for these two individuals.
- The remaining 93 individuals had their date of birth recorded inconsistently during subsequent hospital admissions but at least one record of their date of birth agreed with that held in the NZCR. For the purposes of analysis, the dates of birth recorded in the NZCR were used for these individuals.
IARC validity checks

The International Association for Research on Cancer has produced software to check the validity of registrations (Ferlay 2005; Ferlay et al 2005). The software checks registrations for various combinations of age, gender, site and histology. The NZCR data were processed using this software. This highlighted that there were:

- 16 registrations of secondary cancer, all of which had no other registrations (ie, the primary site was not identified) and therefore were included in subsequent analysis
- 48 registrations of haematological malignancies where the morphology code and anatomical site code were slightly inconsistent, but these did not affect ICCC classification.
- 117 warnings that were checked individually, leading to reclassification of five registrations under the ICCC-3 scheme.

Multiple primaries

There were 33 individuals with multiple registrations. These were checked against the IARC rules for reporting of multiple primaries (IARC 2004), which led to the exclusion of four registrations. The reasons are set out in Table 14.

Table 14: Reasons for exclusion of multiple registrations

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Reason for exclusion*</th>
</tr>
</thead>
<tbody>
<tr>
<td>C436 – Malignant melanoma of upper limb, including shoulder</td>
<td>Only register once per rule 3</td>
</tr>
<tr>
<td>C819 – Hodgkin's disease, unspecified</td>
<td>Change in histological diagnosis of same cancer</td>
</tr>
<tr>
<td>C819 – Hodgkin's disease, unspecified</td>
<td>Only register once per rule 4.1</td>
</tr>
<tr>
<td>C9230 – Myeloid sarcoma, without mention of remission</td>
<td>Only register once per rule 4.1</td>
</tr>
</tbody>
</table>

Note:
* Refer to IARC (2004) for explanation of rules.

Summary of data checks and exclusions

8113 total registrations extracted less 5190 in situ diagnoses less two date of birth errors less four multiple registrations = 2917 registrations for analysis.
Appendix 2: Legislative Framework Underpinning the New Zealand Cancer Registry

Two pieces of legislation have governed cancer registrations since 1994. The relevant sections governing definitions of cancers for inclusion are shown below.

**Cancer Registry Act 1993**

Under the Cancer Registry Act 1993, ‘cancer’:

2. ...

(a) means a malignant growth of human tissue that, if unchecked,

   (i) is likely to spread to adjacent tissue or beyond its place of origin; and

   (ii) may have the propensity to recur; and

(b) without limiting the generality of paragraph (a) of this definition, includes carcinoma-in-situ, carcinoma, sarcoma (including Kaposi’s sarcoma), any mixed tumour, leukaemia, any type of lymphoma, and melanoma; but

(c) does not include:

   (i) any secondary or metastatic cancer, except where the primary cancer is not identified:

   (ii) any type of cancer that is declared by regulations made under this Act to be a cancer to which this Act does not apply: ...’

**Cancer Registry Regulations 1994**

Under the Cancer Registry Regulations 1994, the Cancer Registry Act does not apply to:

6. ...

(a) Basal cell cancer arising in the skin:

(b) Squamous cell cancer arising in the skin.’
## Appendix 3: Census Derived Data


### Table 15: Estimated mean annual population by ethnicity

<table>
<thead>
<tr>
<th>Calendar year</th>
<th>10–14 years</th>
<th>15–19 years</th>
<th>20–24 years</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Māori</td>
<td>Non-Māori</td>
<td>Total</td>
</tr>
<tr>
<td>1988*</td>
<td>32,830</td>
<td>233,570</td>
<td>266,400</td>
</tr>
<tr>
<td>1990*</td>
<td>32,210</td>
<td>224,090</td>
<td>256,300</td>
</tr>
<tr>
<td>1991</td>
<td>56,110</td>
<td>203,770</td>
<td>259,880</td>
</tr>
<tr>
<td>1992</td>
<td>55,850</td>
<td>203,030</td>
<td>258,880</td>
</tr>
<tr>
<td>1993</td>
<td>55,370</td>
<td>205,110</td>
<td>260,480</td>
</tr>
<tr>
<td>1994</td>
<td>54,990</td>
<td>207,710</td>
<td>262,700</td>
</tr>
<tr>
<td>1997</td>
<td>58,120</td>
<td>215,610</td>
<td>273,730</td>
</tr>
<tr>
<td>1998</td>
<td>60,580</td>
<td>219,380</td>
<td>279,960</td>
</tr>
<tr>
<td>1999</td>
<td>63,350</td>
<td>222,920</td>
<td>286,270</td>
</tr>
<tr>
<td>2000</td>
<td>66,250</td>
<td>227,310</td>
<td>293,560</td>
</tr>
<tr>
<td>2001</td>
<td>68,830</td>
<td>231,210</td>
<td>301,040</td>
</tr>
<tr>
<td>2002</td>
<td>70,650</td>
<td>236,920</td>
<td>307,570</td>
</tr>
</tbody>
</table>

Note:
* Ethnicity was assigned on a ‘sole ethnicity’ basis for 1988–1990.

### Table 16: Estimated mean annual population by gender

<table>
<thead>
<tr>
<th>Calendar year</th>
<th>10–14 years</th>
<th>15–19 years</th>
<th>20–24 years</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Female</td>
<td>Male</td>
<td>Total</td>
</tr>
<tr>
<td>1988</td>
<td>130,060</td>
<td>136,340</td>
<td>266,400</td>
</tr>
<tr>
<td>1989</td>
<td>126,930</td>
<td>132,700</td>
<td>259,630</td>
</tr>
<tr>
<td>1990</td>
<td>125,780</td>
<td>130,520</td>
<td>256,300</td>
</tr>
<tr>
<td>1991</td>
<td>127,490</td>
<td>132,390</td>
<td>259,880</td>
</tr>
<tr>
<td>1992</td>
<td>126,480</td>
<td>132,400</td>
<td>258,880</td>
</tr>
<tr>
<td>1993</td>
<td>126,870</td>
<td>133,610</td>
<td>260,480</td>
</tr>
<tr>
<td>1994</td>
<td>127,620</td>
<td>135,080</td>
<td>262,700</td>
</tr>
<tr>
<td>1995</td>
<td>129,000</td>
<td>136,610</td>
<td>265,610</td>
</tr>
<tr>
<td>1996</td>
<td>130,880</td>
<td>138,270</td>
<td>269,150</td>
</tr>
<tr>
<td>1997</td>
<td>133,280</td>
<td>140,450</td>
<td>273,730</td>
</tr>
<tr>
<td>1998</td>
<td>136,480</td>
<td>143,480</td>
<td>279,960</td>
</tr>
<tr>
<td>1999</td>
<td>139,650</td>
<td>146,620</td>
<td>286,270</td>
</tr>
<tr>
<td>2000</td>
<td>143,400</td>
<td>150,160</td>
<td>293,560</td>
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<tr>
<td>2001</td>
<td>147,110</td>
<td>153,930</td>
<td>301,040</td>
</tr>
<tr>
<td>2002</td>
<td>149,990</td>
<td>157,580</td>
<td>307,570</td>
</tr>
</tbody>
</table>
### Table 17: Actuarial five-year survival of New Zealand population for selected ages

<table>
<thead>
<tr>
<th>Current exact age (years)</th>
<th>Probability of surviving five years (percentage)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Females</td>
</tr>
<tr>
<td>10</td>
<td>99.9</td>
</tr>
<tr>
<td>11</td>
<td>99.9</td>
</tr>
<tr>
<td>12</td>
<td>99.9</td>
</tr>
<tr>
<td>13</td>
<td>99.9</td>
</tr>
<tr>
<td>14</td>
<td>99.8</td>
</tr>
<tr>
<td>15</td>
<td>99.8</td>
</tr>
<tr>
<td>16</td>
<td>99.8</td>
</tr>
<tr>
<td>17</td>
<td>99.8</td>
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<tr>
<td>18</td>
<td>99.8</td>
</tr>
<tr>
<td>19</td>
<td>99.8</td>
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<tr>
<td>20</td>
<td>99.8</td>
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<td>99.8</td>
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<td>22</td>
<td>99.8</td>
</tr>
<tr>
<td>23</td>
<td>99.8</td>
</tr>
<tr>
<td>24</td>
<td>99.8</td>
</tr>
</tbody>
</table>

Note:
Calculated from data in Statistics New Zealand (2004).
Appendix 4: Deprivation

NZDep categorises geographical areas into centiles of deprivation, with each centile containing 10 percent of the total population. Within specific age groups, however, the age distribution is not even (i.e., generally speaking there are more young people living in areas of greater deprivation). The actual age distributions for NZDep 1996 and 2001 for each age group of this study are shown in Table 18.

Table 18: Actual proportions of each age group within each decile

<table>
<thead>
<tr>
<th>Deprivation centile</th>
<th>Age at diagnosis (years)</th>
<th>10–14</th>
<th>15–19</th>
<th>20–24</th>
</tr>
</thead>
<tbody>
<tr>
<td>NZDep year</td>
<td>1996 %</td>
<td>2001 %</td>
<td>1996 %</td>
<td>2001 %</td>
</tr>
<tr>
<td>1</td>
<td>9.0</td>
<td>10.8</td>
<td>8.7</td>
<td>10.3</td>
</tr>
<tr>
<td>2</td>
<td>9.6</td>
<td>10.5</td>
<td>9.2</td>
<td>10.4</td>
</tr>
<tr>
<td>3</td>
<td>9.2</td>
<td>9.9</td>
<td>8.7</td>
<td>9.6</td>
</tr>
<tr>
<td>4</td>
<td>10.1</td>
<td>9.8</td>
<td>9.6</td>
<td>9.4</td>
</tr>
<tr>
<td>5</td>
<td>8.8</td>
<td>9.5</td>
<td>8.7</td>
<td>9.3</td>
</tr>
<tr>
<td>6</td>
<td>10.0</td>
<td>9.3</td>
<td>10.0</td>
<td>9.3</td>
</tr>
<tr>
<td>7</td>
<td>9.8</td>
<td>9.2</td>
<td>10.2</td>
<td>10.1</td>
</tr>
<tr>
<td>8</td>
<td>10.6</td>
<td>9.3</td>
<td>11.0</td>
<td>10.0</td>
</tr>
<tr>
<td>9</td>
<td>10.6</td>
<td>10.0</td>
<td>12.6</td>
<td>10.7</td>
</tr>
<tr>
<td>10</td>
<td>12.3</td>
<td>11.7</td>
<td>11.3</td>
<td>10.8</td>
</tr>
</tbody>
</table>
Appendix 5: Statistical Methods

This study was a population-based retrospective analysis of cancer in the 10–24-year-old age group. While the incidence rates represent the actual population rates for this group in the period of this study, the study population can also be seen as a sample from a longer period. On this basis, statistical techniques can be applied to calculate confidence intervals and tests of significance between different groups within the sample.

Age-specific incidence rates

Throughout this study, age-specific incidence rates (IR) are calculated for each five-year age group. The calculation is as follows:

\[
\text{Incidence rate for period} = \frac{\text{Number of new cases over period}}{\text{Person-years at risk over period}}
\]

where ‘person-years at risk’ equals the sum of the annual mean population at risk for the period, taken from the population estimates in Appendix 3.

Confidence intervals are calculated assuming that the incidence rate has a Poisson distribution, and therefore the log of the incidence rate is normally distributed. The Poisson distribution assumes that:

- the population size is infinite compared with the number of events (which is a close enough approximation for a rare event such as cancer in young people)
- the probability of \( y \) events in time period \( t \) is expressed as:
  \[
P(y) = \frac{(\lambda t)^y e^{-\lambda t}}{y!}
\]
  where \( \lambda \) represents the rate per unit time (eg, 120 per million person-years)
- events occur independently of each other (ie, one case of cancer does not alter the probability of someone else developing cancer)
- the rate is constant
- the probability of an event tends towards 0 as \( t \) becomes small
- in large populations, person-years at risk can be approximated by mean population at risk over time \( t \), and the rate = (number of events over time \( t \)) ÷ (mean population over time \( t \)).

The standard error of the log(IR) is calculated as \( \frac{1}{\sqrt{y}} \) and the 95 percent confidence interval is calculated as: \( IR \times e^{\pm 1.96SE} \) (Pearce 2003).

Comparison between incidence rates

Where incidence rates are compared between subgroups within each age group, an incidence rate ratio (IRR) has been calculated (which is equivalent to relative risk in a cohort study, or odds ratio in a case-control study). Calculation of \( p \)-values can be derived from the person-time version of the Mantel-Haenszel chi-square statistic (Breslow and Day 1987; Pearce 2003).
The 95 percent confidence interval for the IRR is given by $\text{IRR} \times e^{+1.96SE}$ where $SE = \sqrt{\frac{1}{y_1} + \frac{1}{y_2}}$, and $y_1$ and $y_2$ are the number of cases in the two groups being compared.

The $p$-value for assessing the significance of a difference between two incident rates is derived from the $z$-statistic:

$$z = \frac{\text{log}(\frac{1}{\text{IRR}})}{\sqrt{\frac{1}{y_1} + \frac{1}{y_2}}}$$

The confidence interval for the log of the IRR is: $\text{log}(\text{IRR}) \pm z \times SE$, where $z$ is the $z$-statistic (ie, $z = 1.96$ for the 95 percent confidence interval), and $SE$ is the standard error of the IRR.

Corresponding two-tailed $p$-values were determined from the $z$-statistic using:

$$p = e^{-(\frac{(83\pm351)z+562)}{703+165z}}$$

**Survival calculations**

In general, actuarial methods were used. Relative survival rates were calculated using data extracted from the *New Zealand Life Tables 1995–1997* (Statistics New Zealand 1998) as the comparison population.

Survival time was calculated as the difference between date of diagnosis as recorded in the New Zealand Cancer Registry and date of death. All other individuals were ‘censored’ at 30 June 2005, which was the last date for which dates of death were available and up to date.

The Cox proportional hazards model was used to test for differences between groups using all-cause mortality. The rationale for use of this model was that for most comparisons, the assumption of proportionality held. All-cause mortality was used due to lack of cause of death information for deaths after 31 December 2002, and concern about accuracy of cause of death as recorded and extracted from death certificates. The difference between all-cause and cancer-specific mortality for the age range of this study is <1 percent.
References


### Glossary and List of Abbreviations

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>CI</td>
<td>Confidence interval</td>
</tr>
<tr>
<td>DHB</td>
<td>District Health Board</td>
</tr>
<tr>
<td>IARC</td>
<td>The International Agency for Research on Cancer, part of the World Health Organization, is charged with the mission ‘to co-ordinate and conduct research on the causes of human cancer, the mechanisms of carcinogenesis, and to develop scientific strategies for cancer control. The Agency is involved in both epidemiological and laboratory research and disseminates scientific information through publications, meetings, courses, and fellowships.’ See <a href="http://www.iarc.fr">http://www.iarc.fr</a> (accessed 27 September 2005) for more information.</td>
</tr>
<tr>
<td>IACR</td>
<td>The International Association of Cancer Registries is a non-governmental organisation dedicated to fostering the aims and activities of cancer registries worldwide. See <a href="http://www.iacr.com.fr">http://www.iacr.com.fr</a> (accessed 12 October 2005) for more information.</td>
</tr>
<tr>
<td>ICCC</td>
<td>International Classification of Childhood Cancer is a classification scheme that groups cancers based on their ICD-O code. In this scheme there are 12 categories, each with a number of subcategories. This allows meaningful summaries based on logical groups, rather than strictly anatomical site. The current version is ICCC-3 (Steliarova-Foucher et al 2005).</td>
</tr>
<tr>
<td>ICD</td>
<td>International Classification of Diseases is used to code diagnoses and treatments. The current version in use in New Zealand is ICD-10-AM-II (NCCH 2000). There is also an oncology specific version, of which the current version in use in New Zealand is ICD-O-3 (Percy et al 2000).</td>
</tr>
<tr>
<td>IR</td>
<td>Incidence rate, expressed as the number of cases per person-year. For example, if there are 10 cases of cancer in a population of 1,000,000 followed for five years, the incidence rate is two per million person-years.</td>
</tr>
<tr>
<td>IRR</td>
<td>Incidence rate ratio, expressed as the ratio of one incidence rate to another. For example, if males experience cancer at an incidence rate of two per million person-years, and females at a rate of four per million person-years, then the incidence rate ratio of males to females is 0.5.</td>
</tr>
<tr>
<td>NZDep</td>
<td>The New Zealand Index of Deprivation assigns a score based on regional deprivation, determined from data contained in the census. This provides a measure of deprivation for individuals living within each region. NZDep has been published for the 1991, 1996 and 2001 censuses (Crampton et al 1997; Salmond and Crampton 2002b; Salmond et al 1998).</td>
</tr>
<tr>
<td>NZHIS</td>
<td>The New Zealand Health Information Service is a Ministry of Health business unit that administers and analyses various data collections including the NMDS, the NZCR and the Mortality collection. See <a href="http://www.nzhis.govt.nz">http://www.nzhis.govt.nz</a> (accessed 12 October 2005) for more information.</td>
</tr>
</tbody>
</table>