Australian National University

ACT Asbestos Health Study II

Data Linkage Study on the Risk of Mesothelioma and Other Cancers in Residents of Affected Properties in the ACT

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We acknowledge and celebrate the First Australians on whose traditional lands this study was conducted, and pay our respect to the Elders past, present and future.

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Plain language summary

Asbestos is known to cause mesothelioma, a cancer of the tissue that lines internal organs including the lungs. It has also been linked to several other cancers. The aim of this study was to examine whether rates of mesothelioma and other selected cancers were elevated among people who had ever lived in a home with loose fill asbestos insulation in the Australian Capital Territory (ACT). To do this, we compared the rates of these cancers among people who had lived in these homes, to the rest of the ACT population. This study is an extension of an initial study, both commissioned by the ACT Government.

This study used historical data on addresses, cancer diagnoses and deaths collected over many years across Australia. The data we accessed did not allow us to identify any individual and we did not approach or contact anyone whose data were included in the study.

We observed a 2-to 3-fold higher rate of mesothelioma among men who lived in a home with loose fill asbestos compared to the rest of the male ACT population. This equates to an excess of 7 to 8 mesothelioma cases among men from affected homes between 1983 and 2019. We also observed slightly higher rates of lung cancer in women, colorectal cancer in both men and women, and prostate cancer in men who had ever lived in an affected home, compared to the rest of the ACT population. For many of the other cancers examined, there were too few cases to enable us to quantify with any certainty if rates were higher (or lower) among people who had ever lived in an affected home.

When interpreting the findings, it is important to keep in mind that the data we used were originally collected for administrative purposes and lacked detail on individual context. This meant that we could not account directly for other causes of cancer, for example past exposure to asbestos in the workplace (rather than at home), smoking or socioeconomic status. In addition, there was a small possibility that a difference in rates was observed due to chance alone, rather than because a true difference exists.

Nevertheless, our findings are consistent with prior research, which shows strong evidence that workplace levels of asbestos exposure cause mesothelioma and lung cancer, with well-described biological processes. Whether non-occupational levels of exposure are sufficient to cause these cancers is less certain. There is limited evidence from other studies that asbestos causes colorectal or prostate cancers.

We conclude that men who lived in homes with loose fill asbestos insulation in the ACT have an increased risk of mesothelioma, although their overall risk remains low (less than 1 case per 10,000 people per year). We could not confidently draw associations between having lived in affected homes and increased risks of the other examined cancers.

Abbreviations

ACD	Australian Cancer Database
ACT	Australian Capital Territory
AIHW	Australian Institute of Health and Welfare
ANU	The Australian National University
APP	Australian Privacy Principles
ARP	Affected residential property
CI	Confidence interval
DIY	Do-it-yourself
HREC	Human Research Ethics Committee
IARC	International Agency for Research on Cancer
ICD	International Classification of Diseases
MCD	Medicare Consumer Directory
NDI	National Death Index
NT	Northern Territory
SIR	Standardised incidence ratio
SURE	Secure Unified Research Environment

Introduction

Background

Asbestos is the common name for a group of naturally occurring minerals that are found in rock and mined for commercial and industrial use. Asbestos consists of extremely flexible fibres that easily separate from one another and quickly become airborne. When inhaled, the thinnest fibres can deposit deeply into the lungs where they can bioaccumulate.[1]

The International Agency for Research on Cancer (IARC) monographs classify all forms of asbestos (chrysotile, crocidolite, amosite, tremolite, actinolite and anthophyllite) as carcinogenic to humans.[1] Asbestos causes mesothelioma and is the attributed cause in more than 80% of pleural mesothelioma cases in men.[2] Most mesothelioma manifests in the lining around the lungs (pleura); and can also occur in the membranes surrounding the abdomen (peritoneal), heart (pericardial) or testes. Asbestos also causes cancers of the lung, larynx and ovary but asbestos exposure is not the predominant risk factor for these cancers. There is more limited evidence that asbestos is linked to cancers of the pharynx, stomach and colorectum.[1]

The risk of mesothelioma increases with the dose, duration and frequency of asbestos exposure. Moreover, the time between exposure and diagnosis (latency period) — estimated to be 20–40 years — is likely to be shorter with higher levels of exposure.[3] While the risk of mesothelioma from occupational exposure to asbestos is well-known, there is increasing concern arising from short-term or low-level exposures in non-occupational settings. This includes household exposure (from asbestos-containing material in the home or renovation/do-it-yourself (DIY) projects), domestic/para-occupational exposure (from fibres brought home by asbestos workers) and neighbourhood exposure (from living near industrial or natural sources).

Loose fill asbestos in the ACT

Between 1968 and 1979, D. Jansen & Co. Pty Limited and its successor firm installed loose fill asbestos (finely crushed raw asbestos) into the roof spaces of homes in the Australian Capital Territory (ACT) region. The type of asbestos used in most of the affected homes was amosite, with crocidolite found in two homes.[4] In the 1980s, the use of asbestos in buildings in Australia started to be phased out as the health risks began to be understood. All forms of asbestos have been banned in Australia since 2004.

Over 1989–93, the Commonwealth and ACT Governments jointly managed the removal of visible and accessible loose fill asbestos insulation from over 1,000 homes (of 65,000 surveyed) in the ACT.[4] At this time it was known that residual fibres remained within the structure of occupied properties due to the challenges of the removal task and the nature of asbestos itself. Subsequent assessments in 2013–14 revealed that fibres had migrated into the living areas in many affected homes. Since 2014, the ACT Government has supported the removal of affected

properties in Canberra through a voluntary buyback and demolition program. The ACT Government maintains a list of known properties that were or are affected by loose fill asbestos insulation.[5]

In 2015, the ACT Government contracted the Australian National University (ANU) to conduct the ACT Asbestos Health Study to assess the health risks of loose fill asbestos insulation in the ACT. The data linkage component of that study quantified the rates of mesothelioma and other cancers in past and current residents of homes with loose fill asbestos, over the period 1983—2013. The main finding was that men who had lived in these homes were 2.5 times as likely to develop mesothelioma as the rest of the male ACT population.[6]

In 2022, the ACT Health Directorate commissioned the ANU to conduct an extension of the initial data linkage study incorporating the latest available data. This included six additional years of data and six additional affected homes that were not identified in the previous study.

Aims and research questions

The primary aim of this study was to examine whether the rates of mesothelioma and other cancers related to, or potentially related to, asbestos exposure were higher among people who had ever lived in a known affected residential property (ARP) than among people who had never lived in an ARP in the ACT. The study also examined other cancers not known or thought to be associated with asbestos exposure (control outcomes).

Specific research questions

1. What are the relative rates of mesothelioma and other cancers in relation to having lived in an ARP, after adjusting for demographic characteristics?
2. What are the relative rates of control outcomes in relation to having lived in an ARP, after adjusting for demographic characteristics?

Methods

Study population

The study population included all individuals in the Medicare Consumer Directory (MCD) who had an address in the ACT at any time between 1983 and 2019.

For each member of the study population, MCD data was linked to the list of ARPs, the Australian Cancer Database (ACD) and the National Death Index (NDI). Linkage was performed by the Australian Institute of Health and Welfare (AIHW), the data integrating authority of this project (see Appendix 2 for details).

We excluded individuals from the study if they had:

a) missing data on their date of birth, date of death (if a death was recorded) or sex
b) invalid dates (for example, where the date of entry into the study or date of cancer diagnosis occurred outside the interval between date of birth and date of death).

Data sources

Medicare Consumer Directory (1983–2019)

Medicare is Australia's universal health care insurance scheme, which covers all Australian citizens and permanent residents of Australia. Medicare is administered by the Department of Social Services through its executive agency, Services Australia. Services Australia collects personal information from customers at the time of enrolment, including name, sex, date of birth and address. The main repository for these data is the MCD.

An individual is required to notify Services Australia, by phone, online or in person, if they change their address. A history of these changes is stored in the MCD, resulting in multiple address records for every individual. A *start date* is associated with each address record in the MCD, which is the date Services Australia was notified of the change. There may be a delay between the actual change of address and this change being recorded in the MCD.

Services Australia collects both residential and mailing addresses. However, it is not mandatory for individuals to provide a residential address. Only mailing addresses were provided to the AIHW for the purposes of data linkage. While mailing and residential addresses are likely to be the same for most of the population, a proportion of addresses in the data are non-residential, including post office box addresses. Multiple members of a family may be associated with a single Medicare registration, where the mailing address for all members is the address nominated by the card contact. An individual may also have more than one Medicare registration, for example when their family circumstances change.

List of Affected Residential Properties (as of July 2022)

The study team constructed a list of ARPs comprising 1,095 addresses for this study. This list combined information from two data sources.

The first is a publicly available register of 1,029 known residential properties in Canberra that contain, or have contained, loose fill asbestos insulation.[5] Each property is identified by its full street address (house number, street name and street type) and alternative street address where applicable, such as corner blocks. We received additional information from the ACT Government on whether each property had been acquired by the ACT. We considered all properties in this list that have been acquired by the ACT to be ARPs up to the date they were added to register. Properties on the register that have not been acquired by the ACT were considered to be ARPs for the entire study period.

We augmented this list by a second file, received from the ACT Government, containing the street addresses of 66 properties that were demolished prior to the announcement of the voluntary buyback program in 2014. This file included the year the property was demolished. We considered all addresses in the second file to be ARPs up to the year the property was demolished. Properties that were built at the same address after demolition were not considered to be ARPs.

The Australian Cancer Database (1982–2019)

The ACD contains data on new cases of cancer diagnosed in Australia since 1 January 1982, excluding basal and squamous cell carcinomas of the skin. Cancer is a notifiable disease in all Australian states and territories. The relevant legislation requires certain individuals and organisations to notify all new cases of cancer to the jurisdiction's central cancer registry. These registries supply data annually to the AIHW, which cleans and standardises the data, notifies the registries of inter-state duplicates, and produces the ACD. Reporting of newly diagnosed cancers has been mandatory in most but not all jurisdictions since 1982.[1]

The National Death Index (1982–2019)

The NDI is a database of death records that is used for epidemiological studies. It is used strictly for health and medical research approved by the AIHW Ethics Committee. The NDI contains records of all deaths occurring in Australia since 1980 obtained from the Registrars of Births, Deaths and Marriage in each state and territory.

[1] Mandatory reporting in: ACT — 1994; New South Wales — 1972; Northern Territory (NT) — 1991; Queensland — 1982; South Australia — 1977; Tasmania — 1992; Victoria — 1982; Western Australia — 1981

Study variables

Outcomes

We examined incidence rates of mesothelioma as the primary outcome of interest, as well as rates of the following cancers: pharyngeal, oesophageal, stomach, colorectal, liver, laryngeal, lung and ovarian cancers. These were the same cancers examined in the initial 2015 study, with the addition of liver and oesophageal cancers based on a review of more recent literature. The initial cancers were selected based on the IARC review of evidence on cancer risks associated with asbestos exposure.[1] We also examined other cancers not known or thought to be related to asbestos exposure as control outcomes: melanoma, and prostate, kidney and bladder cancers. All cancers were identified based on International Classification of Diseases (ICD) and Related Health Problems, 10th revision (ICD-10) as recorded on the ACD. Table 1 shows all cancer outcomes examined in this study and their ICD-10 codes.

Table 1: ICD-10 codes for cancer outcomes

Cancer	International Classification of Diseases-10 codes
Mesothelioma	C45
Other cancers	
Pharyngeal	C09 — C14
Oesophageal	C15
Stomach	C16
Colorectal	C18 — C20
Liver	C22
Laryngeal	C32
Lung	C33, C34
Ovarian	C56
Control outcomes	
Melanoma	C43
Prostate	C61
Kidney	C64
Bladder	C67

Table notes

Jurisdictional cancer registries code records based on International Classification of Disease for Oncology, 3rd edition, which are then mapped to ICD-10 in the Australian Cancer Database.

Exposure and other variables

We defined exposure in terms of whether, and when, someone had an ARP address. Each day that an individual was followed in the study was accumulated as their 'person-time'. We classified each day as either exposed or unexposed. Person-time was classified as exposed

based on the start date of their first ARP address as recorded in the MCD. Unexposed time included any time prior to this, or total person-time if no ARP address was recorded. Individuals who moved into an ARP during the study period may contribute both unexposed and exposed person-time. Classification of exposure is further detailed under statistical analysis.

Sex, age and calendar year, used for adjustments in the analysis, were sourced from the MCD. Age was calculated based on the 15th day of the recorded month and year of birth. Age and calendar year were treated as time-varying variables — that is, variables whose values increase with follow-up time, thus accounting for 'acquired' age and time during the study period. We considered adjusting for area-level socioeconomic status. However, published Socio-Economic Indexes for Areas measures are a poor measure of individual-level disadvantage in the ACT.[7]

Statistical analysis

Main analysis

We calculated incidence rates as the number of incident cancers divided by total person-time at risk. Person-time at risk is the time observed from study entry, defined as the start date of the individual's first address in any state in the MCD, to the first occurrence of any of the following:

a) date of cancer diagnosis
b) date of death
c) age 100
d) end of the study period (31 December 2019).

Each cancer was analysed separately. Individuals who had a cancer diagnosis before their entry into the study were excluded from analysis of that particular cancer.

All person-time units (days) contributed by each individual into the study were classified as exposed or unexposed. For individuals who ever lived in an ARP, all person-time before the start date of the first ARP address, plus a lag period (see below), was classified as unexposed. All subsequent person-time was classified as exposed even if the individual moved from the ARP. All person-time contributed by those who had never lived in an ARP was classified as unexposed. Person-time for individuals who first moved into an ARP address after the property was demolished or after the property was acquired by the ACT was classified as unexposed.

We used a lag period of 10 years as a minimum possible latency period — the minimum expected amount of time between first exposure (living in an ARP) and cancer diagnosis. Application of a lag period meant that outcomes were only attributed to exposure if the diagnosis occurred at least 10 years after the start date of living in an ARP. Individuals who enrolled with an ARP address at the start of the MCD collection were assumed to have been living there for at least 10 years, therefore a lag period was not applied to these individuals.

We estimated the standardised incidence ratio (SIR) for each cancer outcome, separately in males and females, with exact Poisson 95% confidence intervals (CI). First, we calculated age- and calendar period-specific incidence rates for the unexposed person-time in the study. This was done for five-year age and calendar period bands, which were widened if there were no individuals in any stratum. We applied these rates to the person-time by age and calendar period in the exposed group and summed across all strata to calculate the total expected number of cases. The SIR was calculated as the ratio of observed cases in the exposed group to expected cases calculated as described. We estimated SIRs separately for males and females due to different levels of expected asbestos exposure from both household and occupational sources.

We did not report SIRs where there were less than six observed cases in the exposed group to minimise statistical disclosure risk. Note, however, SIRs based on <6 cases in the exposed group were unlikely to be sufficiently powered to detect effect sizes within reasonably expected magnitudes.

We also calculated the number of excess cases by subtracting the number of estimated expected cases from the number of observed cases.

Sensitivity analyses

We conducted the following separate sensitivity analyses:

1. A lag period of 5 years was applied.
2. A lag period of 15 years was applied.
3. Individuals who ever had a post office box address were excluded, as we were unable to determine their exposure status during this time. We did not exclude individuals who were already exposed (had an ARP address) prior to their first post office box address.
4. A 10-year lag period was applied to those who enrolled with an ARP address at the start of the MCD collection in 1983–84.
5. Participants were censored at 85 years old.
6. Approximately 600 individuals had ties of two or more addresses with the same start date but different ARP status. In the main analysis, we broke ties by selecting the ARP address. In this sensitivity analysis, we broke ties by selecting the non-ARP address.

Projection of mesothelioma cases 2020–2024

We estimated the projected number of mesothelioma cases in men who ever lived in an ARP over the five years following the study period. To do this, we used coefficients from a Poisson regression model based on observed data (1983-2019). This model fitted the observed number of cases where the linear predictor included covariates for the calendar year and exposure status, and the log of the expected number of cases as an offset (see Equation).

$$Observed\ cases \sim Poisson\ (\exp(\beta 0 + \beta 1(year) + \beta 2(exposure\ status) + \log(expected\ cases)))$$

β_0 = intercept, β_1 = coefficient for year, β_2 = coefficient for exposure status

To generate the expected number of cases for the model, we first calculated age-specific incidence rates for the unexposed person-time in the study (1983–2019). We applied these rates to the person-time by age and calendar year for both the exposed and unexposed groups and summed across age groups per year.

We used model coefficients and a synthetic expected number of cases to estimate the projected number of mesothelioma cases among exposed men over 2020–2024. To generate the expected number of cases for the projection, we derived person-time for exposed men who were at-risk for mesothelioma between 2020–2024. We obtained this by applying survival probabilities for men in the ACT by age.[8] Note this also included person-time contributed by men who first lived in an ARP over 2010–2014 (these men were considered unexposed in the main analysis due to application of the 10-year lag period).

We used the delta method with the gradients calculated using finite differences to calculate the 95% confidence interval for the number of projected cases.

Ethics

Ethics approvals

This study involved linkages of Commonwealth and jurisdictional data. The MCD, NDI and ACD are Commonwealth data, however state and territory governments retain ownership over their jurisdiction's data in the ACD.

We obtained data custodian approvals from each state or territory cancer registry for use of jurisdictional data in the ACD. The AIHW facilitated data custodian approvals for the use of the ACD, NDI and the MCD including acquisition of a Public Interest Certificate signed by the Australian Minister for Health and Aged Care for the use of the MCD.

We also obtained ethics approvals from the following committees:

- ACT Health Human Research Ethics Committee (HREC) (2022.ETH.00197)
- AIHW Ethics Committee (EO2022-5-1387)
- ANU HREC (2022/623)
- NT Health and Menzies School of Health Research HREC (2023-4513)

Privacy and waiver of consent

This study was compliant with all Australian Privacy Principles (APP) under section 95 of the Privacy Act 1988 except APP6 (use or disclosure of personal information). We sought a waiver of consent for the use or disclosure of personal information from the ANU HREC on the basis that provisions under the National Statement on Ethical Conduct in Human Research 2023 chapter 2.3.10 were satisfied.[9]

In pursuing this waiver, we considered that: the study did not involve direct recruitment of contact with any participant, obtaining consent is impracticable due to the large number of participants including those no longer living, and there is sufficient protection of privacy and confidentiality (see Appendix 2) including that we will take measures to minimise the risk of re-identification from published results. We considered the benefits from this study to outweigh the harms associated with not seeking consent, including helping to inform residents about their risk of exposure and to inform policy on asbestos insulation in the ACT.

Secure data storage and access

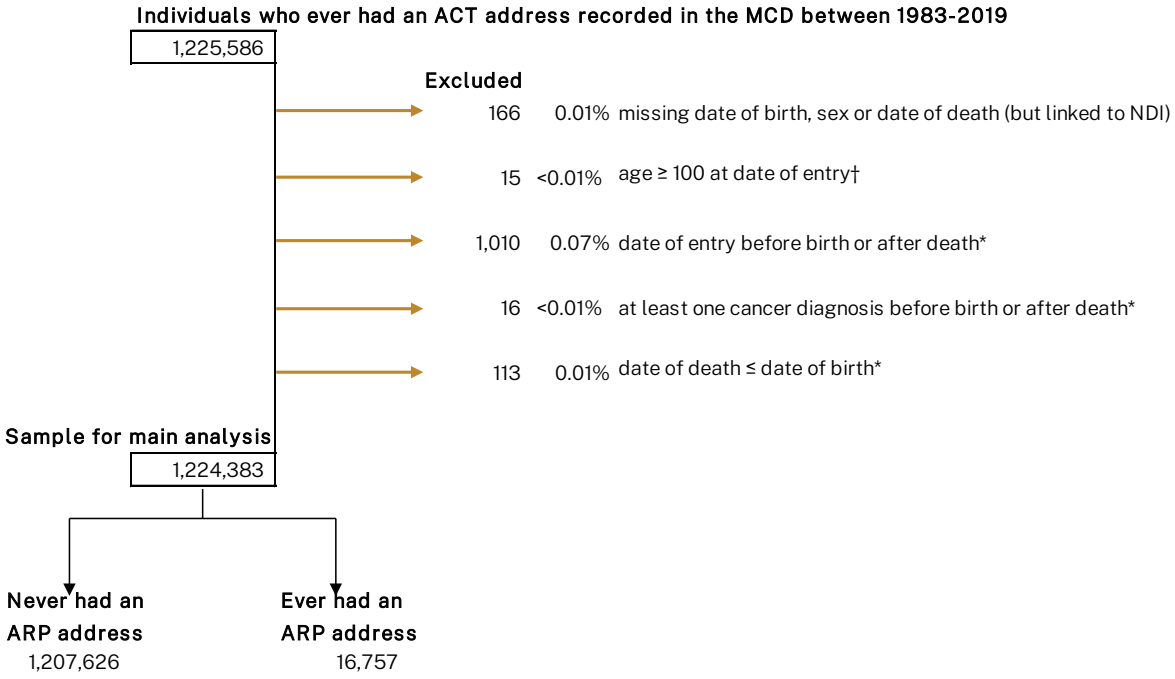
All linked (de-identified) data were stored, accessed and analysed in the Secure Unified Research Environment (SURE) computing environment through the Sax Institute. Access to SURE is via the Australian Academic and Research Network or the internet using an encrypted connection from researchers' local computers. Only two members of the study team carrying out analysis were provided access to the data. All study results were downloaded from SURE under curator surveillance and are stored on secure, password-protected networks at the ANU.

Results

Description of the study population

A total of 1,225,586 individuals were identified from the MCD who ever had an ACT address recorded between 1 October 1983 and 31 December 2019. After excluding 1,203 (0.1%) individuals, including those with missing or temporally inconsistent dates, our study population comprised 1,224,383 individuals. Figure 1 depicts a flow diagram of the sample selection.

Figure 1: Flow diagram of sample selection



ACT = Australian Capital Territory
MCD = Medicare Consumer Directory
NDI = National Death Index
ARP = Affected residential property

†Date of entry is the date of first address recorded in the MCD
*Not mutually exclusive

Of our study population, 16,757 individuals (1.4%) had at least one address that was identified as an ARP. These individuals were followed for an average of 18 years, contributing a total of 0.3 million exposed person-years (after accounting for the 10-year lag period). The remaining 1,207,626 individuals (99%) had never lived in an ARP. The total contribution of unexposed person-time from all participants was 29 million person-years.

Among the study population, 45,423 individuals (3.7%) had at least one cancer recorded on the ACD (including cancers not examined in this study) during the study period, with a total of 49,094 cancer cases diagnosed. A total of 78,567 individuals (6.4%) had a death recorded on the NDI in the same period. Table 2 shows the demographic characteristics of those who ever/never lived in an ARP.

Table 2: Demographic characteristics of the study population, 1983–2019

	Ever lived in an ARP (exposed)		Never lived in an ARP (unexposed)	
Characteristic	**n**	**%**	**n**	**%**
Total sample	16,757		1,207,626	
Sex				
Female	8,638	52%	619,195	51%
Male	8,119	48%	588,431	49%
Year of entry into study*				
1983–1989	12,658	76%	598,784	50%
1990–1994	1,367	8%	106,071	9%
1995–1999	963	6%	102,014	8%
2000–2004	762	5%	92,998	8%
2005–2009	577	3%	91,831	8%
2010–2014	398	2%	103,773	9%
2015–2019	32	0%	112,155	9%
Age at entry into study*				
0–9	6,221	37%	450,982	37%
10–19	3,306	20%	154,829	13%
20–29	2,885	17%	235,999	20%
30–39	2,058	12%	150,274	12%
40–49	1,101	7%	71,664	6%
50–59	638	4%	52,034	4%
60–69	347	2%	55,410	5%
70–79	150	1%	29,181	2%
80–100	51	0%	7,253	1%
Year of first recorded exposure†				
1983–1989	6,655	40%	NA	NA
1990–1994	2,653	16%	NA	NA
1995–1999	2,030	12%	NA	NA
2000–2004	2,236	13%	NA	NA
2005–2009	1,627	10%	NA	NA
2010–2014	1,463	9%	NA	NA
2015–2019	93	1%	NA	NA
Age at first recorded exposure†				
0–9	3,884	23%	NA	NA
10–19	2,678	16%	NA	NA
20–29	3,493	21%	NA	NA
30–39	3,076	18%	NA	NA
40–49	1,901	11%	NA	NA
50–59	943	6%	NA	NA

	Ever lived in an ARP (exposed)		Never lived in an ARP (unexposed)	
Characteristic	n	%	n	%
Age at first recorded exposure†				
60–69	433	3%	NA	NA
70–79	207	1%	NA	NA
80–89	119	1%	NA	NA
90–100	23	0%	NA	NA

Table notes

*Entry into the study is defined as the date of first address recorded in the Medicare Consumer Directory (MCD), regardless of state.

†Year/age of first recorded exposure is the year/age at first ARP address in the MCD.

†First recorded exposure for those who enrolled at the start of the MCD (1983–84) will not reflect the time of moving into an ARP.

NA=not applicable

Mesothelioma cases

There were 356 incident cases of mesothelioma among males in the study population (exposed and unexposed) during the study period. The total number of incident cases of mesothelioma among women is not reported (due to <6 cases in the exposed group). There were 12 cases among men and <6 cases among women who ever lived in an ARP. All of these cases were diagnosed at least 10 years after the person's first recorded exposure, that is, after the lag period.

Men who ever lived in an ARP were diagnosed at a slightly younger age than those who had never lived in an ARP (Table 3). The median time to diagnosis after the first recorded exposure was 26 years (Q1, Q3: 16, 30). This is a minimum estimate, as 6 of the 12 men had enrolled at the start of the MCD collection with an ARP address, meaning the date of their first recorded ARP address may be much later than the date of moving into that address. We did not report these statistics for women due to the small number of cases among exposed women.

Table 3: Mesothelioma cases in the ACT by exposure status, 1983–2019

	Ever lived in an ARP (exposed)		Never lived in an ARP (unexposed)	
	Male	**Female**	**Male**	**Female**
Total number of cases	12	<6	344	62
Age at diagnosis				
0-49	<6	<6	18	10
50-59	<6	<6	38	12
60-69	<6	<6	106	18
70-79	<6	<6	123	13
80-100	<6	<6	59	9
Mean (SD)	67 (17)	np	70 (11)	64 (14)
Median (IQR)	70 (27)	np	71 (14)	65 (17)
Range	37-90	np	17-93	31-93
Diagnosis year				
Earliest	1996	np	1985	1984
Latest	2019	np	2019	2019
Time to diagnosis (years) †				
Mean (SD)	24 (18)	np		
Median (IQR)	26 (14)	np		
Range	13-36	np		

Table notes
† From first recorded exposure (date of first ARP address in the MCD)
np=not provided (to minimise statistical disclosure risk)

Risk of cancer associated with living in an ARP

The number of observed and expected cases for all cancers examined are shown in Figure 2, together with a forest plot of SIRs. Crude rates are presented in Appendix Table A3–1.

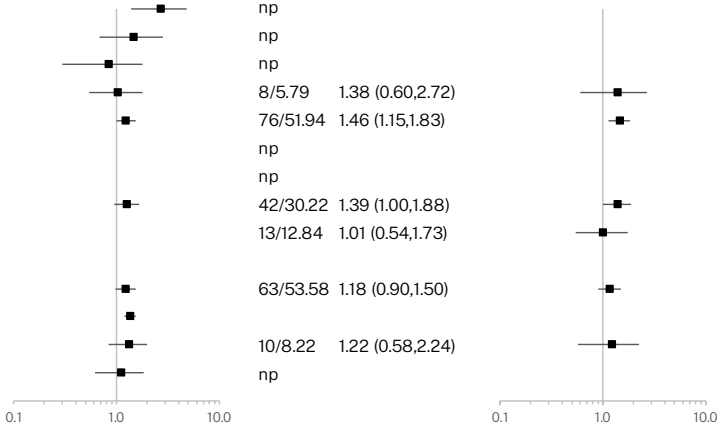
Mesothelioma

Among men, the crude mesothelioma rates were 0.93 cases (95% CI 0.48–1.58) per 10,000 exposed person-years and 0.24 cases (95% CI 0.22–0.27) per 10,000 unexposed person years (Appendix Table A3–1). After adjustment for age and calendar time, the mesothelioma rate among men who ever lived in an ARP was 2.7 times (SIR = 2.72, 95% CI 1.41–4.76) that of men who never lived in an ARP (i.e., 172% higher) (Figure 2). This equated to an excess 7.6 cases (12 minus 4.4) of mesothelioma over the study period (1983-2019) among men who ever lived in an ARP.

Among women, the crude mesothelioma rate was 0.04 cases (95% CI 0.03–0.05) per 10,000 unexposed person years. The crude mesothelioma rate among exposed women and SIRs are not reported due to <6 diagnosed cases among women who ever lived in an ARP.

Figure 2: Observed (O) and expected (E) case numbers in the exposed population and standardised incidence ratios (SIR)

	Males		Females	
Type of cancer	**O/E**	**SIR (95% CI)**	**O/E**	**SIR (95% CI)**
Mesothelioma	12/4.41	2.72 (1.41,4.76)	np	
Pharyngeal	9/6.08	1.48 (0.68,2.81)	np	
Oesophageal	6/7.26	0.83 (0.30,1.80)	np	
Stomach	12/11.55	1.04 (0.54,1.81)	8/5.79	1.38 (0.60,2.72)
Colorectal	82/65.97	1.24 (0.99,1.54)	76/51.94	1.46 (1.15,1.83)
Liver	np		np	
Laryngeal	np		np	
Lung	53/41.69	1.27 (0.95,1.66)	42/30.22	1.39 (1.00,1.88)
Ovarian			13/12.84	1.01 (0.54,1.73)
Control outcomes				
Melanoma	84/68.05	1.23 (0.98,1.53)	63/53.58	1.18 (0.90,1.50)
Prostate	225/165.1	1.36 (1.19,1.55)		
Kidney	23/17.13	1.34 (0.85,2.01)	10/8.22	1.22 (0.58,2.24)
Bladder	15/13.53	1.11 (0.62,1.83)	np	



np=not provided

Figure notes

Forest plots show point estimates of SIRs (filled squares) and 95% confidence intervals (horizontal lines). SIRs are plotted on a log scale.

Other cancers

After adjustment for age and calendar time, the rate of colorectal cancer was 24% higher among men (SIR = 1.24, 95% CI 0.99–1.54) and 46% higher among women (SIR = 1.46, 95% CI 1.15 — 1.83) who ever lived in an ARP, compared to those who never lived in an ARP (16 and 24 excess cases, respectively). The adjusted rate for lung cancer was 39% higher in women who ever lived in an ARP than among those who never lived in an ARP (SIR = 1.39, 95% CI 1.00–1.88) (12 excess cases). For all other cancers examined, we did not observe statistically significant higher-than-expected rates, noting that for rarer cancers there may not have been sufficient statistical power to detect associations should these exist (see Discussion).

Control outcomes

After adjustment for age and calendar time, the rate of prostate cancer was 36% higher among men who ever lived in an ARP, compared to those who had not (SIR = 1.36, 95% CI 1.19 — 1.55). For all other control cancers examined, we did not observe statistically significant higher- nor lower-than-expected rates in those who had lived in an ARP. At the same time, we could not conclude that the rates of these control cancers were similar among those who ever lived in an ARP and those who had not, as the confidence intervals were too wide.

Sensitivity analyses

The SIR for mesothelioma in men remained statistically significant with a magnitude above two across all sensitivity analyses. The SIRs for colorectal and lung cancers were affected in a few of the sensitivity analyses. The findings for most other cancers remained in the same direction with a few exceptions.

Applying a lag period of 5 years resulted in additional cases attributed to exposed person-time, while a lag period of 15 years resulted in classification in the opposite direction (cases re-attributed to unexposed). We no longer saw an association for colorectal cancer in men and lung cancer in women after applying a lag period of 5 years (Appendix Figure A4-1) but saw slightly higher SIRs for these cancers after applying a lag period of 15 years (Appendix Figure A4-2).

When we excluded participants who ever had a post office box, unless this occurred after their first ARP address (constituting 40% of the study population), the SIR for mesothelioma in men became slightly weaker (SIR = 2.07 95% CI 1.03–3.70) and we no longer observed associations for colorectal cancers in either sex nor for lung cancer in women (Appendix Figure A4-3).

Study results were generally not sensitive to application of a 10-year lag among those who entered at the inception of the MCD; the exception to this was a stronger association for melanoma in men (SIR = 1.28 95% CI 1.02–1.59) (Appendix Figure A4-4). There was little material impact when we censored participants at 85 years old or broke ties by choosing non-ARP addresses (Appendix Figure A4-5 and Figure A4-6).

Projected mesothelioma cases associated with living in an ARP over 2020–2024

Parameter coefficients from the fitted Poisson regression model are shown in Table 4. Note that the estimated standardised incidence ratio of 2.73 is very similar to that from the main analysis (2.72), and that there was no evidence for a calendar trend in mesothelioma cases.

Our model projected 5.2 (95% CI 2.9–9.3) mesothelioma cases over 2020–2024 among men who ever lived in an ARP at least 10 years ago (i.e., allowing for the 10-year lag period). This amounts to 3.3 (95% CI 0.3–6.3) additional cases of mesothelioma in these men compared to if they had the same age-adjusted rates of mesothelioma as the rest of the male ACT population over 1983–2019.

Table 4: Parameter coefficients (exponentiated with base e) from the fitted Poisson regression model

Covariate	Estimate (95% CI)
Exposure status	2.73 (1.54 – 4.86)
Year	1.00 (0.99 — 1.01)

Discussion

Summary of main findings

We observed a 172% higher-than-expected rate of mesothelioma in men who ever lived in an ARP between 1983 and 2019 compared to the rest of the male ACT population. This equated to 7.6 more cases of mesothelioma over this time period among men who ever lived in an ARP than we would have expected if they had not lived in an ARP. We also observed modestly elevated rates of lung cancer in women, of colorectal cancer in both men and women and of prostate cancer in men. We did not observe higher-than-expected rates of the other cancers examined. See Box 1 for a summary of key results.

This was an extension to an initial data linkage study commissioned by the ACT Government to study the risks of cancer in residents of homes with loose fill asbestos insulation in the ACT. These studies are unique as this type of loose fill, finely crushed asbestos insulation has not been used widely in Australia or the rest of the world. Compared to the first study, the current study used newer linkage methods (see Appendix 1) and six further years of data. We observed generally consistent results to the initial study; however, it is important to note that the findings of the two studies are not independent and consist of largely overlapping populations.

Box 1. Summary of key results: adjusted relative rates and estimated excess cases for selected cancer outcomes (1983–2019)

Cancer	Adjusted relative rate	Excess cases among exposed population over 1983-2019
Males		
Mesothelioma	Rate 2.72-fold that of unexposed population (172% higher)	7.6 excess cases
Colorectal	Rate 1.24-fold that of unexposed population (24% higher)	16 excess cases
Females		
Lung	Rate 1.39-fold that of unexposed population (39% higher)	12 excess cases
Colorectal	Rate 1.46-fold that of unexposed population (46% higher)	24 excess cases

Notes
Relative rates should be considered alongside their confidence intervals in **Figure 2**.

Interpretation of findings in the context of other evidence

Mesothelioma

We observed a higher-than-expected rate of mesothelioma in men who ever lived in an ARP. This is consistent with the well-established causal link between asbestos exposure and

mesothelioma, primarily based on studies of occupational sources of exposure. There is much less known about non-occupational exposures.

Although the risk of mesothelioma from neighbourhood asbestos exposure is increasingly recognised,[10, 11] few studies have analysed household exposure as distinct from neighbourhood or domestic/para-occupational exposures. Household exposure has generally meant exposure to asbestos-containing materials in or around the home or from home maintenance/DIY projects. We identified three studies related to household exposure, but not involving loose-fill asbestos insulation specifically. One reported increased risk of mesothelioma associated with certain asbestos-containing materials (e.g. asbestos-cement roofing and tailings) around the home (OR between 1.9—2.4).[12] Two other studies did not report associations to either asbestos-containing materials around the home [13] or to DIY activities.[14]

Notably, the sources or activities that constitute household exposure are varied and not consistently defined across studies. In addition, the levels and pathways of asbestos exposure from these sources are generally unclear or unknown, including in our study. Although non-occupational exposure is generally thought to be much lower than in occupational settings,[15] we do not know the fibre measurements in ARPs and expect exposure to vary across homes. In some cases, asbestos-like dust had been visible on household furnishings.[5] Thus, the ability to compare our findings to existing studies of household exposure is limited.

We identified one study that examined passive exposure to asbestos insulation, in buildings of a French university campus. This study did not find a difference in the prevalence of pleural anomalies between those who worked in these buildings (and were not maintenance staff) and the unexposed group.[16] However, a later case study reported five mesothelioma cases among campus employees (with no other occupational, household or domestic exposures identified) that were likely induced by workplace passive exposure to asbestos.[17]

While our study estimated that men who ever lived in ARPs had 2.7 times the rate of mesothelioma (or 172% higher) than the rest of the male ACT population, the level of uncertainty suggested that this estimate may have been anywhere from 40% to 380% higher. Further, our estimate may be biased given we did not know study participants' history of asbestos exposure, occupational or otherwise. For example, if those who ever lived in ARPs were more likely to have occupational exposure to asbestos (for example, through professional connection with the company that installed asbestos insulation) than the unexposed population, we would have overestimated the risk of mesothelioma associated with living in an ARP. Conversely, if the unexposed population was more likely to have occupational exposure to asbestos than those who ever lived in ARPs, we would have underestimated the SIR for mesothelioma.

The higher-than-expected rate of mesothelioma in men who ever lived in ARPs, but not women, may relate to the possibility that men were more likely to enter roof spaces or carry out

structural maintenance.[18] However, it is possible that future studies with added observation time may see an effect in women due to as yet undiagnosed cases. Mesothelioma is challenging to study because of its low incidence and long latency period, requiring large and long-term cohort studies to achieve satisfactory statistical power.

Lung cancer

We observed a modestly elevated rate of lung cancer in women who ever lived in an ARP. This is consistent with strong evidence from occupational studies that asbestos exposure increases the risks of lung cancer incidence and mortality.[10] Furthermore, the biological mechanisms for asbestos-related causation of lung cancer are well-described and involve similar mechanisms to those in the development of mesothelioma. These include genetic and molecular alterations that cause dysregulation of cell growth functions, activation of oncogenes and chronic inflammation due to trapped fibres.[1]

Nevertheless, the risk of lung cancer from asbestos exposure in non-occupational settings is less certain. Apart from the first study of ARPs in the ACT,[6] we identified only one other study on the link between household exposure and lung cancer. The latter case-control study reported an increased risk of lung cancer in women associated with the use of tremolite-based household whitewash in New Caledonia.[19]

The current study estimated a slightly higher rate of lung cancer in women who ever lived in ARPs but did not find an elevated rate in men. The first study by our research team did not report an association between having lived in an ARP and lung cancer in men or women. This is likely, at least in part, due to the extra observation time in the current study that provided additional statistical power (sensitivity to detect a statistically significant result).

Nonetheless, we cannot rule out that the SIRs for lung cancer may be biased given that we lacked data on other risk factors that study participants' may have had, most importantly tobacco use. For example, if those who ever lived in ARPs were more likely to smoke than the unexposed population, we would have overestimated the risk of lung cancer associated with living in an ARP (and vice versa). Additionally, co-exposure to smoking and asbestos fibres is recognised to amplify the risk of lung cancer.[20] Due to the known relationships between smoking and lung cancer, and its interaction with asbestos to cause lung cancer, we were limited in our ability to interpret findings for lung cancer without knowledge of smoking.

Laryngeal and ovarian cancers

The IARC considers asbestos to be a cause of laryngeal and ovarian cancers. We did not have enough cases to report results for laryngeal cancer. We did not observe a higher-than-expected rate for ovarian cancer in women who ever lived in ARPs but had limited statistical power to detect an association.

Gastrointestinal tract cancers (pharyngeal, oesophageal, stomach, colorectal cancers including liver cancer)

We observed slightly higher-than-expected rates of colorectal cancer among those who ever lived in an ARP, but not for any of the other gastrointestinal cancers examined. These findings were broadly consistent with existing evidence on the relationship between occupational levels of asbestos and gastrointestinal cancers.

Inhaled asbestos can reach tissues of the gastrointestinal tract, either directly (via translocation) or by swallowing clearances from the respiratory tract.[21] However, the IARC monographs[1] and Institute of Medicine[22] did not find sufficient evidence that asbestos causes cancers of the gastrointestinal tract (pharyngeal, oesophageal, stomach and colorectal cancers). Some meta-analyses since then have reported small but positive associations between occupational exposures and increased risks of oesophageal cancer[23] (one of which did not find an association in subgroup analyses for amosite or crocidolite asbestos[24]), as well as stomach[25, 26] and colorectal cancers.[27, 28] However, a recent umbrella review considered that the evidence for associations between asbestos exposure and these cancers remains weak.[10]

We observed SIRs slightly above 1 for colorectal cancer among men and women who ever lived in ARPs. These estimates (1.24 and 1.46 respectively) appear larger than one might expect based on estimates from occupational exposure studies (e.g. overall SIR/Standard Mortality Rate of 1.07–1.16 for colorectal cancer estimated in two recent meta-analyses of occupationally exposed workers[27, 28]). Study participants' exposure to asbestos in ARPs, on average, is probably lower than that of occupational exposure levels. Currently, the carcinogenic mechanism of asbestos in the colorectum has not been confirmed in animal studies.[1]

More recently, a review has suggested a putative link between asbestos exposure and intrahepatic cholangiocarcinoma (intrahepatic bile duct cancer). This was supported by two case-control studies of occupational asbestos exposure,[29, 30] the detection of asbestos fibres in the liver of those living in a highly asbestos-polluted town,[31] and the possibility that asbestos can reach the liver and biliary tract through gastrointestinal absorption.[32] There were not enough cases to report results for liver cancer, which includes intrahepatic choloangiocarcinoma.

For the other cancers (pharynx, oesophagus and stomach), there were either not enough cases (in women) to report results, or we did not observe higher-than-expected rates. However, the study lacked statistical power to detect smaller associations should these exist.

Control outcomes

Control outcomes are outcomes not thought to be associated with asbestos exposure. Recent literature does not support associations between asbestos exposure and prostate, kidney and bladder cancers or melanoma.[10, 33, 34] Therefore, in the absence of confounding or chance, we do

not expect to see any differences in the rates of control outcomes in relation to asbestos exposure. However, our study estimated a marginally elevated rate of prostate cancer in men who ever lived in ARPs.

Observing an association between our exposure measurement (living in an ARP) and prostate cancer, a control outcome, gives us cause to question whether the exposed and unexposed populations in our study were dissimilar in ways that could have led to significant biases in study estimates. For example, it is possible that the exposed and unexposed group were dissimilar, on average, in socioeconomic circumstances, which might explain the higher rates of prostate cancer in men who ever lived in ARPs. Socioeconomic circumstances are associated with a range of cancers including prostate cancer, but perhaps least so for mesothelioma due to its specific relationship with asbestos and presumably fewer social determinants. The higher rate of prostate cancer may also have been related to enhanced health-seeking in residents of ARPs as they became aware of potential asbestos exposure.

Finally, we cannot rule out the role of chance in explaining study findings for all outcomes, including control outcomes. When estimating the SIR for each outcome, there was a small probability (set at 5%) that the statistic will imply that there was an association when one does not in fact exist. The probability of this increases with the number of outcomes tested in the study.

Strengths and weaknesses

A strength of this study was the use of the MCD, which is the only known data repository that collects address history for most Australians. Thus, we expected to have captured the vast majority of those who ever lived in the ACT since 1983. The potential omissions from the study population included those who were not eligible for Medicare (for example, residents on certain temporary visas or international students) or those who did not update their address after moving to the ACT. We do not expect either population to have been more or less likely to have lived in an ARP.

While the study is likely to have captured most of the historical ACT population, there was some potential for misclassification of exposure. First, errors in address details recorded in the MCD may have precluded their matching to the list of ARPs. These addresses would not have been recognised as ARPs and individuals associated with such addresses would be misclassified as unexposed. We know that three addresses in the list of ARPs did not link to any address in the MCD, but we do not know the extent to which other ARP addresses may have linked at least once but not to all possible permutations of the same address in the MCD. Second, some individuals who lived in an ARP may only have supplied a post office box address during that time, and thus be misclassified as unexposed. Third, the inception of the MCD in 1983 meant that we began observing the study population up to 15 years after the time of first exposure (which is possible from 1968). Consequently, individuals who had lived in an ARP from 1968

onwards but moved to a non-ARP address by 1983 would have been misclassified as unexposed. All such misclassifications would have led to an underestimation of SIRs, if an effect exists.

A further limitation is inaccuracies in the date of first exposure. Over time, electronic reimbursements for Medicare-subsidised services have reduced the incentive for individuals to update their addresses. We expect delays between actual changes of addresses and recorded changes in the MCD, especially post-2007. Among those who moved into an ARP, this delay period would be misclassified as unexposed. This misclassification would not have applied to individuals who never lived in an ARP as all person-time was classified as unexposed regardless of whether there was delay. We also expect such misclassification to have led to an underestimation of SIRs, if an effect exists.

We were not able to assess exposure levels (dose response) because we could not reliably quantify duration of exposure/residency at an address. First, it was not possible to quantify duration of residency for those who enrolled at the inception of Medicare (circa 1983) as the start dates of addresses at this time would not have reflected the time of moving into the address. Second, expected delays between actual changes of address and recording of changes meant that we were unable to reliably use start and end dates of addresses in the MCD to estimate duration of living at an address.

While our sample size was large (most of the historical ACT population since 1983), we did not have much information on participants' characteristics apart from sex and age at diagnosis. This meant that we were unable to account for other risk factors for the cancers examined. Importantly, we could not account for other exposures to asbestos. Specifically, we do not know if residents of ARPs were more or less likely to have been occupationally exposed to asbestos than the rest of the ACT population. For other cancers apart from mesothelioma, we could not account for smoking, alcohol consumption, socioeconomic status, obesity or occupational exposure to other irritants (for lung and laryngeal cancers). We have no prior expectations that these risk factors would be more or less likely in residents of ARPs.

We only had data to 2019 but projected around 3.3 excess cases of mesothelioma over 2020–2024 among men who ever lived in an ARP. This estimate should be considered in relation to its uncertainty (reflected in the width of its confidence interval, 1.3–8.2 excess cases) and the strong assumption made that men who had lived in an ARP had the same background risk of mesothelioma (barring ARP exposure) over time as the rest of the male ACT population. While this is conceivable, there were not sufficient observed cases in the exposed group over time to verify this assumption. Further, the estimated number of projected excess cases should also consider any potential biases that we could not measure in the examined relationship between having lived in an ARP and mesothelioma.

Finally, this study did not have adequate statistical power to detect SIRs below approximately 2.5–4 for many of the rarer cancers in the study, including cancers of the pharynx, oesophagus,

stomach, liver and larynx (with baseline incidence of 2–6 cases per 100,000 person years). This meant that the estimated SIRs will not indicate an association if the magnitude of association was below these limits.

Conclusion

This cohort study adds to the scientific literature on non-occupational exposure to asbestos in relation to a wide range of cancers across all age groups. Although non-occupational exposures are generally at much lower levels than occupational exposure, they are not negligible, are often continuous in nature and include children and women rather than mainly male workers. Our study suggests that living in a house with loose fill asbestos insulation may be sufficient to cause cancer, in particular mesothelioma. We could not confidently rule out that the higher rates of lung cancer or colorectal cancer observed in our study among those living in these houses were explained by other factors.

Glossary

Affected residential property (ARP): An ARP is a property in the ACT that was installed with loose fill asbestos insulation by D. Jansen & Co Pty Ltd between 1968 and 1979.

Confidence interval (CI): Expresses the degree of statistical uncertainty in a result. The 95% confidence interval can be interpreted to mean that if the study were to be repeated numerous times, 95% of the calculated confidence intervals would contain the true value.

Exposed and unexposed: In epidemiology, the term 'exposure' can be broadly applied to any factor that may be associated with an outcome of interest. Participants are exposed if they have experienced the exposure (in this study, having lived at an ARP), and unexposed if they have not.

Incidence rate: The number of new cases of disease per person-years of follow-up. A **crude rate** is the incidence rate before adjustment for any other factors, such as age.

Person-time: The time individuals are observed in a study. Participants contribute person-time so long as they do not yet have the health outcome under study (in this study, cancer) and have not died, hence are still at risk of developing the outcome. Person-time can be expressed in any unit of time, for example, person-years or person-days. It is used as the denominator/divisor when calculating the incidence rate.

Standardised incidence ratio (SIR): The ratio of the observed number of cases in the exposed population to the number that would be expected if the exposed population had the same disease incidence rates as the unexposed population. SIRs can be standardised (adjusted) for age, sex and other factors.

Funding and governance

The ACT Asbestos Health Study II was funded by the ACT Government. The progress of the study was overseen by the ACT Asbestos Health Study II Steering Committee. The Executive Branch Manager, Research, Programs and Services, from ACT Health chaired the Steering Committee, which was comprised of staff from ACT Health, the Loose Fill Asbestos Coordination Team, a community representative, an external academic expert on cancer epidemiology and staff from the ACT Asbestos Health Study II team.

Role of the funding agency

The ACT Asbestos Health Study II team was solely responsible for developing the methodology, collecting and analysing data, and interpreting the findings of this study. The ACT Asbestos Health Study II Steering Committee provided comments on the methodology and the final report. Neither the ACT Government nor the Steering Committee made decisions about what data to include, analyses to carry out or interpretation of the results, nor the decision to submit the final report.

Appendices

Appendix 1: Methodological differences between the previous and current study

Identification of ARPs

In both the previous and current studies, addresses in the MCD were identified as ARPs if they were found to match any address in the list of ARPs. Otherwise, they were classified as non-ARPs by default. The main difference between the previous and current study was the method of matching (linking) addresses; the current study employed geocoding software that had not been previously available. Additionally, six new ARP addresses were added to the list of ARPs in the current study that were not known in the previous study.

Additional data

The current study (to 31 December 2019) added six years of data to the previous study, as well as the addition of 358 individuals who started living in an ARP after 31 December 2013. The additional contributions of person-time resulted in a larger sample size, which is expected to increase the precision of SIR estimates (narrower confidence intervals) and increase the study's power (sensitivity to detect an effect).

Appendix 2: Data integration

The AIHW Data Integration Services Centre — a Commonwealth-accredited data integration authority — facilitated access to and performed linkages between the MCD to:

- the list of ARPs
- the ACD
- the NDI

Separation principle

The separation principle was in place throughout all data linkages performed in this study. The separation principle ensures that no one working with the data is able to view both the linking (identifying) information and the merged analysis (content) data in an integrated dataset.

In particular, AIHW staff undertaking linkage have access only to identifying variables (such as names and dates of birth), staff undertaking data merging have access only to content variables (such as diagnosis of cancer) and data analysts have access only to non-identifiable and appropriately confidentialised integrated datasets.

Linkage method

Data linkage was undertaken using the AIHW in-house methods based on probabilistic linkage. Probabilistic linkage is the linkage of records in two files based on the probabilities of agreement and disagreement between a range of linkage variables (fields used for comparison).

Appendix 3: Crude rates

Table A3–1: Number of cases, person-years at risk and crude rates for males and females, 1983–2019

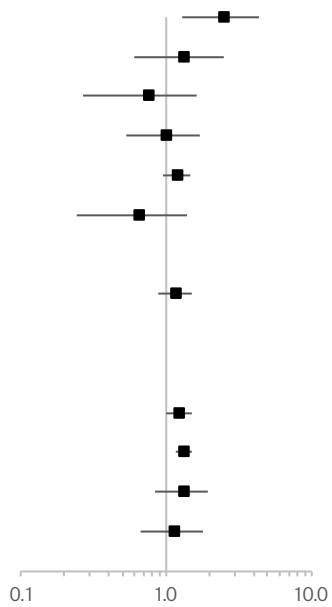
	Males						Females					
	Exposed			Unexposed			Exposed			Unexposed		
	No.	PY (x10,000)	Crude rate (95%CI)	No.	PY (x10,000)	Crude rate (95%CI)	No.	PY (x10,000)	Crude rate (95%CI)	No.	PY (x10,000)	Crude rate (95%CI)
Mesothelioma	12	12.90	0.93 (0.48,1.58)	344	1426	0.24 (0.22,0.27)	<6	np	np	62	1499	0.04 (0.03,0.05)
Other cancers												
Pharyngeal	9	12.89	0.70 (0.32,1.27)	455	1426	0.32 (0.29,0.35)	<6	np	np	118	1499	0.08 (0.07,0.09)
Oesophageal	6	12.89	0.47 (0.17,0.96)	565	1426	0.40 (0.36,0.43)	<6	np	np	258	1499	0.17 (0.15,0.19)
Stomach	12	12.89	0.93 (0.48,1.58)	943	1426	0.66 (0.62,0.70)	8	13.73	0.58 (0.25,1.10)	525	1499	0.35 (0.32,0.38)
Colorectal	82	12.84	6.39 (5.08,7.89)	5278	1422	3.71 (3.61,3.81)	76	13.68	5.56 (4.38,6.91)	4471	1495	2.99 (2.90,3.08)
Liver	<6	np	np	620	1426	0.43 (0.40,0.47)	<6	np	np	222	1499	0.15 (0.13,0.17)
Laryngeal	<6	np	np	328	1426	0.23 (0.21,0.26)	<6	np	np	41	1499	0.03 (0.02,0.04)
Lung	53	12.89	4.11 (3.08,5.34)	3400	1425	2.39 (2.31,2.47)	42	13.73	3.06 (2.21,4.09)	2443	1498	1.63 (1.57,1.70)
Ovarian	NA	NA	NA	NA	NA	NA	13	13.72	0.95 (0.50,1.57)	1058	1498	0.71 (0.66,0.75)
Control outcomes												
Melanoma	84	12.80	6.56 (5.23,8.08)	5484	1421	3.86 (3.76,3.96)	63	13.64	4.62 (3.55,5.87)	4459	1493	2.99 (2.90,3.07)
Prostate	225	12.70	17.72 (15.48,20.15)	12382	1417	8.74 (8.59,8.90)	NA	NA	NA	NA	NA	NA
Kidney	23	12.88	1.79 (1.13,2.63)	1306	1426	0.92 (0.87,0.97)	10	13.73	0.73 (0.35,1.29)	673	1498	0.45 (0.42,0.48)
Bladder	15	12.89	1.16 (0.65,1.87)	1152	1426	0.81 (0.76,0.86)	<6	np	np	341	1499	0.23 (0.20,0.25)

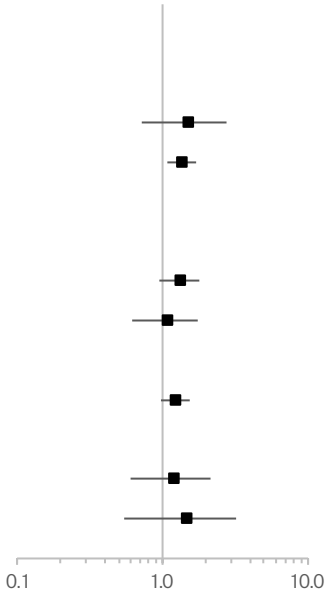
np=not available
NA=not applicable

Appendix 4: Sensitivity analyses

Figure A4-1: Observed (O) and expected (E) case numbers in the exposed population and standardised incidence ratios (SIR), 5-year lag

	Males		Females	
Type of cancer	**O/E**	**SIR (95% CI)**	**O/E**	**SIR (95% CI)**
Mesothelioma	12/4.84	2.48 (1.28,4.33)	np	
Pharyngeal	9/6.88	1.31 (0.60,2.48)	np	
Oesophageal	6/8.01	0.75 (0.27,1.63)	np	
Stomach	13/12.95	1.00 (0.53,1.72)	10/6.59	1.52 (0.73,2.79)
Colorectal	88/73.87	1.19 (0.96,1.47)	80/58.47	1.37 (1.08,1.70)
Liver	6/9.28	0.65 (0.24,1.41)	np	
Laryngeal	np		np	
Lung	54/46.25	1.17 (0.88,1.52)	44/33.2	1.33 (0.96,1.78)
Ovarian			16/14.64	1.09 (0.62,1.77)
Control outcomes				
Melanoma	84/68.07	1.23 (0.98,1.53)	76/61.93	1.23 (0.97,1.54)
Prostate	225/165.1	1.36 (1.19,1.55)		
Kidney	23/17.12	1.34 (0.85,2.02)	11/9.18	1.20 (0.60,2.14)
Bladder	15/13.52	1.11 (0.62,1.83)	6/4.05	1.48 (0.54,3.23)





np=not provided

Figure A4-2: Observed (O) and expected (E) case numbers in the exposed population and standardised incidence ratios (SIR), 15-year lag

	Males		Females	
Type of cancer	O/E	SIR (95% CI)	O/E	SIR (95% CI)
Mesothelioma	10/3.88	2.58 (1.23,4.74)	np	
Pharyngeal	8/5.17	1.55 (0.67,3.05)	np	
Oesophageal	6/6.36	0.94 (0.35,2.05)	np	
Stomach	9/10.04	0.90 (0.41,1.70)	7/4.96	1.41 (0.57,2.91)
Colorectal	75/56.9	1.32 (1.04,1.65)	62/44.78	1.38 (1.06,1.77)
Liver	np		np	
Laryngeal	np		np	
Lung	45/36.57	1.23 (0.90,1.65)	41/26.62	1.54 (1.11,2.09)
Ovarian			11/10.86	1.01 (0.51,1.81)
Control outcomes				
Melanoma	73/58	1.26 (0.99,1.58)	49/44.66	1.10 (0.81,1.45)
Prostate	199/144.9	1.37 (1.19,1.58)		
Kidney	21/14.8	1.42 (0.88,2.17)	9/7.08	1.27 (0.58,2.41)
Bladder	13/11.84	1.10 (0.58,1.88)	np	

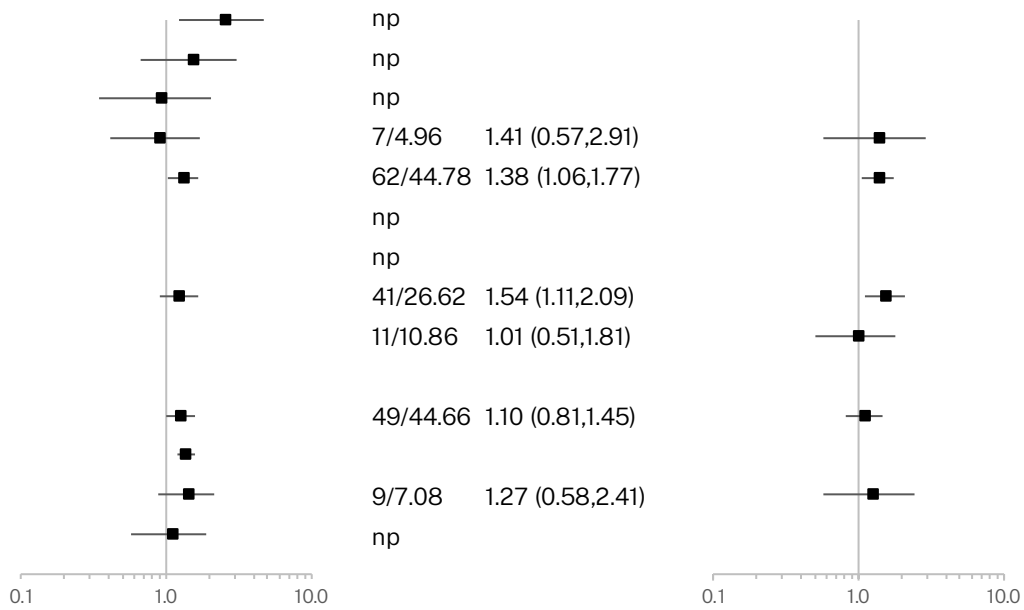


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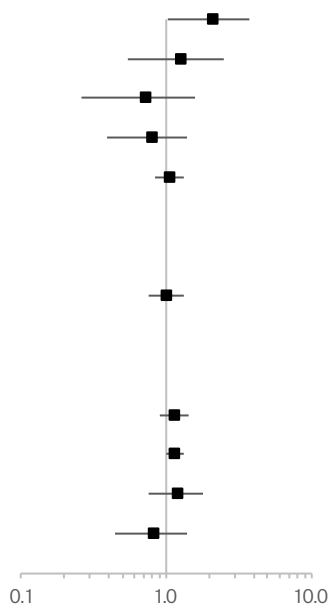
Figure A4-3: Observed (O) and expected (E) case numbers in the exposed population and standardised incidence ratios (SIR), excluding those who ever had a PO box address

	Males		Females	
Type of cancer	O/E	SIR (95% CI)	O/E	SIR (95% CI)
Mesothelioma	11/5.32	2.07 (1.03,3.70)	np	
Pharyngeal	8/6.3	1.27 (0.55,2.50)	np	
Oesophageal	6/8.33	0.72 (0.26,1.57)	np	
Stomach	11/13.97	0.79 (0.39,1.41)	6/6.72	0.89 (0.33,1.94)
Colorectal	79/75.12	1.05 (0.83,1.31)	74/59.78	1.24 (0.97,1.55)
Liver	np		np	
Laryngeal	np		np	
Lung	50/49.72	1.01 (0.75,1.33)	39/35.74	1.09 (0.78,1.49)
Ovarian			13/14.68	0.89 (0.47,1.51)
Control outcomes				
Melanoma	79/69.3	1.14 (0.90,1.42)	55/54.26	1.01 (0.76,1.32)
Prostate	205/178.8	1.15 (1.00,1.31)		
Kidney	22/18.48	1.19 (0.75,1.80)	10/9.28	1.08 (0.52,1.98)
Bladder	13/15.79	0.82 (0.44,1.41)	np	



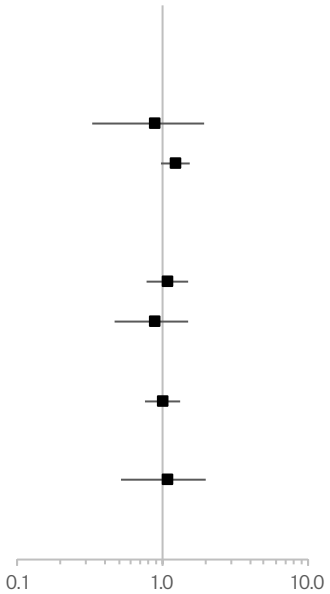


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Figure A4-4: Observed (O) and expected (E) case numbers in the exposed population and standardised incidence ratios (SIR), 10-year lag also applied to those who entered the study in 1983—84

	Males		Females	
Type of cancer	**O/E**	**SIR (95% CI)**	**O/E**	**SIR (95% CI)**
Mesothelioma	12/4.2	2.86 (1.48,4.99)	np	
Pharyngeal	9/5.71	1.58 (0.72,2.99)	np	
Oesophageal	6/6.86	0.87 (0.32,1.90)	np	
Stomach	12/10.62	1.13 (0.58,1.97)	8/5.36	1.49 (0.64,2.94)
Colorectal	77/61.02	1.26 (1.00,1.58)	75/48.67	1.54 (1.21,1.93)
Liver	np		np	
Laryngeal	np		np	
Lung	51/38.43	1.33 (0.99,1.74)	40/28.65	1.40 (1.00,1.90)
Ovarian			12/11.89	1.01 (0.52,1.76)
Control outcomes				
Melanoma	81/63.41	1.28 (1.01,1.59)	58/49.98	1.16 (0.88,1.50)
Prostate	220/157.5	1.40 (1.22,1.59)		
Kidney	22/16.27	1.35 (0.85,2.05)	10/7.88	1.27 (0.61,2.33)
Bladder	15/12.39	1.21 (0.68,2.00)	np	

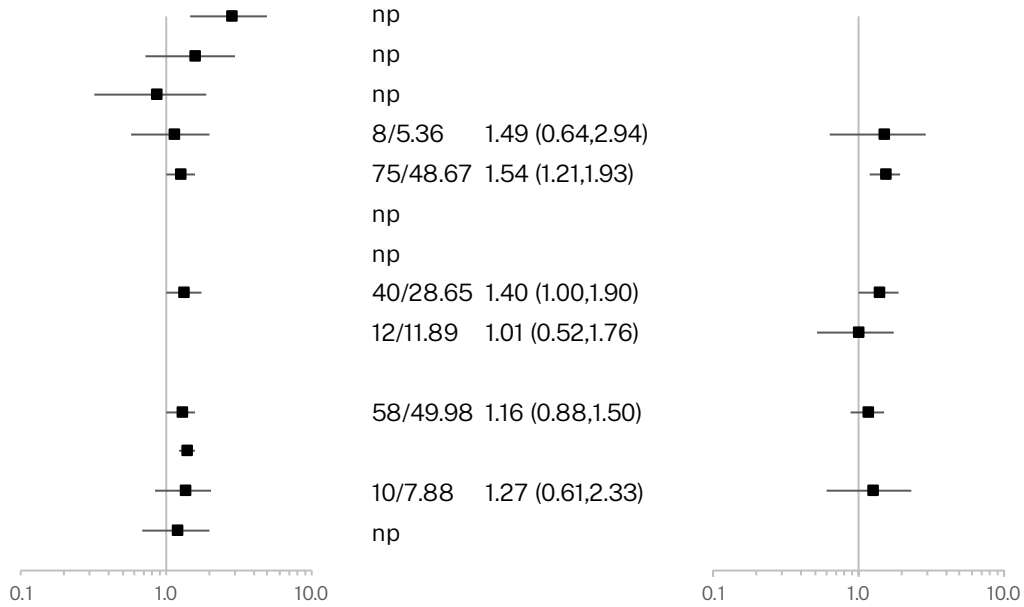

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Figure A4-5: Observed (O) and expected (E) case numbers in the exposed population and standardised incidence ratios (SIR), with censoring at 85 years

	Males		Females	
Type of cancer	**O/E**	**SIR (95% CI)**	**O/E**	**SIR (95% CI)**
Mesothelioma	10/4.13	2.42 (1.16,4.45)	np	
Pharyngeal	8/6.03	1.33 (0.57,2.62)	np	
Oesophageal	6/6.96	0.86 (0.32,1.88)	np	
Stomach	11/11.14	0.99 (0.49,1.77)	8/5.45	1.47 (0.63,2.89)
Colorectal	82/63.29	1.30 (1.03,1.61)	69/49.03	1.41 (1.09,1.78)
Liver	np		np	
Laryngeal	np		np	
Lung	50/39.63	1.26 (0.94,1.66)	39/28.96	1.35 (0.96,1.84)
Ovarian			13/12.46	1.04 (0.56,1.78)
Control outcomes				
Melanoma	83/65.92	1.26 (1.00,1.56)	61/52.42	1.16 (0.89,1.49)
Prostate	221/161.3	1.37 (1.20,1.56)		
Kidney	22/16.66	1.32 (0.83,2.00)	8/7.91	1.01 (0.44,1.99)
Bladder	13/12.4	1.05 (0.56,1.79)	np	

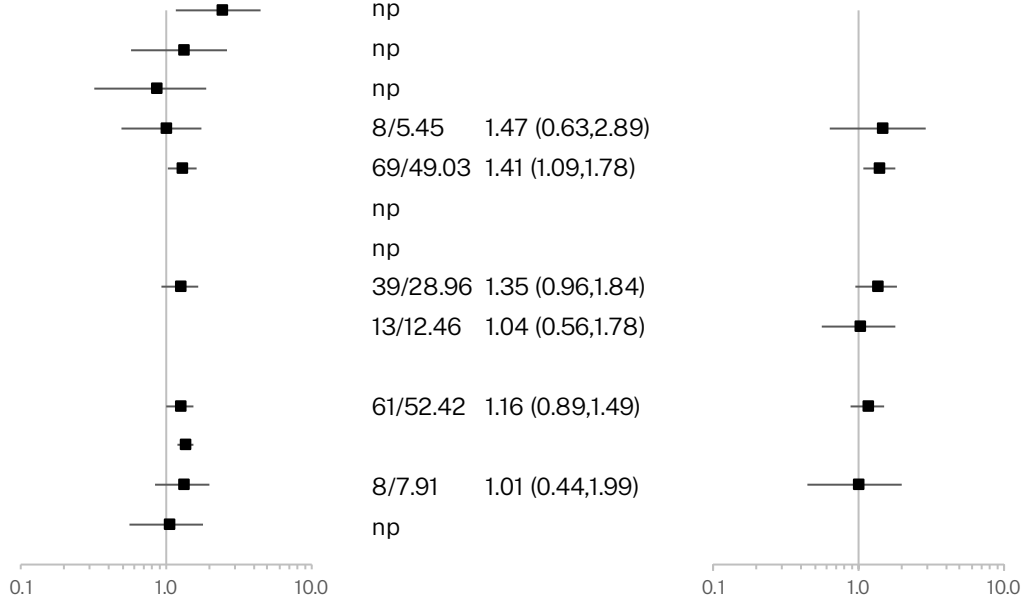

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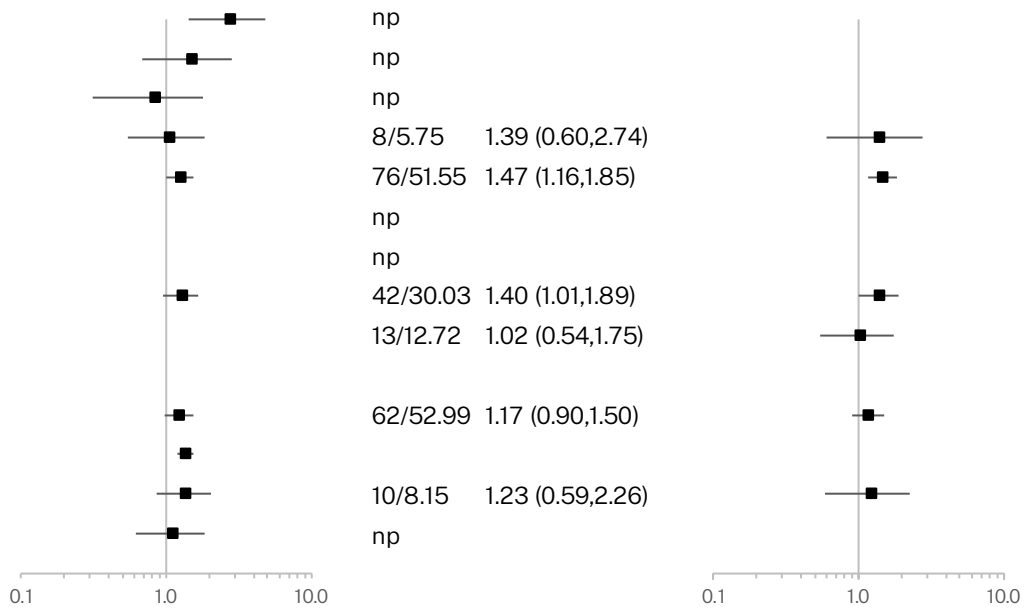
Figure A4-6: Observed (O) and expected (E) case numbers in the exposed population and standardised incidence ratios (SIR), with breaking of address ties by selecting the non-ARP address

	Males		Females	
Type of cancer	**O/E**	**SIR (95% CI)**	**O/E**	**SIR (95% CI)**
Mesothelioma	12/4.38	2.74 (1.42,4.79)	np	
Pharyngeal	9/6.03	1.49 (0.68,2.83)	np	
Oesophageal	6/7.21	0.83 (0.31,1.81)	np	
Stomach	12/11.48	1.05 (0.54,1.83)	8/5.75	1.39 (0.60,2.74)
Colorectal	82/65.47	1.25 (1.00,1.55)	76/51.55	1.47 (1.16,1.85)
Liver	np		np	
Laryngeal	np		np	
Lung	53/41.43	1.28 (0.96,1.67)	42/30.03	1.40 (1.01,1.89)
Ovarian			13/12.72	1.02 (0.54,1.75)
Control outcomes				
Melanoma	83/67.45	1.23 (0.98,1.53)	62/52.99	1.17 (0.90,1.50)
Prostate	224/164	1.37 (1.19,1.56)		
Kidney	23/16.99	1.35 (0.86,2.03)	10/8.15	1.23 (0.59,2.26)
Bladder	15/13.45	1.12 (0.62,1.84)	np	



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