THE UNIVERSITY OF SYDNEY

Cancer Elimination Collaboration

Emerging risk factors and interventions in early-onset cancers: Final report

Executive Summary

Cancer Elimination Collaboration, Sydney School of Public Health

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Plain language summary

This report summarises a detailed research project that looked at **early-onset cancer in Australia**, which means cancers diagnosed between the ages of **20 and 49 years**. The report brings together key information on how often these cancers occur, what may increase the risk, how cancers are prevented and detected, and how cancer affects people's lives at younger ages.

The project focused on cancers that have shown the **largest increases in incidence rates (number of cases diagnosed each year per 100,000 people) over time** and are **relatively common**. Using this approach, the project identified **12 priority cancers**, called "Tier 1" cancers. These comprised three female cancers (breast, cervical and uterine cancer), two male cancers (prostate and testicular cancer), and seven cancers affecting both sexes (bowel cancer, Hodgkin lymphoma, kidney cancer, leukaemia, neuroendocrine, pancreatic and thyroid cancer).

Australian data showed that **early-onset cancer rates increased between 2000 and 2021** for both women and men. The increases each year were generally small, but even small changes can add up over time. Early-onset cancers are not new, but the evidence shows that rates are continuing to rise for several cancer types. Cervical cancer is a specific case; there was an observed increase in incidence rates overall, likely due to a long-term decline in screening participation by younger women before changes to the screening program in 2017. Since that time, the evidence suggests that HPV screening is reducing rates of cervical cancer in younger females, and HPV vaccination is doing so in females under 25 years of age.

Cancer incidence trends were sometimes different between **older and younger populations**. Some cancers that increased in younger populations also increased in older populations (e.g. kidney cancer in both sexes, pancreatic cancer in both sexes), while some remained stable in older populations (e.g. female breast cancer, female Hodgkin lymphoma) and some decreased (e.g. colorectal cancer in both sexes, leukaemia for males).

Even though more young people were being diagnosed, cancer mortality, the death rate, in those aged 20–49 either stayed the same or went down for all the main (Tier 1) cancers. For people aged 50–79, death rates also stayed the same or went down for all the main cancers, except for uterine cancer and pancreatic cancer in women, where rates of death increased.

Trends were different depending on the **type of cancer, age group and sex**. To help understand these differences, the project reviewed evidence on factors that may increase cancer risk in younger adults.

Some **risk factors**, such as drinking alcohol, smoking, and being overweight and obese, are well known, while others are still being studied. International cancer agencies such as the World Cancer Research Fund (WCRF) and the International Agency for Research on Cancer (IARC) regularly review evidence on risk factors for cancer at all ages. While this evidence is not specific to early-onset cancer, it is important because it can help us understand what might be driving changes in early-onset cancers in Australia.

Adding to this information, the **scoping review looked at international evidence for risk factors for early-onset cancers.** Some risk factors identified by WCRF and IARC are also linked to cancers that occur at a younger age. These risk factors include drinking alcohol, smoking, and being overweight and obese.

This review also identified **other possible risk factors for early-onset cancers that are still being studied.** These include metabolic factors (such as diabetes and metabolic syndrome) as well as biological, hormonal and genetic factors. Overall, this body of evidence indicates that **health-related behaviours** such as smoking and drinking alcohol are still important, and that other factors inside the body, such as **genetics and physiological factors**, may also play a role in the risk of early-onset cancer risk.

After identifying these risk factors, the project looked at how common they are among younger people in Australia, and how they have changed over time. **Some indicative patterns were found**. For example, alcohol use is a risk factor for several early-onset cancers that are increasing. However, alcohol consumption among younger Australians has gone down over time. The consistency of these patterns – even considering the likely lag time between alcohol exposure and development of cancer – suggests that alcohol use is potentially less likely to explain the observed increases in alcohol-related early-onset cancers.

Further work is required to assess causality, to look in more detail at risk factor patterns and trends among younger Australians, and to consider how different risk factors might interact with each other.

The project also reviewed **cancer prevention guidelines and strategies**. The results suggest that some of these may need to be reviewed to better suit people aged 20–49 years.

The study found gaps in **psychological and social support guidelines** for younger adults with cancer. Many guidelines focus on teenagers or older adults, but younger adults often have unique needs related to work, family life and long-term health.

Overall, the project highlights important gaps in knowledge and services. It calls for **better data, more research and clearer guidelines** to improve prevention, care and support for Australians with early-onset cancer.

Overview

This scoping review generated an important overview of early-onset cancer in Australia (defined as cancers diagnosed in the age range of 20-49 years), assembling evidence on national cancer incidence and mortality, risk factors and aetiology, prevention and detection, and psychosocial impacts and services.

The analysis focused on cancers prioritised due to having the largest absolute increases in incidence over time and being sufficiently common, for either or both sexes. The 12 'Tier 1' cancers for analysis comprised three female-specific cancers (breast, cervical and uterine cancer), two male-specific cancers (prostate and testicular cancer), and seven non-sex-specific cancers (colorectal cancer, Hodgkin lymphoma, kidney cancer, leukaemia, neuroendocrine neoplasms, pancreatic cancer and thyroid cancer).

Epidemiological analysis of national Australian data confirmed overall increases in sex-specific early-onset cancer incidence from 2000-2021, for Tier 1 cancers and all cancers combined. The average annual proportional increases were modest, but even modest annual increases will accumulate to substantial increases over time if current trends continue.

For example, for all Tier 1 early-onset cancers combined, over the period 2000-2021, the average annual percentage change in incidence rate for females was 1.1% per year (95% confidence interval 1.0% - 1.2%), and for males this figure was 1.6% per year (1.4% to 1.8%).

For all early-onset cancers (including but not limited to Tier 1 cancers), over the period 2000-2021 the average annual percentage change in incidence rate for females was 0.6% per year (95% confidence interval 0.5% - 0.7%), and for males this figure was 0.3% per year (0.1% to 0.4%).

Cervical cancer is a specific case; there was an observed increase in incidence rates overall, likely due to a long-term decline in screening participation by younger women before changes to the screening program in 2017. Since that time, the evidence suggests that HPV screening is reducing rates of cervical cancer in younger females, and HPV vaccination is doing so in females under 25 years of age.

Early-onset cancer incidence trends vary by cancer, age group and sex. From the scoping review of risk factors for early-onset cancers and a high-level comparison of prevalence trends for those risk factors with their associated cancer incidence trends, candidate risk factors warranting further analysis through targeted research were identified. This research would be supported by enhanced data collection and consideration of interaction between risk factors.

Comparison of cancer-specific mortality trends for combined Tier 1 cancers over 2000-2021 for the early-onset age range (20-49 years) versus older populations (50-79 years) showed a clear overall downwards trend in cancer mortality in younger (20-49 years) and older (50-79 years) populations for both sexes. When assessed by cancer type (for Tier 1 cancers), there was either a similar downward trend or no significant change, with the exception of female pancreatic and uterine cancers (50-79 years), where there was an increase in mortality rates.

Established modifiable risk factors for cancer - particularly alcohol, tobacco use and overweight and obesity - are consistently associated with an increased risk of early-onset cancer. Emerging potential biological and hormonal, metabolic, and genetic risk factors have also been identified, although these are not currently formally classified by international cancer agencies such as the World Cancer Research Fund (WCRF) and the International Agency for Research on Cancer (IARC). Several modifiable risk factors – either established risk factors or emerging as potential risk factors – have been shown to be significantly associated with increased cancer risk across multiple early-onset cancer types, alongside family history of the same cancer type.

After identifying these risk factors, the project assessed available risk factor prevalence trends among younger people in Australia, and identified some indicative patterns in relation to trends in cancer incidence. For example, alcohol use is a risk factor for several early-onset cancers that are increasing. However, alcohol consumption among younger Australians has gone down over time. The consistency of these patterns – even considering the likely lag time between alcohol exposure and development of cancer – suggests that alcohol use is potentially less likely to explain the observed increases in alcohol-related early-onset cancers. The insights from this analysis can be used to guide future research from cohort studies, where the association between exposures and outcomes can be reliably assessed from individual-level histories. This analysis also highlighted that population-level prevalence data was not readily available for many risk factors for early-onset cancer.

Prevention and detection initiatives for early-onset cancers include guidelines, prevention strategies and screening programs that are generally designed for the whole adult population or for older adults; evaluation and refinement of these initiatives for the population aged 20-49 is therefore warranted. Various screening programs extend to early-onset cancers (cervical, bowel and breast); there would be some value in undertaking a rigorous analysis of their contribution to incidence, including consideration of overdiagnosis[1] (and lead time bias[2]), through analysis of longitudinal datasets. The psychosocial impacts and needs of people with early-onset cancer, and the associated guidelines, identify a clear opportunity to develop Australian guidelines targeted to ages 20-49 years, or to 25-49 years (mid-adults) to address the specific impacts and needs of this age group. Existing guidelines for adolescents and all adults can likely be adapted for this purpose.

The evidence gaps and priority next steps identified in this project can help guide policy and research initiatives to address the recorded increase in incidence of early-onset cancers in Australia. Priority next steps include more comprehensive data analyses, assessment of the mortality burden of early-onset cancers (accounting for deaths occurring in older age groups), improved data on the prevalence of risk factors in the Australian population to inform rigorous analyses of the drivers of increased early-onset cancers, national evidence- and consensus-based guidelines for improved management of early-onset cancers, and psychosocial guidelines for early-onset cancers (beyond existing guidelines available for adolescents).

In cancer research and policy, there is a history of greater attention to *adolescents and young adults* (AYAs), all adults, or older populations. This is reflected in the Australian Cancer Plan, where AYAs and older people are priority populations. The age range of 20-49 years requires increased attention. In Australia this is particularly true for the mid-adult range of 25-49 years, as young adults up to 24 years (or sometimes 25 years) are considered to be in the AYA category. The upper age range for AYAs is often higher in studies from outside Australia (usually up to 39 years), so the 'mid-adult' gap in Australia is particularly marked.

Clearly defining a new mid-adult age group (25–49 years) within the population at risk of early-onset cancer would identify a group with distinct needs compared with AYAs and older populations, spanning many life stages with common changes in education, employment, family composition, fertility planning, caring responsibilities and co-morbidities. A comprehensive definition of early-onset cancer sub-populations in Australia would therefore include children (0–14 years), AYAs (usually 15–24 or 15–25 years) and mid-adults (25–49 years).

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[1] Diagnosis of cancer that would not have otherwise been diagnosed during a person's lifetime.
[2] Lead time is the duration of time by which a cancer diagnosis is advanced due to early detection (like routine screening) compared to when it would have been diagnosed naturally through symptoms. This can lead to lead time bias, where an individual appears to survival for longer following their cancer diagnosis, but this may be due only to earlier diagnosis with no change to the natural history of their disease.

Analysing narrower age groups within the early-onset age band, such as 10-year age groups, would be valuable. Trends in cancer incidence can differ by 10-year age group (within sex), such that additional cancers would have been classified as 'Tier 1' if the criteria been applied by age group. Understanding these age-group-specific patterns and their drivers is important, including cohort-specific exposure to modifiable risk factors and differences in engagement with prevention and early detection strategies.

There are important differences in both incidence and mortality by sex. Global and Australian incidence trends vary markedly by sex, and it followed that 'Tier 1' cancers were assessed based on sex-specific incidence. While this project's scoping reviews and landscape analyses of prevention, early detection and psychosocial support guidelines identified some sex-specific evidence and guidelines, further work is required to fully capture and address known differences by sex – and by gender – and how different populations can best be supported by cancer health services. This extends to priority populations based on remoteness of residence, First Nations status, cultural and linguistic background, disability, socioeconomic status, and sexuality, with consideration of intersectionality (i.e., that people can and often do belong to more than one identified priority population).

The strengths of this project include rigorous and transparent analysis of observed national data, with consideration of age groups and sex. Detailed scoping reviews were conducted on risk factors and psychosocial impacts, and while these were not systematic reviews, systematic approaches were applied where possible to identify, summarise and assess the quality of available evidence. Landscape analyses of prevention and early detection strategies and psychosocial impacts drew on a broad range of sources and used the findings to inform priority areas requiring attention to improve cancer control and reduce the burden of cancer for younger populations.

Of note, an overarching goal for this project was to examine the socio-economic and environmental factors contributing to the prevalence of early-onset cancers. This was addressed to some extent through the analysis of prevalence trends for risk factors, including risk factors that could be classified as socio-economic or environmental in nature, however a comprehensive analysis of such factors would require analysis of longitudinal datasets.

This project identified a clear need for momentum and increased investment in the evidence space on early-onset cancers in Australia. Priorities include **better data, more research and clearer guidelines** to improve prevention, care and support for Australians with early-onset cancer.

Background and methodology

Australian data indicate a high age-standardised incidence rate of early-onset cancers among people aged 20-49 years, relative to global trends, with a rising incidence of various cancers in adults.

In response to emerging evidence, media coverage and stakeholder concerns, this rapid-response (6-month) project aimed to explore and identify opportunities for policy and research initiatives to address the recorded increase in incidence of early-onset cancers in Australia.

The high-level aims of this project were to:

1) Identify the potential epidemiological reasons for a recorded rise in early-onset cancer, examine the socio-economic and environmental factors contributing to the prevalence of early-onset cancers, and provide an analysis of current intervention strategies;
2) Identify existing knowledge gaps, offering a foundation for targeted research and tailored interventions that could potentially alter the trajectory of early-onset cancer rates in Australia;

through addressing the following key questions:

1. What are the causes of the recorded increase in early-onset cancers in Australia?
2. What are the psychosocial impacts of early-onset cancer for people affected by cancer?
3. What evidence-based interventions have been implemented to reduce the incidence of early-onset cancers?
4. What are the research gaps in causation and potential interventions for early-onset cancers?

To address the key questions, a set of objectives were identified. The objectives, methods and associated technical chapters (unpublished) are outlined in Table 10 (page 37). Of note, within the time available, evidence reviews utilised scoping reviews rather than systematic reviews, and data collection was limited to publicly available data, peer-reviewed publications and grey literature.

To focus the analysis, a set of priority cancers for analysis was identified early in the project. The methods for identifying these priority cancers, and resulting list of cancers, are outlined below.

The project reflects the following themes: Priority cancers and cancer incidence and mortality trends; Risk factors; Prevention and detection; and Psychosocial aspects. Key findings from these themes, and associated evidence gaps, are summarised below.

Additionally, the project identified a set of priority next steps to help guide momentum and increased investment in and development of the evidence space on early-onset cancers in Australia. These are outlined below.

To assist with knowledge translation, the project also produced single page ‘Snapshots’ using infographics to help summarise key findings. These were generated for: each priority cancer (by sex), highlighting recent incidence and mortality trends and established and potential risk factors; incidence for early-onset cancers compared to cancer incidence in older populations; and cancer mortality among early-onset populations compared to mortality older populations

Artificial intelligence was selectively used to check and refine grammar (Microsoft Copilot, with any changes to text manually reviewed) and as a tool when searching for grey literature (Google Gemini 3 and Microsoft Copilot, complemented by non-AI guided internet searches).

A list of acronyms and a glossary of terms used in this Executive Summary is shown from page 32.

Priority cancers

The project sought to focus on a shortlist of cancers considered to be high priority in the consideration of early-onset cancers in Australia. Based on methodologies applied in global studies, cancer incidence (and mortality) should be assessed separately by sex in the first instance. It is also clear that ranking cancers by a single metric can be misleading; it is important to simultaneously consider both absolute incidence rates as well as relative or absolute changes in rates.

On this basis, cancers were shortlisted for analysis (denoted 'Tier 1' or 'Tier 2' cancers) if they ranked highly based on absolute increases in incidence over time and if they were sufficiently common by the end of the analysis period, for either or both sexes. Specifically, cancers were deemed to be Tier 1 cancers if they (i) ranked in the top 10 in terms of absolute increase in cancer incidence rate between the periods 2000-2004 to 2015-2019, and (ii) if the incidence rate in 2015-2019 was above a threshold of sufficiently common incidence (as detailed in Figure 5), where this was assessed separately by sex. These criteria were applied for the whole target age range (20-49 years), by sex, and at a national level. Non-sex-specific cancers that met these criteria for one sex only were included, with analysis reported for both sexes. Cancers were deemed to be Tier 2 cancers if they (i) ranked in the top 10 in terms of relative increases in cancer incidence between the periods 2000-2004 to 2015-2019 or (ii) if they met the first criteria for Tier 1 cancers (absolute increases), but not the second criteria (incidence rate above threshold).

A flow diagram of the process used to define Tier 1 and 2 cancers is shown in Figure 5 (Appendix, page 38). This led to 12 cancers being defined as 'Tier 1' cancers' (Figure 1).

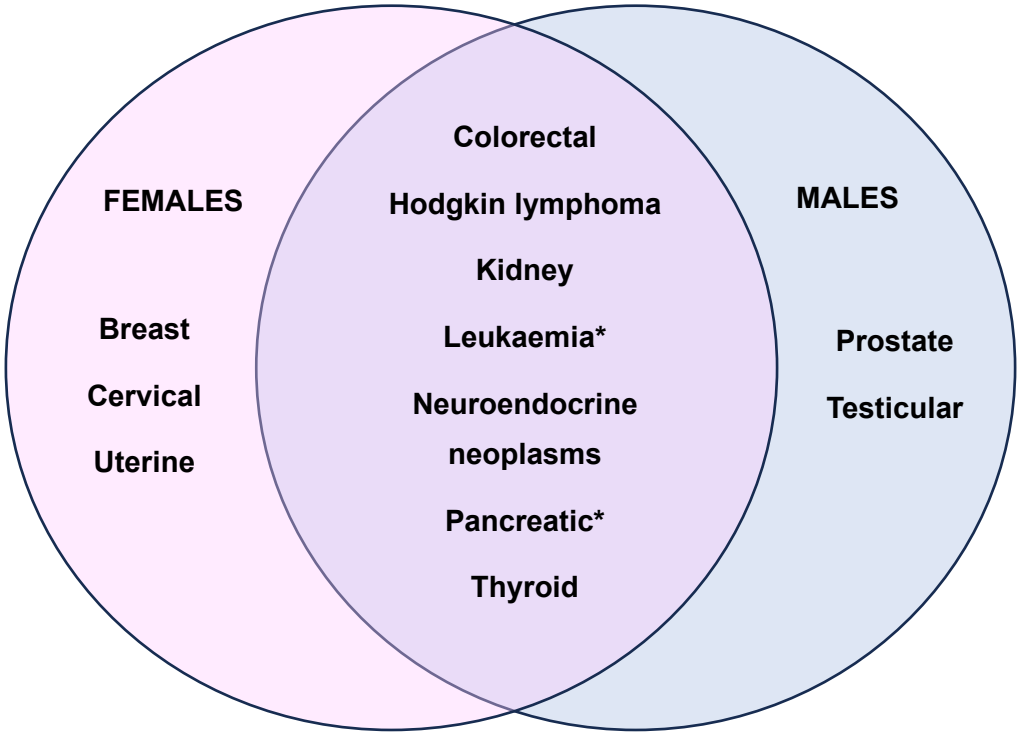


Figure 1. Identified high priority ('Tier 1') cancers, by sex. *Denotes cancers that met the Tier 1 criteria for one sex only (in both cases, males but not females).

As shown, Tier 1 cancers comprised:

- Three female-specific cancers (breast[3], cervical and uterine cancer);
- Two male-specific cancers (prostate and testicular cancer); and
- Seven non-sex-specific cancers (colorectal cancer, Hodgkin lymphoma, kidney cancer, leukaemia, neuroendocrine neoplasms, pancreatic cancer and thyroid cancer).

Two of the non-sex-specific cancers were included because they met the criteria for one sex only (leukaemia and pancreatic cancer, for which the criteria were met for males but not females).

Reflecting the project's primary focus on cancer incidence rather than overall cancer burden, the shortlisting criteria did not include mortality rates. Recommendation for a rigorous assessment of the mortality burden of early-onset cancers is included in the priority next steps (Table 9, from page 30), noting that this would need to account for the variation in time from diagnosis to death for different cancers.

Key findings

Based on detailed analyses of observed cancer incidence trends, and complemented by scoping reviews and landscape analyses of important contexts to these trends, the key findings of this project are summarised below.

1. Priority cancers and cancer incidence and mortality trends

- Global incidence trends for early-onset cancer vary markedly by cancer, setting and (when reported) sex. In literature comparing global incidence by country, Australia has historically reported one of the highest all-cancer incidence rates. For example, over the period 1998-2012 in the 15-39 year age group, the Australian age-standardised incidence rate was the second-highest among 41 countries (66.4 cases per 100,000, below only Ireland, which reported a rate of 66.5 cases per 100,000).[1]
- Global comparisons have reported increasing trends for early-onset cancer incidence rates in Australia. For example, over the period 2002-2012, significant increases in Australian incidence (based on average annual percentage changes (AAPCs)) were reported for breast cancer (females), colorectal cancer (males and females), endometrial cancer (females), kidney cancer (males and females), liver cancer (males and females), pancreatic cancer (females only), prostate cancer (males), and thyroid cancer (males and females).[2]
- Review of methodologies and findings in global studies highlighted that cancer incidence and mortality should be assessed separately by sex in the first instance, and that while Australian data are included in these global comparisons, additional analysis is required to assess age- and sex-specific trends for the Australian population. Focussing on the age range 20-49 years is reasonably consistent with publications of global data, and there is likely to be additional value in reporting separately for younger age groups where feasible (i.e., by either ten-year age group or separately for 20-39 and 40-49 years).
- Analysis of more recent data indicated that early-onset cancer incidence rates increased in Australia between 2000-2004 and 2015-2019, with females experiencing a greater absolute increase in rates compared to males for all cancers combined. In females, the cancers contributing most to the absolute increase in early-onset cancer rates were (in order from greatest contributor) thyroid, colorectal, neuroendocrine, breast, uterine and cervical cancers. In males, the cancers contributing most to the absolute increase in early-onset cancer rates were

[3] While breast cancer does occur in males, male breast cancer did not meet the inclusion criteria for Tier 1, so for the purposes of this project, breast cancer was considered a sex-specific (female) cancer.

prostate, colorectal, thyroid, kidney, neuroendocrine and testicular cancers. These data were used to identify Tier 1 and Tier 2 cancers for analysis.

- For most Tier 1 cancers, sex-specific incidence was higher for more recent birth cohorts.
- Subsequent joinpoint regression analyses of sex-specific cancer incidence, extended to more recent data (up to year 2021) confirmed statistically significant increases in incidence for all Tier 1 cancers for the age group 20-49 years, and for each 10-year age group (Figure 2), when assessed for the most recent two decades (2000-2021) or for a longer period (1982-2021). For example, for the period 2000-2021, for all Tier 1 cancers combined, the AAPC for females was 1.1% per year (95% confidence interval 1.0% - 1.2%), and the AAPC for males was 1.6% per year (1.4% to 1.8%). For all early-onset cancers, over the period 2000-2021 the AAPC in incidence for females was 0.6% per year (95% confidence interval 0.5% - 0.7%), and for males this figure was 0.3% per year (0.1% to 0.4%).

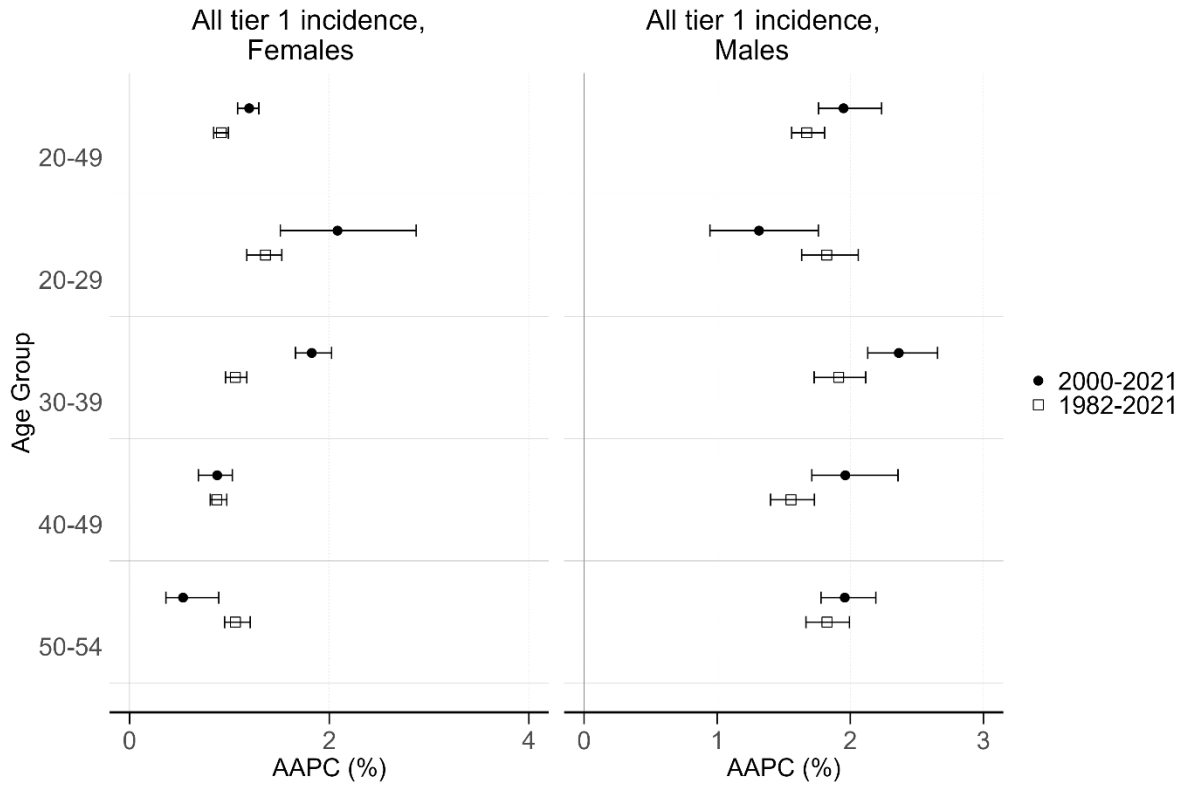


Figure 2. Average annual percentage change (AAPC) for Tier 1 cancer incidence in Australian females and males by age group and time period, joinpoint regression.

- The proportional contribution of cancers to aggregate cancer incidence varied over time and by age group and sex (Figure 3 and Figure 4). This was confirmed by joinpoint regression analyses by cancer, 10-year age group and sex, which showed that incidence trends could vary between age groups for some cancers (e.g., leukaemia (males) and cervical cancer), suggesting that additional insights would be gained through further age-group specific analysis.

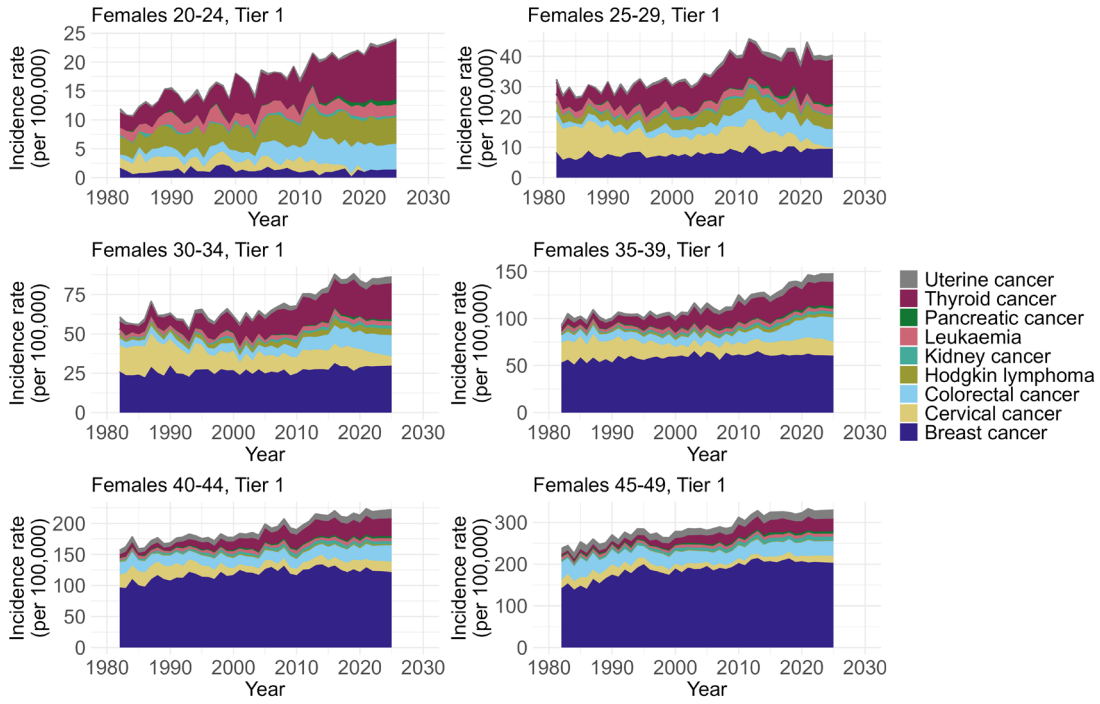


Figure 3. Aggregate cancer incidence for Tier 1 cancers by cancer type and year, females.

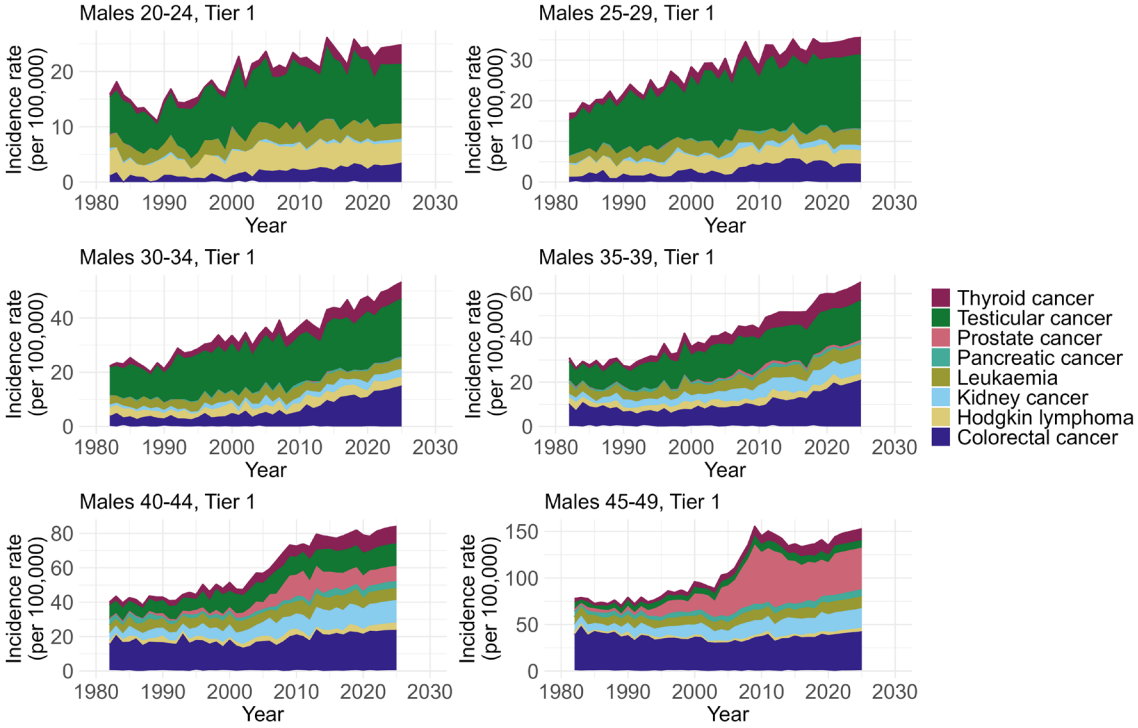


Figure 4. Aggregate cancer incidence for Tier 1 cancers by cancer type and year, males.

- Some Tier 1 cancers have started to decrease or remain stable in more recent years, including breast cancer (females aged 40-49 years only), cervical cancer (females aged 20-29 years), colorectal cancer (males and females aged 20-29 years), neuroendocrine neoplasms (females only), prostate cancer, and thyroid cancer (females aged 40-49 years).
- Comparison of sex-specific cancer incidence for ages 20-49 versus ages 50-79 years showed an overall significant increase (based on AAPCs) in overall cancer incidence in younger ages and in older ages among males, but no significant increase in older females. The same patterns were observed for all Tier 1 cancers combined. When assessed for each Tier 1 cancer, some cancers had increased in younger and older populations (e.g., kidney cancer in both sexes, pancreatic cancer in both sexes), some cancers had increased in younger populations with no change in older populations (e.g., female breast cancer, female Hodgkin lymphoma), and some cancers had increased in younger populations and decreased in older populations (e.g., cervical cancer, colorectal cancer in both sexes). These findings are summarised in Table 1.

Table 1. Trends for average annual percentage change (AAPC) in Australian cancer incidence rates between 2000 and 2021 for early-onset cancers (20-49 years) and older-onset cancers (50-79 years).

Cancer	Females		Males	
	20-49 years	50-79 years	20-49 years	50-79 years
All cancers combined	▲	—	▲	▲
All Tier 1 cancers	▲	—	▲	▲
Breast cancer	▲	—	*NA*	*NA*
Cervical cancer	▲	▼	*NA*	*NA*
Colorectal cancer	▲	▼	▲	▼
Hodgkin lymphoma	▲	—	▲	▲
Kidney cancer	▲	▲	▲	▲
Leukaemia	—	—	▲	▼
Neuroendocrine neoplasms	▲	▲	▲	▲
Pancreatic cancer	▲	▲	▲	▲
Prostate cancer	*NA*	*NA*	▲	▲
Testicular cancer	*NA*	*NA*	▲	▲
Thyroid cancer	▲	▲	▲	▲
Uterine cancer	▲	▲	*NA*	*NA*
Other cancers (non-tier 1)	▼	—	▼	—

Black arrows indicate a significant increase in AAPC, blue arrows indicate a significant decrease in AAPC, while grey bars indicate no statistically significant change (95% confidence interval included zero). NA: not applicable.

- Cervical cancer rates in women 20-49 increased from 2000 to 2021, likely due to a number of changes in screening, HPV exposure and vaccination. Before this period, cervical cancer incidence had reduced due to opportunistic screening from the 1980s, and organised screening from 1991.[3] In the early 2000s, this decline slowed, partly because the cytology-based screening program was reaching the limits of its impact in terms of regularly reaching females every two years, and partly due to the limited effectiveness of cytology screening for adenocarcinoma cervical cancers, which were on the rise (increasing from 11% of cervical cancers in 1982 to 26% in 2021[4]).[5] In 2017, the program changed to HPV screening, which likely caused a temporary increase in reported cervical cancer incidence rates due to earlier detection of prevalent cervical cancer in the first round of screening with the more sensitive HPV test.[6] Additionally, incidence may have increased due to increasing HPV exposure among younger Australian females. Looking ahead, HPV vaccination and screening are expected to drive cervical cancer elimination by 2035.[7] This is supported by falling cervical cancer incidence rates between 2013 and 2021 in women aged 20-29 years, and no cases being recorded in women

under 25 in 2021 for the first time since records commenced in 1982, attributed to HPV vaccination.[8]

- Comparison of cancer-specific mortality trends over 2000-2021 for the early-onset age range (20-49 years) versus older populations (50-79 years) showed a clear overall downwards trend in cancer mortality in younger (20-49 years) and older (50-79 years) populations in all groupings for both sexes. When assessed by cancer type (for Tier 1 cancers), there was either a similar downward trend or no significant change, with the exception of pancreatic and uterine cancers for older females (50-79 years), where there was an increase in mortality rates. These findings are summarised in Table 2.

Table 2. Trends for average annual percentage change (AAPC) in Australian cancer mortality rates between 2000 and 2021 for early-onset cancers (20-49 years) and older-onset cancers (50-79 years).

Cancer	Females		Males	
	20-49 years	50-79 years	20-49 years	50-79 years
All cancers combined	▼	▼	▼	▼
All Tier 1 cancers	▼	▼	▼	▼
Breast cancer	▼	▼	*NA*	*NA*
Cervical cancer	—	▼	*NA*	*NA*
Colorectal cancer	—	▼	—	▼
Hodgkin lymphoma	▼	▼	▼	▼
Kidney cancer	—	▼	▼	▼
Leukaemia	▼	▼	▼	▼
Neuroendocrine neoplasms	*	*	*	*
Pancreatic cancer	—	▲	—	—
Prostate cancer	*NA*	*NA*	▼	▼
Testicular cancer	*NA*	*NA*	—	—
Thyroid cancer	*	—	*	—
Uterine cancer	—	▲	*NA*	*NA*
Other cancers (non-tier 1)	▼	▼	▼	▼

Black arrows indicate a significant increase in AAPC, blue arrows indicated a significant decrease in AAPC, while grey bars indicate no statistically significant change (95% confidence interval included zero). *AAPC unable to be estimated for 2000-2021 due to either lack of historical mortality data (neuroendocrine neoplasms) or periods with zero deaths (thyroid cancer). NA: not applicable

2. Risk factors

Risk factors

- A scoping review of risk factors associated with early-onset cancer found that established modifiable risk factors - particularly alcohol, tobacco use and overweight and obesity - showed consistent statistically significant associations with early-onset cancers.
- The scoping review also identified 13 emerging potential risk factors, spanning biological and hormonal, metabolic, and genetic factors, which are not currently recognised by major agencies. Most could be grouped into three broad (non-exhaustive) domains:
    i. Clinical or medical history factors, including inflammatory bowel disease, diabetes, fasting insulin (as a separate metabolic measure), basal metabolic rate, hyperlipidaemia, metabolic syndrome, non-alcoholic fatty liver disease, and triglycerides;
    ii. Biological and hormonal factors, such as free testosterone, insulin-like growth factor-1, and a history of primary infertility; and
    iii. Genetic factors, including a family history of the same type of cancer and cancer-predisposing germline pathogenic or likely pathogenic variants.
- Specifically, several modifiable risk factors were associated with increased risk across multiple early-onset cancer types. These included both established and emerging potential risk factors. Established risk factors included alcohol use, tobacco use, high BMI, obesity, high waist circumference, and a high waist-to-hip ratio, while emerging potential risk factors included diabetes, metabolic syndrome, non-alcoholic fatty liver disease, and unhealthy dietary patterns. A family history of the same cancer type was also associated with increased risk for several early-onset cancers.
- These findings are summarised by cancer type in Table 3. Collectively, these results suggest that while traditional health-related behaviours remain important, a wider set of underlying physiological and genetic influences may also contribute to early-onset cancer risk.

Table 3. Emerging potential risk factors for early-onset cancers identified in the scoping review (for ages 20-49 years; studies published 2015-2025 for colorectal cancer and 2021-2025 for other cancers), according to level of evidence as assessed by IARC (for all ages; published 2025) or WCRF (for all ages; published 2018) and direction of evidence as identified from the scoping review.

Level of evidence according to IARC or WCRF:	Convincing, probable or limited evidence		NOT convincing, probable or limited evidence	
Direction of risk as assessed in the scoping review:	**Increased risk**	**Decreased risk**	**Increased risk**	**Decreased risk**
Alcohol, tobacco and other drug use				
Alcohol use	Breast, colorectal, stomach			
Tobacco use	Breast, colorectal, pancreas, lung, stomach			
Biological and hormonal mechanisms				
Biological mechanisms				
Free testosterone			Prostate*	
Insulin-like growth factor-1			Prostate*	
Hormonal and reproductive factors				
Age at menarche (older)				Uterus (endometrium)
Breastfeeding		Breast		
History of primary infertility			Breast	
Parity/pregnancy (≥2 pregnancies)				Breast
Sex hormone-binding globulin				Prostate*
Clinical and medical history				
Benign disease				
History of benign disease corresponding to the cancer site/type			Breast	
Chronic conditions				
Inflammatory bowel disease			Colorectal	
Glycaemic traits				
Diabetes (Type I/II)			Colorectal, pancreas	
Fasting insulin			Colorectal*	
Metabolic syndromes/disorders				
Basal metabolic rate			Colorectal*	
Hyperlipidaemia			Bladder, colorectal	
Metabolic syndrome/metabolic burden			Colorectal, thyroid	
Non-alcoholic fatty liver disease			Uterus (endometrium), thyroid, ovary	
Triglycerides			Colorectal	
Developmental and birth related				
Body size at age 10 years (high)	Uterus (endometrium)			
Height at age 10 years (high)	Uterus (endometrium)			
Diet and other health-related behaviours				
Body size and composition				
Body mass index (high)	Hodgkin lymphoma, colorectal, oesophagus, ovary,	Breast†		

Level of evidence according to IARC or WCRF:	Convincing, probable or limited evidence		NOT convincing, probable or limited evidence	
Direction of risk as assessed in the scoping review:	**Increased risk**	**Decreased risk**	**Increased risk**	**Decreased risk**
	stomach, vagina, vulva			
Body fat percentage (high)	Colorectal*			
Height (high)	Breast			
Hip circumference (high)		Breast		
History of overweight/obesity	Breast			
Obesity	Uterus (endometrium), stomach			
Waist circumference (high)	Breast, colorectal, uterus (endometrium)			
Waist to hip ratio (high)	Breast, colorectal*			
Weight gain	Liver			
Weight loss		Breast (pre-menopausal)†		
Dietary patterns and nutrient intake				
Unhealthy dietary factors‡			Colorectal, stomach	
Vitamin D supplements		Colorectal		
Physical activity				
Sedentary behaviours	Colorectal			
Vigorous physical activity		Breast, lung		
Environmental and occupational exposure				
Fluoranthene exposure			Breast	
Radon exposure	Melanoma			
Genetics and genomics				
A family history of the same type of cancer			Any haematological malignancies§, breast, colorectal, stomach	
Cancer-predisposing germline pathogenic/likely pathogenic variants			Breast, neuroendocrine neoplasm (Merkel cell carcinoma)	
Infectious and microbiome				
Helicobacter pylori infection	Stomach			

IARC – International Agency for Research on Cancer; WCRF - World Cancer Research Fund.

Note: The scoping review and hence this table is not confined to Tier 1 cancers. Key limitations in the interpretation of this scoping review include: 1) exposures reported solely prior to 2021 were missed, including capture of key known cancer risk factors (e.g., human papilloma virus for cervical cancer, UV exposure for melanoma, and alcohol consumption and non-alcoholic fatty liver disease for liver cancer), as they were not reported in more recent studies; 2) the analysis may have missed relevant subgroup analyses of early-onset populations within broader studies of all ages where early-onset was not explicitly specified in the title or abstract. This was particularly relevant for pathogenic germline mutations (e.g., *BRCA1/2* and Lynch syndrome-associated variants), largely because studies focused on familial cancers do not often specify "early-onset cancers" specifically; and 3) the search terms limited capture of mechanistic studies and studies focused on pre-cancer outcomes which excluded several suspected risk factors (e.g., ultra-processed foods, PFAS, microplastics).

* Genetically predicted exposure and outcome estimated using Mendelian randomisation.

† In the scoping review, one study indicated that greater body fatness was protective, while another found that weight loss could be protective.

‡ Unhealthy dietary factors (e.g., red meat, sugar-sweetened beverage, Western dietary patterns, salty food)

§ Among first-degree relatives of probands (i.e., the first family member identified with a genetic condition).

Attributable exposures

- For this rapid-response project, an analysis of attributable exposures was undertaken by comparing national trends in known or suspected cancer risk factors which may have changed over time among young Australians, and whether the direction of this trend was consistent with observed increases in Tier 1 cancer incidence.
- A total of 100 exposures relating to the twelve Tier 1 cancers were assessed. Of these, 40 had convincing or sufficient evidence of causing at least one cancer type according to the WCRF or IARC, and 60 had probable or limited evidence. An additional 17 potential exposures were identified during the scoping review. Therefore, a total of 117 established and potential exposures were assessed.
- Where possible, nationally representative age- and sex-specific prevalence data across multiple timepoints were sourced for each of the identified exposures.
- The likelihood of a risk factor potentially contributing to observed cancer incidence trends for early-onset cancers was assessed using the rubric shown in Table 4. Risk factor and cancer incidence trends were visually assessed, comparing patterns for corresponding periods. These comparisons did not factor in a specified lag time between risk factor exposure and development of cancer, so the findings will be more reliable for risk factors with a shorter lag time to cancer development.
- This is an indicative analysis only, mindful that correlation does not constitute causation, that the effects of risk factors can be interactive and cumulative, and that lag time between exposure to risk factors and the development of cancer is likely to vary by both risk factor and cancer type. However, the observed patterns may be used to inform and guide priority questions for future research, and the limited availability of population-level risk factor prevalence trends reinforces the value of capturing high-quality population-level data on risk factors among young Australians.

Table 4. Exposure attribution classifications

Classification	Definition
Potentially contributed to increases in early-onset cancers in Australia	➢ Potentially harmful exposures that increased in prevalence ➢ Potentially protective exposures that decreased in prevalence
Potentially less likely to have contributed to increases in early-onset cancers in Australia	➢ Exposures that did not change in prevalence ➢ Potentially harmful exposures that decreased in prevalence ➢ Potentially protective exposures that increased in prevalence ➢ Exposures for which prevalence is thought to be zero or near-zero in Australia.
Existing data are inconsistent or unclear	➢ Trends are inconsistent across age groups ➢ Trends are unclear
Existing data are inadequate	➢ Data do not exist or are not publicly available ➢ Data are not available for at least two timepoints, or are not available over a period of at least 10 years

- The review identified several risk factors that may partly explain the rise in early-onset cancers in Australia. These comprised:
  - Two exposures with *convincing* evidence of a causal relationship with cancer:
    - One exposure associated with increased risk: Overweight and obesity (increased prevalence among younger Australians)

        - One exposure associated with decreased risk overall: Cervical screening participation (specifically, decreased cytology-based testing among younger Australians between 1996 and 2017, prior to the transition to HPV testing). As noted earlier, cervical cancer incidence trends are also likely to be associated with changes in screening technologies, HPV exposure and HPV vaccination.
    - Three exposures with *probable* evidence of a causal relationship with cancer:
        - Three exposures associated with decreased risk: Alcohol use for kidney cancer only (noting that alcohol use is causally related to increased risk of at least seven types of cancer)[4], consumption of fibre-containing foods, and consumption of dairy products (for colorectal cancer only) – each declined in prevalence.
    - Three exposures with *limited* evidence of a causal relationship with cancer:
        - Three exposures associated with decreased risk: Fruit consumption, consumption of foods containing vitamin C, and diets high in calcium – each declined in prevalence.
    - Three exposures identified only in the scoping review:
        - Two exposures associated with increased risk: Type I & II diabetes, and non-alcoholic fatty liver disease – each increased in prevalence.
        - One exposure associated with decreased risk: Pregnancy/parity – fertility rates declined and the proportion of females who had never given birth rose.
- The review also identified several risk factors that were <u>potentially less likely to have contributed to the rise</u> in early-onset cancers in Australia. These comprised:
    - Five exposures with *convincing* evidence of a causal relationship with cancer:
        - Four exposures associated with increased risk: Adult attained height, alcohol use (for breast, colorectal and pancreatic cancer), tobacco smoking, and processed meat consumption (for females only). – prevalence was either decreasing or stable
        - One exposure associated with decreased risk: Colorectal cancer screening participation – prevalence was either stable or zero
    - Six exposures with *probable* evidence of a causal relationship with cancer:
        - Four exposures associated with increased risk: Greater birthweight, red meat consumption (for males only), diets high in calcium, and glycaemic load. prevalence was either decreasing or stable
        - Two exposures associated with decreased risk: Body fatness in young adulthood for breast cancer only[5], and coffee consumption[6]. – each increased in prevalence

[4] There is a causal relationship between alcohol use and increased risk of at least seven types of cancer. According to the World Cancer Research Fund, There is also 'probable' evidence for a protective association between alcohol use and kidney cancer risk, for levels of drinking up to 30 grams of ethanol per day (three Australian standard drinks per day, or 21 Australian standard drinks per week).

[5] The World Cancer Research Fund reports 'convincing' evidence that overweight and obesity increases the risk of breast (post-menopausal), colorectal, kidney, pancreatic and uterine cancer, 'probable' evidence that it increases the risk of prostate cancer, and 'limited' evidence that it increases the risk of the cervical cancer, and a 'probable' protective association between body fatness in young adulthood and breast cancer. The IARC reports 'sufficient' evidence that the absence of excess body fatness decreases the risk of breast (post-menopausal), colorectal, kidney, pancreatic, thyroid and uterine cancer.

[6] According to the World Cancer Research Fund, there is 'probable' evidence for a protective association between coffee consumption and liver cancer risk. According to the International Agency for Research on Cancer, coffee consumption is classified as Group 3 (not classifiable as to its carcinogenicity to humans).

  - Five exposures with *limited* evidence of a causal relationship with cancer:
    - Three exposures associated with increased risk: Dairy product consumption (for prostate cancer only)[7], consumption of foods containing saturated fatty acids (males only), and opium consumption – each decreased in prevalence
    - Two exposures associated with decreased risk: Multivitamin supplement use, and vitamin D supplement use. – each increased in prevalence
  - Two exposures identified only in the scoping review:
    - Two exposures associated with increased risk: Triglycerides and hyperlipidaemia – each were either decreasing or stable
- For 10 exposures, it was not possible to determine whether they contributed to increases in early-onset cancers in Australia because while prevalence data were available, **prevalence trends were inconsistent or unclear**. These included:
  - Two exposures with a *convincing* level of evidence of a causal relationship with cancer:
    - One exposure associated with increased risk: Processed meat consumption (for males only)
    - One exposure associated with decreased risk: Physical activity.
  - Two exposures with a *probable* level of evidence:
    - One exposure associated with increased risk: Red meat consumption (for females only).
    - One exposure associated with decreased risk: Calcium supplement use.
  - Five exposures with a *limited* level of evidence:
    - Three exposures associated with increased risk: Androgenic (anabolic) steroid use, consumption of foods containing saturated fatty acids (for females only), and consumption of foods containing heme iron.
    - Two exposures associated with decreased risk: Non-starchy vegetable consumption, and serum vitamin D levels.
  - One exposure identified only in the scoping review:
    - One exposure associated with increased risk: Inflammatory bowel disease.
- For established and potential risk factors in addition to those highlighted above, it was not possible to determine whether they may have contributed to rising early-onset cancer rates in Australia, because prevalence data for young Australians were either unavailable or only covered a period of less than 10 years. Exposures in this category with a *convincing* level of evidence for a causal relationship with cancer included smokeless tobacco use, infections, medications, and most environmental and occupational exposures. Exposures identified only in the scoping review in this category included basal metabolic rate, fasting insulin, a ‘unhealthy dietary factors’, history of benign breast disease, metabolic syndrome, fluoranthene, age at menarche, free testosterone, history of primary infertility, insulin-like growth factor-1, and sex hormone-binding globulin.
- These findings are summarised below in Table 5, for exposures with sufficient prevalence data for this analysis (at least 10 years of observed prevalence trends).

[7] The World Cancer Research Fund reports ‘probable’ evidence of a protective association between dairy product consumption and colorectal cancer, limited evidence of a protective association with pre-menopausal breast cancer, and ‘limited’ evidence that dairy product consumption increases the risk of prostate cancer.

Table 5. Summary assessment of the potential contribution of exposures to increases in early-onset cancer rates in Australia (Tier 1 cancers).

CONTRIBUTION TO INCREASED EARLY- ONSET CANCER INCIDENCE	PREVALENCE TREND	EVIDENCE OF CAUSAL RELATIONSHIP WITH CANCER AT ALL AGES (IARC/WCRF)			EXPOSURE IDENTIFIED IN EARLY-ONSET CANCER SCOPING REVIEW ONLY
		CONVINCING	PROBABLE	LIMITED	
POTENTIAL CONTRIBUTION	Increasing	Overweight and obesity[a]			Diabetes Type I & II Non-alcoholic fatty liver disease
	Decreasing	Cervical cancer screening participation (protective)[b]	Alcohol use (kidney cancer only - protective)[c] Consumption of fibre-containing foods (protective) Dairy product consumption (colorectal cancer only - protective)[d]	Fruit consumption (protective) Consumption of foods containing vitamin C (protective) Diets high in calcium (protective)	Pregnancy rates and parity (protective)
POTENTIALLY LESS LIKELY	Increasing		Body fatness in young adulthood (breast cancer only - protective)[a] Coffee consumption (protective)	Multivitamin supplement use (protective) Vitamin D supplement use (protective)	
	Stable or zero	Colorectal cancer screening participation (protective)		Opium use	Triglycerides
	Decreasing or stable	Adult attained height	Greater birthweight		Hyperlipidaemia
	Decreasing	Alcohol use (breast and colorectal cancers only - harmful)[c] Tobacco smoking Processed meat consumption (females only)	Red meat consumption (males only) Diets high in calcium Glycaemic load	Dairy product consumption (prostate cancer only - harmful)[d] Consumption of foods containing saturated fatty acids (males only)	
UNDETERMINED	Inconsistent or unclear	Physical activity (protective) Processed meat consumption (males only)	Red meat consumption (females only) Calcium supplement use (protective)	Androgenic (anabolic) steroid use Non-starchy vegetable consumption (protective) Consumption of foods containing saturated fatty acids (females only) Consumption of foods containing heme iron Serum vitamin D levels (protective)	Inflammatory bowel disease

IARC, International Agency for Research on Cancer. WCRF, World Cancer Research Fund. [a]The WCRF reports convincing evidence that overweight and obesity increases the risk of breast (post-menopausal), colorectal, kidney, pancreatic and uterine cancer, probable evidence that it increases the risk of prostate cancer, and limited evidence that it increases the risk of the cervical cancer, and a probable protective association between body fatness in young adulthood and breast cancer. The IARC reports sufficient evidence that the absence of excess body fatness decreases the risk of breast (post-menopausal), colorectal, kidney, pancreatic, thyroid and uterine cancer. [b]Based on decreased participation in the National Cervical Screening Program (Pap testing) between 1996-1997 and January 2016-June 2017. [c]The WCRF reports convincing evidence that alcohol use increases the risk of breast (post-menopausal) and colorectal cancer, probable evidence that it increases the risk of pre-menopausal breast cancer, limited evidence that it increases the risk of pancreatic cancer, and probable evidence of a protective association between alcohol use and kidney cancer. [d]The WCRF reports probable evidence of a protective association between dairy product consumption and colorectal cancer, limited evidence of a protective association with pre-menopausal breast cancer, and limited evidence that dairy product consumption increases the risk of prostate cancer.

Emerging risk factors

- Recent Australian media coverage of early-onset cancers highlights the public interest in emerging potential risk factors, including for Tier 1 cancers as classified in this project.
- Risk factors mentioned in the media included some captured by this project's scoping review, and some additional potential risk factors not captured, including dietary factors, reproductive and developmental factors, medical factors and environmental factors (Table 6).
- The extent of media coverage is not always aligned with the level of evidence. For example, microplastics have received recent media coverage in Australia (Table 6), however systematic reviews of microplastics in relation to cancer do not find conclusive evidence of cancer risk. That research does tend to describe similar potential mechanisms for how microplastics might increase cancer risk, suggesting convergence across the evidence base. Potential mechanisms include DNA damaging potential, plausible biological pathways, ubiquitous presence in human tissue, exposure determinants and 'Trojan horse' effects (i.e., microplastics as carriers of other toxins). As for many emerging risk factors, this is a very active area of research and ongoing monitoring is warranted.

Table 6. Potential risk factors for early-onset cancer with recent Australian media coverage. *Included in findings from the scoping review.

Theme	Factors
Dietary	Convenience foods
	Diet (not further specified)
	Fatty foods
	Low carbohydrate intake
	Low soluble fibre intake
	Refined grains
	Sugars
	Sugary drinks*
	Taurine (as an ingredient in energy drinks)
	Western-style diet*
	Ultra-processed foods/processed foods
Reproductive/ Developmental	Caesarean section† rates increasing (and effects on the baby's microbiome)
	Exposures dating back to childhood, birth or pregnancy (not further specified)
	Having children later in life
	Not having children*
Medical	Antibiotic use/early exposure to antibiotics†
	Diabetes*
	Gut microbiome changes (mother's or children's), including:
	The toxin colibactin (released by a number of bacteria, including a strain of *Escherichia coli*)
	Escherichia coli bacterial infection
	Hydrogen sulphide-metabolising bacteria
	Pathogenic bacteria (not further specified)
	Lower use of protective medications (e.g. aspirin)†
	Stress
Environmental	Microplastics/nanoplastics/plastics/toxins in plastic (in food and drinking water)
	Per- and polyfluoroalkyl substances (PFAS) (including in cosmetics, non-stick frying pans, and stain-resistant fabrics)
	Perfluorooctanoic acid (PFOA) as a potential risk factor for breast cancer
	Synthetic chemicals/environmental chemicals/chemicals/toxins (not further specified) (food additives mentioned as an example of a chemical)

* Potential exposures identified in the scoping review and significantly associated with early-onset cancer risk.

† Included in the scoping review but not significantly associated with early-onset cancer risk.

3. Prevention and detection

Prevention

- The following national strategies and programs have a defined cancer prevention focus within their core objectives: Australian Cancer Plan (2023 - 2033); Aboriginal and Torres Strait Islander Cancer Plan (2023); National Preventive Health Strategy (2021–2030); National Tobacco Strategy (2023 – 2030); National Obesity Strategy (2022 - 2032); National Alcohol Strategy (2019 - 2028); Human papillomavirus immunisation program (2007 -); Hepatitis B immunisation program (2000 -); Fourth National Hepatitis B Strategy (2025 – 2030); Sixth National Hepatitis C Strategy (2025 – 2030); Roadmap to Liver Cancer Control (2023).
- These plans and strategies vary considerably in their scope and the extent of focus on cancer and/or early-onset cancer. The main differences relate to either the direct focus on objectives and priority areas specific to cancer or the extent to which the strategies work across disease control modalities.
- All strategies flag opportunities in policy and practice across the life course, however none promote interventions designed specifically and exclusively for people in the early-onset cancers age group.

Early detection

- Three national programs for the early detection of cancers are currently available to populations under 50 years - bowel, breast and cervical cancer screening programs (all Tier 1 cancers).
- In addition to the earlier detection of invasive cancer, bowel and cervical cancer screening also identify pre-cancerous disease, effectively contributing to cancer prevention, and breast screening identifies pre-invasive cancer (i.e., Ductal Carcinoma in Situ (DCIS)), effectively contributing to prevention of invasive breast cancer.
- Analysis of the potential impact of the three screening programs on early-onset cancer incidence could be instructive. For example, bowel cancer incidence in the screened population (50-74 year-olds) has fallen significantly since the program's introduction due to potential cancers being detected as precancerous adenomas. The phase-in of 45-49-year-olds from 2024 is expected to reduce incidence rates in that cohort.

Case studies

- The relationship between observed incidence trends and associated changes in health services and related outcomes varies by cancer, depending on factors such as prevention, referral pathways, diagnostic accuracy, cancer classification and overdiagnosis. For example:
  - **Breast cancer:** Early-onset breast cancer has increased significantly over the last 20 years. This has occurred among BreastScreen Australia-eligible age group (40-49 years) and among younger females (20-29 and 30-39 years). Improvements in breast imaging and risk assessment may have contributed to these increases in incidence, but this is not yet known. Incidence may have been greater without the contribution of medical prevention targeted to females at very high risk of breast cancer. Reassuringly, for females aged 20-49 years improvements in breast cancer mortality by age group and by birth cohort are more marked over time, compared to changes in incidence.
  - **Prostate cancer:** Early-onset prostate cancer incidence has fluctuated over time and tends to mirror PSA testing rates. These trends are potentially also influenced by a reduced threshold for biopsies introduced in 2002 and, more recently, increased availability of mpMRI. Such changes in practice may have affected trends in incidence. Overdiagnosis remains an ongoing concern, with likely increases due to the reduced

threshold for biopsies, and a less certain impact due to mpMRI imaging (noting that mpMRI is thought to reduce incidence by preventing unnecessary biopsies, which could contribute to overdiagnosis, although there are no population-level Australian studies and data). There is some evidence suggesting that prostate cancer in younger males might be a distinct subtype.

- **Thyroid cancer:** Early-onset thyroid cancer incidence has increased markedly for both sexes and for all age groups assessed, with ongoing low mortality rates. The contribution of changes in testing technologies is challenging to identify, due to a lack of unique Medicare items for the imaging tests involved. Significant rates of overdiagnosis are likely.

4. Psychosocial aspects

Psychosocial impacts of early-onset cancer

- International systematic reviews, largely defining adolescents and young adults (AYA) as ages 15–39 years, report a wide range of psychosocial challenges and needs across domains, such as psychological wellbeing, social connectedness, sexual and reproductive health, coping, education and employment, and health-related quality of life (HRQoL).
- Evidence remains limited in Australia, with most evidence focussed on the AYA age range, usually considered as a younger age range in Australia (15-24 or 15-25 years) compared to other settings (usually up to 39 years). In general, there is a notable variation in how AYAs are defined across the sector (Table 7).

Table 7. Comparison of adolescent and young adult (AYA) definitions and age ranges by country and institution

Country	Institution	Definition	Age range
Australia	Australian Institute of Health and Welfare (AIHW)[9]	Young people	15-24
	Australian Cancer Plan[10]	Adolescents and young adults	15-24
	Canteen[11]	Young people	12-25
UK	Cancer Research UK[12]	Young people	15-24
	Teenage Cancer Support[13]	Young people	13-24
US	National Cancer Institute (NCI)[14]	Adolescents and young adults	15-39
	Surveillance, Epidemiology, and End Results Program (SEER)[15]	Adolescents and young adults	15-39 (previously 15-29)
	US Census Bureau[16]	Young adults	18-34
Canada	Canadian Cancer Society[17]	Adolescents and young adults	15-39
International	World Health Organization (WHO)[18]	Adolescents	10-19
	World Health Organization (WHO)[18]	Young adults	20-24
	EUROCARE[19]	Adolescents and young adults	15-39
	European Society for Medical Oncology (ESMO)/the European Society for Paediatric Oncology (SIOPE)[20]	Adolescents and young adults	15-39

- Key issues from Australian studies included lack of survivorship care plans, poor information sharing, unmanaged treatment side effects, insufficient sexual health counselling, and employment loss linked to neurocognitive complications.
- Differences in psychosocial impacts and needs according to sex were noted, often highlighting greater impacts for females, but further work is required to more comprehensively assess the differing impacts by sex, and to consider psychosocial impact and unmet needs related to early-onset cancer in gender and sexually diverse people.
- Addressing these gaps will require large, collaborative studies, age-stratified analyses, and integrated survivorship models that incorporate psychosocial screening, vocational support, and sensitive discussions on fertility and sexual health.

Guidelines for psychosocial support services:

- The review identified no existing Australian guidelines for psychosocial support services specifically focused on the age range aligning with early-onset cancers (20-49 years).
- Two such guidelines are available for the AYA age range, as well as various guidelines for all adults.
- There is a clear need for the development of guidelines for all people with early-onset cancer.
- These guidelines and the methods for their development could draw from existing guidelines for other age groups.

Evidence gaps

This project identified a range of evidence gaps, summarised in Table 8.

Table 8. Evidence Gaps as identified in this project.

Topic	Evidence gap
1. Priority cancers and cancer incidence and mortality trends	1.1 Standard definitions for the age range of interest for early-onset cancers in studies reporting the global incidence of early-onset cancers, with reference to both the lower (e.g., 15 vs 20 years) and upper age limits (e.g., 39 vs 49 years)
	1.2 Recent global trends including joinpoint regression findings (AAPCs) reported by age and sex since 2012
	1.3 A systematic synthesis, comparison and analysis of Australian outcomes compared to global outcomes
	1.4 Analysis of priority cancers and updated trends incorporating a sufficient duration of observed incidence and mortality data following the COVID-19 pandemic
	1.5 Analysis of cancers meeting Tier 1 criteria only within specific age and sex groups (e.g., 20-29, 30-39, or 40-49 years), even if not for the whole early-onset age range (20-49 years) . This would include, for example, ovarian cancer, which met the criteria for ages 20-29 years only.
	1.6 Evaluation of the mortality burden of early-onset cancers through analysis of linked datasets
	1.7 Analysis focused on priority populations (e.g. by socio-economic status, remoteness area and Indigenous status)
2. Risk factors	2.1 Standard definitions of the age threshold for early-onset cancers used in studies examining risk factors for early-onset cancers, regardless of cancer type, particularly with reference to the upper age limit (e.g., <50 vs <55 years)
	2.2 A systematic review of evidence about risk factors associated with early-onset cancers, building on the scoping review conducted for this project, incorporating a more comprehensive search strategy (including primary studies published prior to 2021 and studies examining risk factors across multiple cancer types rather than focusing on a specific cancer), and undertaking a critical appraisal of included studies
	2.3 Available evidence on risk factors for some early-onset cancers, including some cancers classified as Tier 1 in the analysis (e.g., kidney cancer or testicular cancer).
	2.4 Evidence reporting the attributable fractions of risk factors (the proportion of cancers that can be linked to each specific risk factor)

	2.5 Evidence on risk factors associated with biological and hormonal mechanisms, clinical and medical history, and genomics/genetics, particularly for less common cancer types
	2.6 Synthesis of evidence on shared or common causal risk factors for various early-onset cancers (e.g., alcohol, smoking, diet)
	2.7 Evidence where risk factor exposure is assessed as an 'exposome' (the totality of environmental exposures over life)
	2.8 Analysis of risk factors according to whether they are modifiable or non-modifiable, and the associated interventions likely to have the greatest impact either overall or to address disparities between identified populations.
	2.9 Analysis of emerging potential exposures such as microplastics, perfluorooctanoic acid and gut microbiome changes
	2.10 Prevalence trends for many risk factors in younger Australians, including infections, medications and environmental exposures
	2.11 Long-term prevalence trends for the majority of risk factors of interest
	2.12 Longitudinal analysis of the causal association between observed prevalence trends of risk factors in younger Australians and associated cancer incidence
	2.13 Evidence on the lag time between exposure to risk factors and cancer development, for each risk factor and cancer type.
3. Prevention and early detection	3.1 A systematic and comprehensive analysis of clinical practices in early detection and prevention of early-onset cancers, including an overview of resourcing and implementation and evaluation plans
	3.2 Research studies concurrently assessing risk factor exposures and the design and utilisation of health services for early detection of early-onset cancers
	3.3 National evidence- and consensus-based guidelines for improved management of early-onset cancers.
	3.4 Colorectal cancer incidence in ages 45-49 compared to older and younger age groups following the 2024 expansion of the National Bowel Cancer Screening Program
4. Psychosocial aspects	4.1 A systematic review of psychosocial impacts of early-onset cancers, building on the scoping review conducted for this project
	4.2 Consensus on definitions of the psychosocial impact of cancer and the age range for adolescents and young adults (AYA)
	4.3 Evidence on psychosocial impact for people older than the AYA age group (i.e. the mid-adult (MA) age group of 26-49 years).
	4.4 Evidence on psychosocial impacts from longitudinal studies

	4.5 Validated or standardised tools to assess psychosocial impacts, and consensus on threshold values defining psychological distress
	4.6 Evidence on psychosocial impacts of early-onset cancers according to different age groups within the early-onset cancer age range (e.g., 20-25 vs 26-49 years)
	4.7 Evidence on psychosocial impacts of early-onset cancers according to sex
	4.8 Australian guidelines for psychosocial support services for Australians with early-onset cancer, with consideration of age groups (e.g., younger vs older) and sex, given that existing guidelines are largely designed for AYAs or all adults
	4.9 Mapping of available psychosocial services for people impacted by early-onset cancer to inform the development of Australian guidelines for psychosocial support services
	4.10 Centralised and consistent information about psychosocial support services for Australians with early-onset cancer

Priority next steps

There is a clear need for momentum and for increased investment in and development of the evidence space on early-onset cancers in Australia. To help guide these activities, the project identified a set of priority next steps as guided by the evidence and the associated gaps in evidence (Table 9).

For all steps identified, it would be important to assess equity where feasible, recognising likely disparities in outcomes for people with lower socio-economic status, people living in rural and remote areas, people with culturally and linguistically diverse (CALD) backgrounds, Aboriginal and Torres Strait Islander people, LGBTQIA+ people, people with disabilities, and people living with mental illness.

Priority next steps include recommendations for research, including longitudinal studies to assess the causal association between observed prevalence trends of risk factors in younger Australians and associated cancer incidence and studies estimating the attributable fractions of risk factors (the proportion of cancers that can be linked to each specific risk factor). Such studies are becoming more feasible on a large scale due to the expansion of Australian linked population datasets such as the AIHW National Health Data Hub,[21] the Enduring Cancer Data Linkage (CanDLe)[22] dataset which has capacity to be linked to the Commonwealth Person Level Integrated Data Asset (PLIDA)[23] collection, and through the future Sax Institute's 18 and Up Study[24] cohort.

Of note, the AIHW is planning to release cancer incidence, mortality and survival statistics by socio-economic disadvantage, remoteness area and Indigenous status in 2026[25], which will enable additional analyses of national incidence for various priority populations.

Table 9. Priority next steps to address key evidence, policy and practice gaps related to early-onset cancers in Australia. For all steps, equity should be considered, recognising likely disparities in outcomes for people with lower socio-economic status, people living in rural and remote areas, people with culturally and linguistically diverse (CALD) backgrounds, Aboriginal and Torres Strait Islander people, LGBTQIA+ people, people with disabilities, and people living with mental illness.

TOPIC	PRIORITY NEXT STEPS
1. Incidence and mortality	**1A.** Updated analysis of early-onset cancer incidence and mortality trends by age and sex, as additional post-COVID observed data becomes available
	1B. Further analysis of cancers meeting Tier 1 criteria within specific age and sex groups (e.g., 20-29, 30-39, or 40-49 years), even where the criteria were not met for the whole early-onset age range (20-49 years)
	1C. Analysis of incidence and mortality trends for understudied and less common early-onset cancers
	1D. Analysis of the mortality burden of early-onset cancers and the contribution of lead time bias and overdiagnosis, using population linked data incorporating longitudinal data on diagnoses and deaths, by age group and sex
2. Risk factors	**2A.** Assessment of emerging and potential risk factors for early-onset cancers, using frameworks such as that developed for the IARC Monographs Programme
	2B. Improved data on the prevalence of risk factors in the Australian population aged under 50 years, especially for infections, medications, and environmental exposures
	2C. Further research to assess associations between risk factors and early-onset cancers in the Australian population, such as analysis of longitudinal population linked datasets, with consideration of interaction between risk factors, lag time between risk factor exposure and cancer development, and the 'exposome (the totality of environmental exposures over life)', and focussed analysis of risk factors associated with multiple early-onset cancers. Engagement in global pooled data initiatives may also be beneficial.
	2D. Assessment of known risk factors for early-onset cancers as targets for public health interventions, using frameworks such as that developed for the IARC Handbooks of Cancer Prevention
3. Prevention and detection	**3A**. A systematic and comprehensive analysis of clinical practices in early detection and prevention of early-onset cancers, including an overview of resourcing and implementation and evaluation plans
	3B. Research studies concurrently assessing risk factor exposures and the design and utilisation of health services for early detection of early-onset cancers
	3C. National evidence- and consensus-based guidelines for improved management of early-onset cancers
	3D. Analysis of colorectal cancer incidence in ages 45-49 compared to older and younger age groups following the 2024 expansion of the National Bowel Cancer Screening Program
4. Psychosocial impacts	**4A.** Updated and expanded evidence review of the psychosocial impacts of early-onset cancers
	4B. Definition of a core set of validated, minimum measures to capture age and sex specific psychosocial impacts of early-onset cancers to strengthen the evidence base and enable comparisons across studies, populations and cancer types

TOPIC	PRIORITY NEXT STEPS
4. Psychosocial impacts (continued)	**4C.** Longitudinal studies of psychosocial impacts in the early-onset cancer age range, using validated tools, including patient-reported experience and outcome measures
	4D. Australian guidelines for psychosocial support of people with early-onset cancer, with consideration of differences according to age group and sex, and including clear processes and resourcing for post-hoc evaluation and implementation, with the development of complementary centralised patient-centric information and support
	4E. Mapping of available psychosocial services for people impacted by early-onset cancer
	4F. Centralised, consumer-centric information tailored for people with early-onset cancers

Acronyms and glossary

Acronyms and a glossary of terms used in this Executive Summary are shown below.

List of acronyms

Acronym	*Definition*
AAPC	Average annual percentage change
AIHW	Australian Institute of Health and Welfare
ASR	Age standardised rate
AYA	Adolescents and young adults
BMI	Body mass index
CALD	Culturally and linguistically diverse
CI	Confidence interval
DCIS	Ductal carcinoma in situ
EOC	Early-onset cancer
GBD	Global Burden of Disease
HR	Hazard ratio
HRQoL	Health-related quality of life
HPV	Human papillomavirus
IARC	International Agency for Research on Cancer
MA	Mid-adults
mpMRI	Multiparametric magnetic resonance imaging
MR	Mendelian randomisation
NAFLD	Non-alcoholic fatty liver disease
OR	Odds ratio
PFAS	Per- and polyfluoroalkyl substances
PFOA	Perfluorooctanoic acid
PREMs	Patient-reported experience measures
PROMs	Patient-reported outcome measures
PSA	Prostate specific antigen
RR	Relative risk
SEER	Surveillance, Epidemiology and End Results Program
WHO	World Health Organization
WCRF	World Cancer Research Fund

Glossary of terms

AAPC (Average Annual Percentage Change)

A summary measure of a trend over a specified period, derived from Joinpoint regression. The AAPC represents the weighted average of the Annual Percentage Changes (APCs) from each segment of the Joinpoint model. AAPCs are often reported for trends of incidence and mortality rates (where those rates are usually age-standardised).

AYA (Adolescent and Young Adult)

A population group for which age definitions vary across the sector; Australian studies commonly define this group as 15–24 or 15–25 years.

Early-onset cancers

For the purpose of this report, cancers diagnosed between 20 and 49 years of age.

Emerging potential risk factors for early-onset cancer

Potential risk factors specifically for early-onset cancer that have not been classified as established risk factors by the International Agency for Research on Cancer (IARC) or as having convincing evidence by the World Cancer Research Fund (WCRF), but have shown statistically significant associations in at least one study.

Established risk factors (exposures)

Risk factors classified as having sufficient evidence for a causal relationship with cancer by the IARC or WCRF.

Exposure attribution

For the purposes of this report, exposures classified according to how likely it is that they contributed to increases in early-onset cancer in Australia based on observed national prevalence trends.

Exposure prevalence

The proportion of a defined population that has been exposed to a risk factor at a specified point in time.

Exposures which are potentially less likely to have contributed to increases in early-onset cancers in Australia

Exposures that did not change in prevalence over time, potentially harmful exposures that decreased in prevalence, potentially protective exposures that increased in prevalence, or exposures thought to be zero or near-zero in Australia.

Exposures which potentially contributed to increases in early-onset cancers in Australia

Potentially harmful exposures that increased in prevalence over time, or potentially protective exposures that decreased in prevalence over time.

HRQoL (Health-related quality of life)

A multidimensional concept reflecting the impact of an individual's physical and mental health on their overall quality of life.

Incidence numbers and rates

The number of new cases of disease occurring in a defined population during a specified period; incidence rates express these cases relative to person-time or population size.

Joinpoint regression (sometimes 'join point')

A type of regression modelling in which linear segments are fitted to a trend (on a logarithmic scale) to identify changes in trends and statistically significant turning points.

Lead time bias

Lead time is the duration of time by which a cancer diagnosis is advanced due to early detection (like routine screening) compared to when it would have been diagnosed naturally through symptoms. This can lead to lead time bias, where an individual appears to survival for longer following their cancer diagnosis, but this may be due only to earlier diagnosis with no change to the natural history of their disease.

Overdiagnosis

Diagnosis of cancer that would not have otherwise been diagnosed during a person's lifetime.

Proportional distribution by type

The relative proportion of each cancer in a given population.

Rank by cancer type

Ranking cancers by a specified metric, such as number of cases or incidence rates in a given population.

Scoping review

A form of evidence synthesis that employs systematic and iterative methods to identify, map and summarise the breadth of existing or emerging literature on a topic, but to a lesser extent than a systematic review.

Sufficiently common early-onset cancers

Early-onset cancers with an incidence rate for ages 20-49 years above a sex-specific threshold, being ≥1.270 cases per 100,000 in males and ≥1.967 cases per 100,000 in females, equating to approximately 1% of overall cancer incidence in those groups.

Systematic review

A method of evidence synthesis that identifies, appraises and analyses all relevant studies addressing a specific research question using predefined criteria.

Tier 1 cancers

Sufficiently common early-onset cancers that are in the top 10 increasing cancer types in terms of absolute rates.

Tier 2 cancers

Early-onset cancers that are in the top 10 increasing cancer types in terms of relative increases, and/or are in the top 10 increasing cancer types in terms of absolute increases but are not sufficiently common to be considered Tier 1 cancers.

References

1. Gupta, S. *et al.* International Trends in the Incidence of Cancer Among Adolescents and Young Adults. *J Natl Cancer Inst* **112**, 1105–1117 (2020).
2. Ugai, T. *et al.* Is early-onset cancer an emerging global epidemic? Current evidence and future implications. *Nat Rev Clin Oncol* **19**, 656–673 (2022).
3. Canfell, K., Sitas, F. & Beral, V. Cervical cancer in Australia and the United Kingdom: comparison of screening policy and uptake, and cancer incidence and mortality. *Med J Aust* **185**, 482–486 (2006).
4. Australian Institute of Health and Welfare. National Cervical Screening Program monitoring report 2025, Cervical cancer. *Australian Institute of Health and Welfare* https://www.aihw.gov.au/reports/cancer-screening/ncsp-monitoring-report-2025/contents/about [Accessed 30 March 2026].
5. Smith, M. & Canfell, K. Impact of the Australian National Cervical Screening Program in women of different ages. *Med. J. Aust.* **205**, (2016).
6. Hall, M. T. *et al.* Projected future impact of HPV vaccination and primary HPV screening on cervical cancer rates from 2017–2035: Example from Australia. *PLOS ONE* **13**, e0185332 (2018).
7. Hall, M. T. *et al.* The projected timeframe until cervical cancer elimination in Australia: a modelling study. *The Lancet Public Health* **4**, e19–e27 (2019).
8. C4 (NHMRC Centre of Research Excellence in Cervical Cancer Control)). 2025 Cervical Cancer Elimination Progress Report. https://report.cervicalcancercontrol.org.au/ [Accessed 30 January 2026].
9. Australian Institute of Health and Welfare. Health of young people. https://www.aihw.gov.au/reports/children-youth/health-of-young-people [Accessed 12 January 2026].
10. Australian Cancer Plan. Adolescents & Young Adults. https://www.australiancancerplan.gov.au/populations/adolescents-and-young-adults [Accessed 12 January 2026].
11. Canteen. About Canteen - Supporting Young People Impacted by Cancer. https://www.canteen.org.au/about-us/about-canteen [Accessed 12 January 2026].
12. Cancer Research UK. Teenage and young adult (TYA) cancers. https://www.cancerresearchuk.org/about-cancer/childrens-cancer/teenage-young-adult-tya [Accessed 12 January 2026].
13. Teenage Cancer Trust. About us. https://www.teenagecancertrust.org/about-us [Accessed 12 January 2026].
14. National Cancer Institute. Adolescents and Young Adults with Cancer. https://www.cancer.gov/types/aya [Accessed 12 January 2026].
15. Surveillance, E. Cancer Stat Facts: Cancer Among Adolescents and Young Adults (AYAs) (Ages 15–39). https://seer.cancer.gov/statfacts/html/aya.html.
16. United States Census Bureau. Young Adults: Then and Now. https://www.census.gov/programs-surveys/sis/resources/visualizations/young-adults.html [Accessed 12 January 2026].
17. Canadian Cancer Society. Adolescents and young adults with cancer. https://cancer.ca/en/about-us/our-health-equity-work/adolescents-and-young-adults-with-cancer [Accessed 12 January 2026].
18. World Health Organization. Adolescent and young adult health. https://www.who.int/news-room/fact-sheets/detail/adolescents-health-risks-and-solutions [Accessed 12 January 2026].
19. Trama, A. *et al.* Survival of European children, adolescents and young adults diagnosed with haematological malignancies in the period 2000-2013: Results from EUROCARE-6, a population-based study. *European journal of cancer (Oxford, England : 1990)* **222**, 115336 (2025).

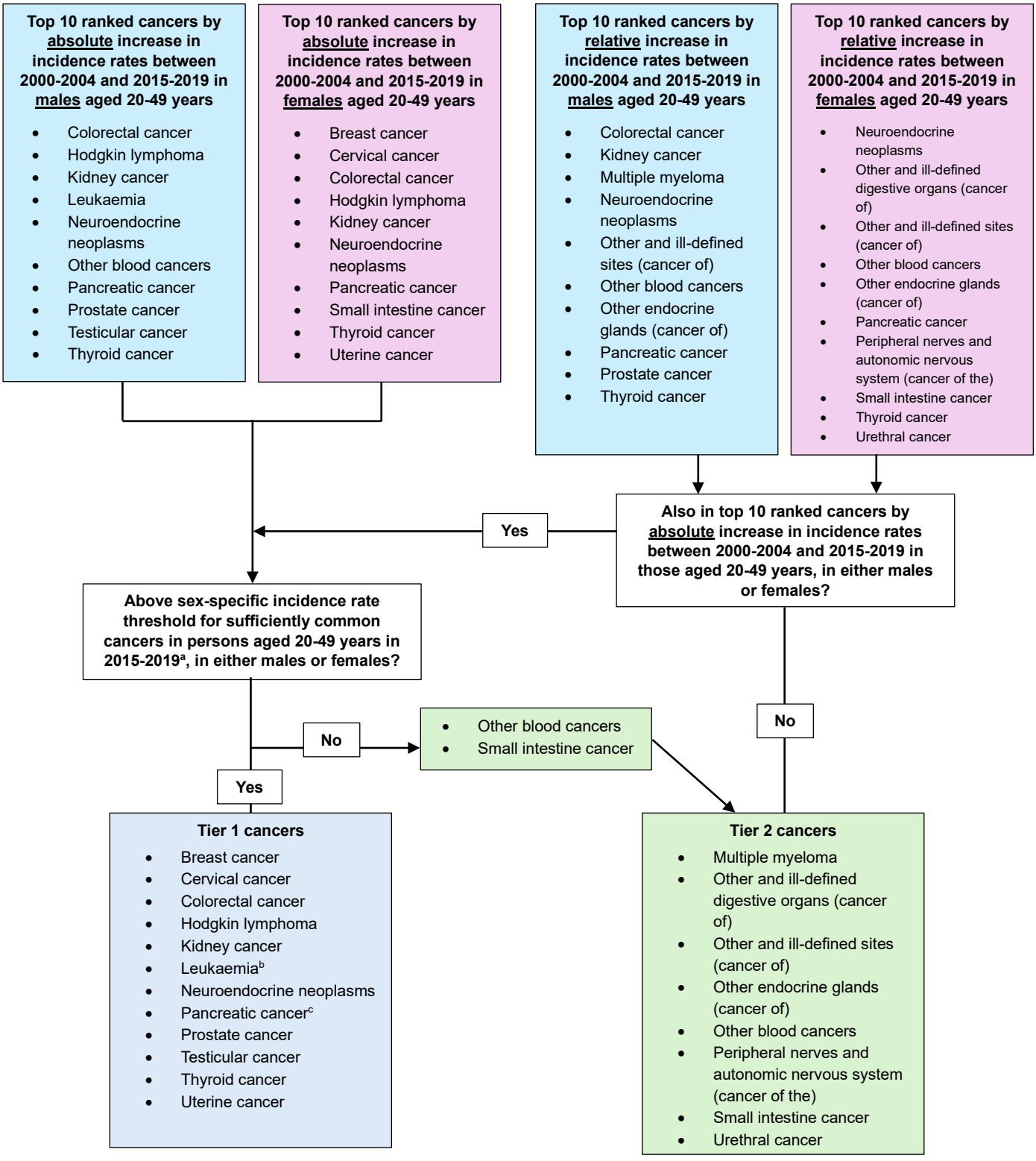
20. Ferrari, A. *et al.* Adolescents and young adults (AYA) with cancer: a position paper from the AYA Working Group of the European Society for Medical Oncology (ESMO) and the European Society for Paediatric Oncology (SIOPE). *ESMO Open* **6**, 100096 (2021).
21. Australian Institute of Health and Welfare. National Health Data Hub. *Australian Institute of Health and Welfare* https://www.aihw.gov.au/reports-data/nhdh [Accessed 23 March 2026].
22. Cancer Institute NSW. Enduring Cancer Data Linkage (CanDLe) Program. *Cancer Institute NSW* https://www.cancer.nsw.gov.au/research-and-data/cancer-data-and-statistics/data-available-on-request/candle-program [Accessed 30 January 2026].
23. Australian Bureau of Statistics. Person Level Integrated Data Asset (PLIDA). https://www.abs.gov.au/about/data-services/data-integration/integrated-data/person-level-integrated-data-asset-plida [Accessed 30 January 2026].
24. Sax Institute. 18 and Up Study. https://www.saxinstitute.org.au/solutions/18-and-up-study/ [Accessed 30 January 2026].
25. Cancer data in Australia, Report content. *Australian Institute of Health and Welfare* https://www.aihw.gov.au/reports/cancer/cancer-data-in-australia/contents/summary [Accessed 29 January 2026].

Appendix

Table 10. Project detailed objectives and their associated methods and technical chapters (*unpublished*).

Topic	Objective	Methods	Chapter
Priority cancers	1.1 Identify which cancer types are recording increased incidence of early-onset (age 20–49-year-olds), both globally and in Australia over the past 30 years.	Epidemiological analyses and a scoping review	Chapter 1. Priority cancers and cancer incidence and mortality trends
Aetiology and risk factors	1.2 Establish confirmed and suspected aetiologic causes and risk factors.	Scoping reviews	Chapter 2. Risk factors
Prevention and detection strategies	1.3 Describe existing prevention and detection strategies in relation to incidence.	Landscape analyses	Chapter 3. Prevention and detection
Incidence and mortality trends	1.4 Indicate where detection interventions, overdiagnosis and lead-time bias might be factors in increased early-onset cancers.	Comparison of incidence and mortality trends (including cf. ages 50-54)	Chapter 1. Priority cancers and cancer incidence and mortality trends
Attributable exposures	1.5 Pinpoint exposures that might be attributable for increased early-onset cancers.	Assessment of cohort trends in prevalence over the past 20-50 years	Chapter 2. Risk factors
Psychosocial outcomes	1.6 Summarise evidence on psychosocial impacts on people affected.	Scoping review	Chapter 4. Psychosocial aspects
Psychosocial support services	1.7 Summarise psychosocial support services available to people affected.	Landscape analyses	Chapter 4. Psychosocial aspects
Evidence gaps	2.1 Analyse findings from key questions 1-3 and produce a summary of evidence gaps according to emerging themes from a proposed *a priori* list.		Executive Summary
Roadmap	2.2 Propose a program of targeted research to address these gaps, presented as a high-level five-year roadmap to reducing the burden of early-onset cancer in Australia and strengthen knowledge about established and prospective causes and interventions.		Executive Summary

Figure 5. The framework applied to identify priority cancers using changes in cancer incidence rates from 2000-2004 to 2015-2019 in Australian males and females aged 20-49 years.



[a] The threshold incidence rate for males was 1.270 cases per 100,000 males in 2015-2019 (1% of the incidence rate of all cancers combined in males), and for females was 1.967 cases per 100,000 females in 2015-2019 (1% of the incidence rate of all cancers combined in females). [b] Non-sex-specific cancer which was in the top 10 ranked cancers by absolute increase in incidence rates in males only, but considered a Tier 1 cancer for both males and females. [c] Above threshold incidence rate in males only, but considered a Tier 1 cancer for both males and females.