Government of **Western Australia**
Department of **Health**

Choosing Cancers for ‘Your Say On Cancer In WA’

HealthyWA
healthywa.wa.gov.au

Contents

Introduction

The Western Australian Chief Health Officer's (CHO) Report is a new series of reports released by the Department of Health Western Australia that examine health related issues in Western Australia (WA). The purpose of the series is to provide a useful resource for the public as well as for policy planners interested in heath related matters.

The current issue set to be released in mid to late 2015 will be titled 'Choices in Cancer Control in Western Australia'. The aim of this report is to reflect on how well WA is progressing in cancer control, with a specific focus on cancer prevention.

This report will be informed by the responses of the public consultation *Your Say on Cancer in WA* (yoursayoncancer.health.wa.gov.au).The consultation enables respondents to give their views in response to cancer statistics and short expert commentaries.

The consultation concentrates on seven cancers with the biggest impact on the WA community and which offer opportunities for prevention. This document describes how these seven cancers of focus were chosen.

Selection Method

To establish the focus for the next Chief Health Officer's report, we initially reviewed the age-standardised incidence and mortality rates of cancers recorded in the WA Cancer Registry (WACR) in 2012 (data extracted December 2013). Data for males and females were analysed separately. From these data we identified the highest-ranking 25 cancers for each sex for incidence and mortality, for further investigation. Twenty-nine cancers in females (Table 1) and 27 cancers in males (Table 2) were identified for consideration.

This list of cancers was then narrowed to a list of cancers for additional consideration based on meeting two of four possible criteria (Table 3):

- Top 12 for incidence rate 2012
- Top 12 for mortality rate 2012
- Statistically significant change in incidence trend (1992-2012)
- Statistically significant change in mortality trend (1992-2012)

For non sex-specific cancers (excluding breast cancer), the cancer had to meet the minimum criteria in both males and females in order to qualify for additional review.

A total of 12 cancers were eligible for additional consideration; breast, cervical, ovarian, uterine, prostate, colorectal, leukaemia, lung, lymphoma, melanoma, pancreatic and oesophageal & stomach cancer. The unknown primary site category also met the criteria for further review. However, this category was excluded from the list due to a lack of specificity regarding the cancers comprising this category [2].

Table 1 Top 29 cancers in females by incidence and mortality rankings for 2012, incidence and mortality trends between 1992 and 2012, and eligibility for additional review

Cancer	Incidence Rank	Mortality Rank	Significant Change in Incidence	Significant Change in Mortality	Eligible for Additional Review
Breast	1	2	Yes (↑)	Yes (↓)	Yes
Colorectal	2	3	Yes (↓)	Yes (↓)	Yes
Melanoma*	3	13	Yes (↓)	No	Yes
Lung	4	1	Yes (↑)	Yes (↑)	Yes
Lymphoma	5	7	Yes (↑)	Yes (↓)	Yes
Thyroid gland	6	34	Yes (↑)	-	Yes
Uterine	7	12	Yes (↑)	No	Yes
Ovarian	8	5	No	Yes (↓)	Yes
Pancreatic	9	4	No	No	Yes
Leukaemia	10	9	No	Yes (↓)	Yes
Unknown primary	11	6	Yes (↓)	Yes (↓)	Yes
Cervical	12	22	Yes (↓)	Yes (↓)	Yes
Kidney	13	19	Yes (↑)	-	No
Oesophageal and Stomach	14	8	Yes (↓)	Yes (↓)	Yes
Brain	15	10	No	No	No
Myeloma	16	11	Yes (↑)	No	Yes
Bladder & urinary tract	17	15	No	No	No
Oropharyngeal/Larynx	18	17	No	-	No
Skin (NMSC exc. SCC/BCC)	19	21	Yes (↓)	-	No
Liver	20	16	-	-	No
Gallbladder / bile ducts	21	14	No	-	No
Myelodysplastic diseases	22	18	No	-	No
Connective/ soft tissues	23	30	-	-	No
Lip (external)	24	-	-	-	No
Other Head & Neck	25	28	-	-	No
Vulva	26	20	-	-	No
Small intestine	27	24	-	-	No
Myeloprolif. d/o (chronic)	29	25	-	-	No
Mesothelioma	30	23	-	-	No

Highlighted cells – meets criteria

Empty Cells – unable to reliably calculate change in incidence or mortality due to 5 or more years with an annual number of cases or deaths equal to or less than 20.

*Melanoma includes the very small proportion of non-skin melanomas that are of unknown primary site.

Table 2 Top 27 cancers in males by incidence and mortality rankings for 2012, incidence and mortality trends between 1992 and 2012, and eligibility for additional review

Cancer	Incidence Rank	Mortality Rank	Significant Change in Incidence	Significant Change in Mortality	Eligible for Additional Review
Prostate	1	2	Yes (↑)	Yes (↓)	Yes
Melanoma*	2	6	No	Yes (↑)	Yes
Colorectal	3	3	No	Yes (↓)	Yes
Lung	4	1	Yes (↓)	Yes (↓)	Yes
Lymphoma	5	8	Yes (↑)	Yes (↓)	Yes
Oesophageal and Stomach	6	4	Yes (↓)	Yes (↓)	Yes
Bladder & urinary tract	7	9	Yes (↓)	Yes (↓)	Yes
Oropharyngeal/Larynx	8	14	Yes (↓)	Yes (↓)	Yes
Kidney	9	16	Yes (↑)	No	Yes
Unknown primary	10	7	Yes (↓)	Yes (↓)	Yes
Leukaemia	11	15	No	Yes (↓)	Yes
Pancreatic	12	5	Yes (↑)	No	Yes
Brain	13	10	No	Yes (↓)	Yes
Liver	14	11	Yes (↑)	Yes (↑)	Yes
Myeloma	15	17	Yes (↑)	No	No
Mesothelioma	16	13	No	Yes (↓)	No
Lip (external)	17	34	Yes (↓)	-	No
Skin (NMSC exc. SCC/BCC)	18	12	No	-	No
Testis	19	31	Yes (↑)	-	No
Thyroid gland	20	23	-	-	No
Myelodysplastic diseases	21	18	Yes (↑)	-	No
Other Head & Neck	22	24	No	-	No
Connective/ soft tissues	23	25	-	-	No
Gallbladder / bile ducts	24	19	No	-	No
Myeloprolif. d/o (chronic)	25	21	-	-	No
Small intestine	26	22	-	-	No
Anus	30	20	-	-	No

Highlighted cells – meets criteria

Empty Cells – unable to reliably calculate change in incidence or mortality due to 5 or more years with an annual number of cases or deaths equal to or less than 20.

*Melanoma includes the very small proportion of non-skin melanomas that are of unknown primary site.

Table 3 Review of eligibility criteria for the top cancers in males and females

Cancer	Meets Female Criteria	Meets Male Criteria	Additional Review?*
Breast	Yes	-	Yes
Cervical	Yes	-	Yes
Ovarian	Yes	-	Yes
Uterine	Yes	-	Yes
Vulva	No	-	No
Prostate	-	Yes	Yes
Testis	-	No	No
Anus	-	No	No
Bladder & urinary tract	No	Yes	No
Brain	No	Yes	No
Colorectal	Yes	Yes	Yes
Connective/ soft tissues	No	No	No
Gallbladder / bile ducts	No	No	No
Kidney	No	Yes	No
Leukaemia	Yes	Yes	Yes
Lip (external)	No	No	No
Liver	No	Yes	No
Lung	Yes	Yes	Yes
Lymphoma	Yes	Yes	Yes
Melanoma	Yes	Yes	Yes
Mesothelioma	No	No	No
Myelodysplastic diseases	No	No	No
Myeloma	Yes	No	No
Myeloprolif. d/o (chronic)	No	No	No
Oesophageal and Stomach	Yes	Yes	Yes
Oropharyngeal/Larynx	No	Yes	No
Other Head & Neck	No	No	No
Pancreatic	Yes	Yes	Yes
Skin (NMSC exc. SCC/BCC)	No	No	No
Small intestine	No	No	No
Thyroid gland	Yes	No	No
Unknown primary	*Yes*	*Yes*	*No^*

Highlighted cells – meets criteria for additional review in both sexes (or just one for sex specific cancers).
*Sex specific cancers (and female breast cancer) only required to meet eligibility for additional review in relevant sex.
^Cancer of unknown primary was excluded from additional review due to a lack of specificity regarding the cancers comprising this category.

Final cancers chosen

Public health implications are central to the Chief Health Officer's report so next we considered the cancers in terms of whether there are clearly established strategies for primary or secondary prevention (Table 8).

This allowed us to group the 12 cancers into 3 categories:

1. Clearly established strategies for primary or secondary prevention (defined as *known modifiable risk factors accounting for more than half of cases*[1] and/or *a viable population-based screening program exists*).
2. No clearly established strategies for primary or secondary prevention, but potential for targeted primary or secondary prevention in population sub-groups.
3. No clearly established strategies for primary or secondary prevention.

Primary prevention: preventing the initial development of a disease in people who are well. For example, immunization and reducing exposure to risk factors.

Secondary prevention: Early detection of an existing disease in people who have the disease, but have not yet developed clinical signs and symptoms of the illness, in order to reduce disease severity and complications [1].

1. Clearly established strategies for primary or secondary prevention

Breast cancer

Risk Factors

Risk factors for breast cancer include nuliparity, first birth after age 30, hormonal history, consumption of alcohol, use of certain contraceptive and menopausal therapies, ionizing radiation exposure, high calorie diets, lack of exercise, family history and genetics [3]. Physical activity has been associated with a 25-30% decrease in breast cancer risk [3]. Unfortunately no advice on the optimal type, dose and timing of physical activity is available. Additionally, the influence of broader dietary and lifestyle factors are also under active investigation, limiting advice to generalities such as reducing alcohol consumption, avoiding obesity (postmenopause) and diabetes [4].

Other protective factors for breast cancer include lactation (effect applies pre and post menopause), late menarche, early pregnancy, bearing children and early menopause [5]. The inverse corollaries of these factors are considered to increase breast cancer risk [5].

[1] Based on data from the UK, may differ in WA due to different prevalence of risk factors

Primary Prevention

Primary prevention is a possibility: in the UK it is estimated that 27% of breast cancers are attributable to potentially modifiable lifestyle and environmental risk factors [6].

Secondary Prevention

National population-based BreastScreen Australia mammography screening service is already an operational and comprehensive secondary prevention strategy [7].

Cervical cancer

Risk Factors

Infection from oncogenic types of human papillomavirus (HPV) have been demonstrated to develop precancerous lesions, a small proportion of which progress to invasive cervical cancer [8]. HPV is the most common sexually transmitted infection in the world, with the majority of sexually active individuals (both sexes) acquiring the virus at some point in their lifetime.

The primary risk factor for persistence and progression of HPV infection are immunodeficiency and the type of HPV, with HPV16 the most likely to persist and progress to high-grade lesions and cancer [8]. HPV16 and 18 are estimated to cause 70-80% of cervical cancers [9, 10]. Other risk factors for cervical cancer are generally considered to be weak in terms of absolute contribution (e.g. smoking tobacco) or simply represent escalations of the basic risk from sexual activity (e.g. number of sexual partner, age at first sexual intercourse, age at first birth, parity, and oral contraceptive use) [3].

Primary Prevention

In Australia there is an established primary prevention strategy: the national human papillomavirus (HPV) vaccination program [11]. Theoretically, 100% of cervical cancers are preventable by eliminating infection with HPV [12]. Vaccines currently available in Australia protect against high-risk HPV types 16 and 18, which are responsible for 70–80% of cervical cancers in Australia [10].

Secondary Prevention

The population-based National Cervical Screening Program is a currently operating secondary prevention strategy. Population-based screening for cervical cancer remains relevant for reducing the incidence and mortality from other HPV sub-types and in unvaccinated women [13].

Colorectal cancer

Risk Factors

Colorectal cancer is primarily considered a lifestyle disease with positive associations of incidence in countries with diets high in calories, animal fat and greater rates of sedentary behaviour [3]. Consumption of red and processed meat and alcohol, and body fat and abdominal fat are considered to increase the risk of colorectal cancer [5]. Physical activity, garlic, milk (calcium) and dietary fibre are associated with a protective effect against colorectal

cancer [5]. Limited evidence has also associated non-starchy vegetables, fruits, folate, fish, selenium and vitamin D with a protective effect [5].

Primary Prevention

Primary prevention is a possibility: it is estimated that 54% of all colorectal cancers are attributable to potentially modifiable lifestyle and environmental risk factors [6].

Secondary Prevention

The population-based National Bowel Cancer Screening Program has been running in Australia since August 2006 [14].

Lung cancer

Risk Factors

The most common cause of lung cancer is smoking tobacco, followed by occupational exposure to carcinogens. Potential workplace exposures include arsenic, metals, fibres, dusts, organic compounds, chemicals and radon [15-17]. Fresh fruit and vegetables have been associated with protecting against lung cancer [5]. There is some limited evidence suggesting red meat, processed meat, fat, butter and obesity are causes of lung cancer [5].

Primary Prevention

Established primary prevention is in place via tobacco control legislation and various health promotion programs. Tobacco smoke is estimated to account for 86% of all lung cancers in the UK [18].

Secondary Prevention

Screening using low dose CT screening is currently being trialled around the world [3].

Melanoma

Risk Factors

Melanoma is the most aggressive form of skin cancer subject to various factors for risk and progression [19, 20]. Factors such as age, sex, sun exposure and anatomical location all play a role in the risk and progression of melanoma [3]. Most melanomas derive from ultraviolet damage to sensitive skin (fair skin, skin with multiple freckles, or skin which does not tan), ultraviolet damage is particularly dangerous during childhood and adolescence [21].

Primary Prevention

Prevention of melanoma revolves around limiting ultraviolet radiation exposure, especially in the first 20 years of life. The adoption of sun avoidance behaviour and protection (appropriate clothing, shaded areas) are widely promoted, especially in Australia. Exposure can also be limited by avoiding the use of tanning beds. While there is no unequivocal evidence of sunscreen preventing melanomas, there is strong evidence regular sunscreen use can avoid some melanomas developing [3]. UV radiation exposure is estimated to account for 86% of all melanoma cases in the UK [6].

2. No clearly established strategies for primary or secondary prevention, but potential for targeted primary or secondary prevention in population sub-groups

Prostate Cancer

Risk Factors

Prostate cancer is dominated by acinar adenocarcinoma, other types of malignancies occur but are rare [3]. The major risk factors identified for prostate cancer are age and family history [3]. The likelihood of detection of prostate cancer is 40 times greater in men over 65 than younger men [3]. Approximately 25% of men diagnosed with prostatic carcinoma have a known family history [22, 23].

While preventable risk factors for prostate cancer are unconfirmed, diet and nutrition have been linked to this cancer. Fat (dietary and saturated), meat (red, processed, grilled), and milk/dairy products may increase risk of prostate cancer while tomatoes (lycopene), fish/omega-3 fatty acids, soy and cruciferous vegetables may decrease risk [3, 5, 24]. Other lifestyle factors, such as physical activity, tobacco smoking, and alcohol consumption have not been definitively linked to prostate cancer. Neither has obesity, although it may be associated with increased likelihood of developing more advanced and fatal prostate cancers [3, 24].

Primary Prevention

Promotion of healthy lifestyles, including physical activity, a diet rich in fruits (especially tomatoes), vegetables and fish may reduce the risk of prostate cancer [3].

Secondary Prevention

Digital rectal examination possibly in combination with PSA testing in men aged 40 and over, could be used as an early detection tool [25]. The European Randomized Study of Screening for Prostate Cancer observed a 20% reduction in relative risk of cancer-specific mortality but this carried through to an absolute reduction of prostate cancer-related deaths of only 0.71 per 1,000 [26]. In the USA the treatment of screen-detected tumours has raised concerns, especially for men with indolent disease and men over 65 years as aggressive treatment can result in a substantial decrease in quality of life (urinary, sexual and bowel dysfunction) with little demonstrated survival benefit [27].

A recent study has suggested improved diagnosis with an MRI biopsy method which reduces the number of biopsy samples required [28]. However, it is unlikely the biopsy method could play a primary role in any large scale screening program.

Stomach & Oesophageal Cancer

Risk Factors

Most stomach cancers are considered sporadic with only a familial clustering in about 10% of cases, an even smaller percentage (1-3%) have a hereditary syndrome characterised [3]. Stomach cancer usually follows a multistep and multifactorial progression from normal gastric mucosa through various stages of inflammation to dysplasia and carcinoma [29]. This

progression may span several years; however it does not necessarily apply to all stomach cancers.

Helicobacter pylori infection is considered the primary environmental risk factor contributing to stomach cancer and often described as necessary, but not sufficient alone, to cause almost all cases of stomach cancer [5, 30]. Lifestyle factors associated with increasing the risk of stomach cancer include high intakes of salt-preserved and/or smoked foods, low intakes of fresh fruit and vegetables, meat consumption (especially red, processed, grilled or barbequed meat) and tobacco smoking [5].

There are two types of oesophageal cancer with distinct risk factors. Squamous-cell carcinoma is predominately attributable to smoking and alcohol consumption while adenocarcinoma is attributed to gastro-oesophageal reflux and *Helicobacter pylori* [31].

Primary Prevention

Primary prevention of stomach & oesophageal cancer is broadly feasible through nutrition and lifestyle choices by increasing consumption of fresh fruit and vegetables and reducing consumption of salt (particularly for Japanese and Korean diets), processed and smoked meats [3, 5]. Helicobacter pylori eradication could also provide an opportunity to further reduce the incidence of stomach & oesophageal cancer, however there is limited published research of the feasibility for such an approach, in the general population or specific at-risk groups [3].

Secondary Prevention

Secondary prevention of stomach & oesophageal cancer via screening is not commonly practiced internationally, although the Republic of South Korea has included endoscopic screening for people aged 40 years or older in their National Cancer Screening Program since 1999 [3]. Japan also employs a screening program [32]. While the South Korean program has seen improvements in 5-year survival rates, the country also has the highest incidence of gastric cancer in the world [3]. The effectiveness of both screening programs has been criticised and may not be applicable given the differences in population risk profiles between Australia, South Korea and Japan [32].

3. No clearly established strategies for primary or secondary prevention

Uterine Cancer

Risk Factors

Uterine cancer is capable of spreading beyond the uterus, however most are contained at diagnosis and treated effectively with a hysterectomy [3]. Over 80% of cases are oestrogen-related, however those which are non-oestrogen-related are more often high-grade carcinomas associated with poor prognosis [3]. Risk factors for uterine cancer include early age at menarche, late age at menopause, nulliparity, hormone replacement therapy (HRT) use and obesity [3, 5].

Obesity is considered to be the most important risk factor and estimated to account for 40% of the worldwide incidence of uterine cancer [3, 5]. This elevated risk of uterine cancer from obesity is present pre and post menopause, although the mechanisms differ either side of menopause [3]. Due to the hormonal interactions that elevated insulin concentrations have with sex steroids, there is a strong association of uterine cancer with diabetes (type 1 and 2) [33].

The use of some oral contraceptives (those containing progestogen and oestrogens) is recognised to provide a long-term protective effect for uterine cancer [34]. HRT is associated with a 2-fold increase in risk for post-menopausal women [35]. Polycystic Ovary Syndrome has also been consistently linked with an increased risk of uterine cancer, as are women who have developed breast cancer [3].

Physical activity is associated with some protective effect, as is bearing children, and early menopause [5].

Primary Prevention

In the UK, 37% of uterine cancers are attributable to potentially modifiable lifestyle and environmental risk factors [6].

Pancreatic Cancer

Risk Factors

Pancreatic cancer is not one single disease but several distinct neoplasms, each with unique clinical and pathological features, captured by the broader topographical category. Risk factors for pancreatic cancers include older age, race (some groups have elevated risks), tobacco smoking (estimated to be responsible for 20% of pancreatic cancer)[36]. There are no established associations between pancreatic cancer and diet, however there is evidence that body fat increases risk, as does diabetes [5].

Primary Prevention

Efforts to reduce smoking and obesity are likely to be most effective in preventing pancreatic cancer. In the UK, 37% of pancreatic cancers are attributable to these potentially modifiable lifestyle risk factors [6].

Ovarian Cancer

Risk Factors

Over 95% of ovarian cancer is comprised of five different types of cancer: high-grade serious (70%), endometrioid (10%), clear cell (10%), mucinous (3%), and low-grade serious carcinomas(<5%) [3]. Ovarian carcinomas are the most lethal gynaecological malignancies [32]. The epidemiological factors, genetic risk factors, disease progression, disease spread, responses to treatment and prognosis, vary across all of these different malignancies.

Ovarian cancer most commonly affects nulliparous women and are least frequent in women with suppressed ovulation (by oral contraceptives or pregnancy) [3, 37]. Talc and asbestos have been connected to increased risk as have elevated gonadotropin levels [3, 37]. 10% of cancer in the ovaries has been attributed to family history, a risk that increases with two or more first degree relatives affected. Faulty BRCA1 and BRCA2 genes, originally associated with breast cancer, have a strong association with increased risk of ovarian cancer [38].

Primary Prevention

This class of cancers are difficult to prevent. Raising awareness of the risks for those with a family history or who are BRCA positive would be one avenue, as would be understanding of

the protective effect provided by oral contraception. In the UK, 21% of ovarian cancers are attributable to potentially modifiable lifestyle and environmental risk factors [6].

Lymphoma and Leukaemia

Risk Factors

Haematological and lymphoid malignancies are a heterogeneous group of cancers with diverse aetiologies, pathologies, genetic relationships and treatment strategies [3].

Primary Prevention

Given the lack of identified risk factors, prevention strategies are currently unavailable.

Table 4 summarises the proportion of cancers caused by modifiable risk factors for all 12 cancers identified and their known modifiable risk factors. Figure 1 summarises the process used to choose the cancers of focus in this report.

Table 4 Percentage of cancers attributable to modifiable lifestyle and environmental risk factors by cancer site and prevention strategy*

Risk Factor	Cancer											
	Category 1					Category 2			Category 3			
	Breast	**Cervical**	**Colorectal**	**Lung**	**Melanoma**	**Prostate**	**Stomach**	**Oesophagus**	**Uterine**	**Pancreatic**	**Ovarian**	**Leukaemia**
Tobacco		7.2	8.1	85.6			22.2	65.5		28.7	2.6	6.2
Alcohol	6.4		11.6					20.6				
Fruit & Vegetables				8.8			35.8	46.1				
Meat			21.1									
Fibre			12.2									
Salt							24.0					
Overweight and Obesity	8.7		13.0					21.7	33.7	12.2		
Physical Exercise	3.4		3.3						3.8			
Post-menopausal Hormones	3.2								1.2		0.7	
Infections		100.0	2.2				31.7					
Radiation - ionising	0.9		1.6	4.7			1.2	2.7				8.9
Radiation - UV					85.9							
Occupation	4.6	0.7		13.2			2.0	2.6			0.5	0.7
Reproduction (including breastfeeding)	3.1										17.6	
All of the Above	26.8	100	54.4	89.2	85.9		74.9	89.0	36.9	37.3	20.7	15.2
Current Primary Prevention		Vaccine		Yes	Yes							
Screening Program	**BreastScreen**	**NCSP**	**NBCSP**	Potential		Potential	Potential					

*% of cancers attributable to modifiable lifestyle and environmental risk factors using UK data, from Parkin, D.M., L. Boyd, and L.C. Walker, 16. The fraction of cancer attributable to lifestyle and environmental factors in the UK in 2010. Br J Cancer, 2011. 105(S2): p. S77-S81 [6].
Note: Lymphoma not included in this table due to absence in Parkin et al. 2011 study.
NCSP: National Cervical Screening Program
NBCSP: National Bowel Cancer Screening Program

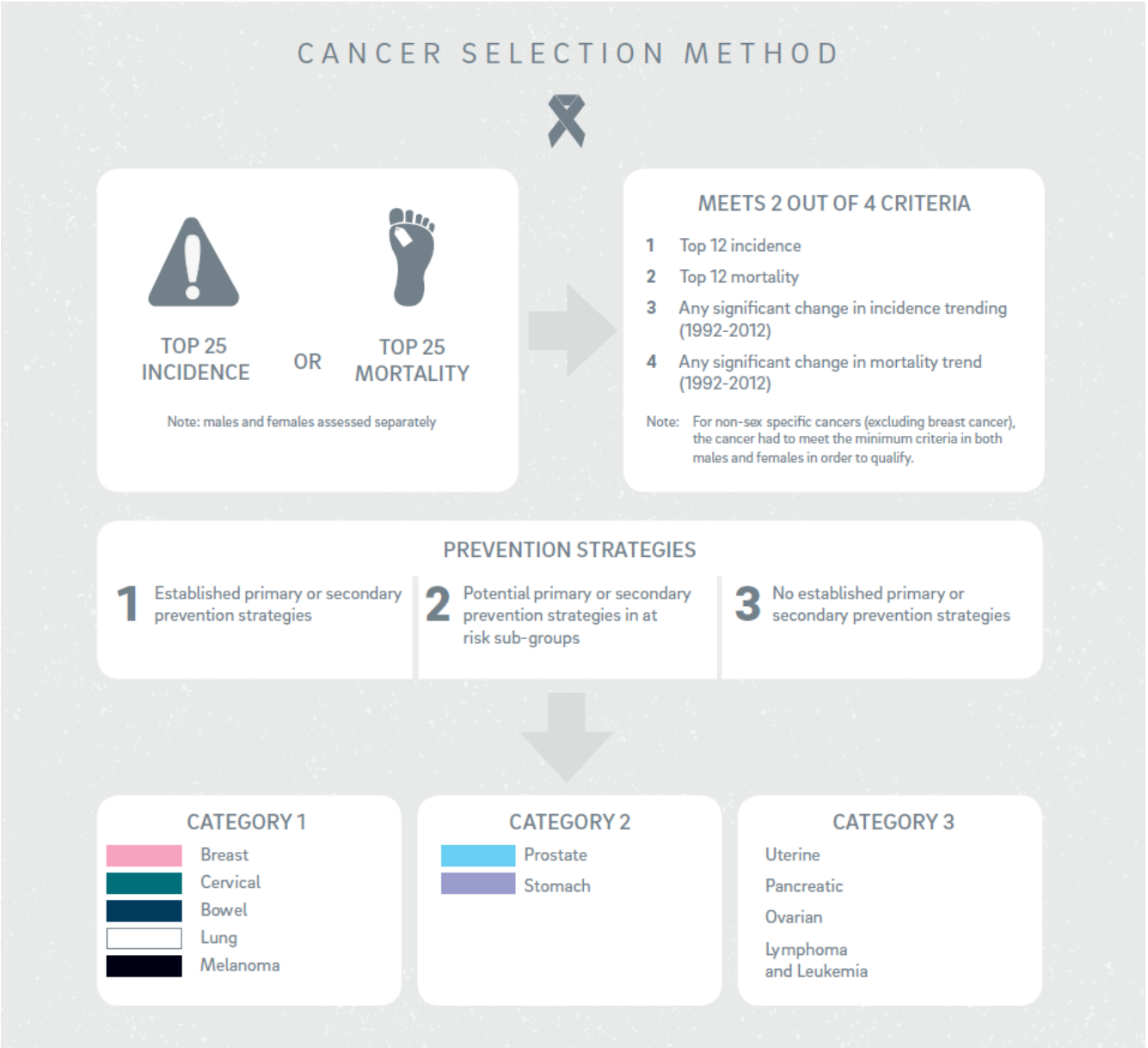

Figure 1 Summary of process for choosing cancers

Conclusion

The next Chief Health Officer's report will focus on the seven cancers comprising category 1 and 2 cancers, which include:

- Bowel cancer
- Breast cancer
- Cervical cancer
- Lung cancer
- Melanoma
- Oesophageal and stomach cancer
- Prostate cancer

This report will include a public consultation phase to incorporate the public's perspective on cancer prevention in WA. The consultation will be open from 4 February 2015 to 27 March 2015, and can be accessed from: yoursayoncancer.health.wa.gov.au.

The results of the consultation will be analysed and included in the next Chief Health Officer's report expected to be published in mid to late 2015.

For more detailed statistics on each of the seven cancers of focus, please refer to the document *2. The data behind 'Your Say On Cancer In WA'* available from: yoursayoncancer.health.wa.gov.au, or by request from the Epidemiology Branch of the WA Department of Health (epi@health.wa.gov.au).

References

1. Gordis, L., *Epidemiology. Fourth Edition*. 2008: Elsevier Health Sciences.
2. Hemminki, K., et al., *Survival in cancer of unknown primary site: population-based analysis by site and histology.* Annals of Oncology, 2012. **23**(7): p. 1854-1863.
3. International Agency for Research on Cancer, *World Cancer Report 2014*, World Health Organization, Editor. 2014, International Agency for Research on Cancer: Lyon.
4. Willett WC, et al., *Nongenetic factors in the causation of breast cancer*, in *Diseases of the Breast*, Harris JR, et al., Editors. 2010, Lippincott Williams & Wilkins: Philadelphia. p. 248-290.
5. World Cancer Research Fund/American Institute for Cancer Research, *Food, Nutrition, Physical Activity and the Prevention of Cancer: A Global Perspective*, American Institute for Cancer Research, Editor. 2007: Washington DC.
6. Parkin, D.M., L. Boyd, and L.C. Walker, *16. The fraction of cancer attributable to lifestyle and environmental factors in the UK in 2010.* Br J Cancer, 2011. **105**(S2): p. S77-S81.
7. AIHW, *BreastScreen Australia monitoring report 2010–2011*, in *Cancer Series No. 77*. 2013, Australian Institute of Health and Welfare: Canberra.
8. International Agency for Research on Cancer, *Biological agents. Volume 100 B. A review of human carcinogens.* IARC Monogr Eval Carcinog Risks Hum, 2012. **100**(Pt B): p. 1-441.
9. Guan, P., et al., *Human papillomavirus types in 115,789 HPV-positive women: A meta-analysis from cervical infection to cancer.* International Journal of Cancer, 2012. **131**(10): p. 2349-2359.
10. Brotherton, J.M.L., *How much cervical cancer in Australia is vaccine preventable? A meta-analysis.* Vaccine, 2008. **26**(2): p. 250-256.
11. Australian Government Department of Health and Ageing. *HPV School Vaccination Program*. 2013 18/12/2013 [cited 2014 2/7/2014]; Available from: http://hpv.health.gov.au/.
12. Parkin, D.M., *11. Cancers attributable to infection in the UK in 2010.* Br J Cancer, 2011. **105**(S2): p. S49-S56.
13. AIHW, *Cervical screening in Australia 2010–2011*, in *Cancer Series No. 76*. 2013, Australian Institute of Health and Welfare: Canberra.
14. AIHW, *National Bowel Cancer Screening Program Monitoring report July 2011–June 2012*, in *Cancer Series No. 75*. 2013, Australian Institute of Health and Welfare: Canberra.
15. Straif, K., et al., *A review of human carcinogens—Part C: metals, arsenic, dusts, and fibres.* The Lancet Oncology, 2009. **10**(5): p. 453-454.
16. Baan, R., et al., *A review of human carcinogens—Part F: Chemical agents and related occupations.* The Lancet Oncology, 2009. **10**(12): p. 1143-1144.
17. International Agency for Research on Cancer, *Radiation*. 2012, IARC Monogr Eval Carcinog Risks Hum. p. 1-437.
18. Parkin, D.M., *2. Tobacco-attributable cancer burden in the UK in 2010.* Br J Cancer, 2011. **105**(S2): p. S6-S13.
19. Siegel, R., D. Naishadham, and A. Jemal, *Cancer statistics, 2012.* CA: A Cancer Journal for Clinicians, 2012. **62**(1): p. 10-29.
20. Tas, F., *Metastatic Behavior in Melanoma: Timing, Pattern, Survival, and Influencing Factors.* Journal of Oncology, 2012. **2012**: p. 9.
21. Tsao, H. and A.J. Sober, *Ultraviolet radiation and malignant melanoma.* Clinics in

Dermatology, 1998. **16**(1): p. 67-73.
22. Johns, L.E. and R.S. Houlston, *A systematic review and meta-analysis of familial prostate cancer risk.* BJU International, 2003. **91**(9): p. 789-794.
23. Walsh, P.C. and A.W. Partin, *Family history facilitates the early diagnosis of prostate carcinoma.* Cancer, 1997. **80**(9): p. 1871-1874.
24. Giovannucci E, Platz EA, and Mucci L, *Epidemiology of Prostate Cancer*, in *Comprehensive Textbook of Genitouriinary Oncology*, Scardino PT, et al., Editors. 2011, Lippincott Williams & Wilkins: Baltimore. p. 1-17.
25. Murphy, D.G., et al., *The Melbourne Consensus Statement on Prostate Cancer Testing*, in *Prostate Cancer World Congress 7-10th August 2013*. 2013: Melbourne
26. Rove, K. and E. Crawford, *Randomized controlled screening trials for prostate cancer using prostate-specific antigen: a tale of contrasts.* World Journal of Urology, 2012. **30**(2): p. 137-142.
27. Sandhu, G.S. and G.L. Andriole, *Overdiagnosis of Prostate Cancer.* JNCI Monographs, 2012. **2012**(45): p. 146-151.
28. Pokorny, M.R., et al., *Prospective Study of Diagnostic Accuracy Comparing Prostate Cancer Detection by Transrectal Ultrasound–Guided Biopsy Versus Magnetic Resonance (MR) Imaging with Subsequent MR-guided Biopsy in Men Without Previous Prostate Biopsies.* European Urology, 2014(0).
29. Correa, P., *Human Gastric Carcinogenesis: A Multistep and Multifactorial Process—First American Cancer Society Award Lecture on Cancer Epidemiology and Prevention.* Cancer Research, 1992. **52**(24): p. 6735-6740.
30. International Agency for Research on Cancer, *Schistosomes, liver flukes and Helicobacter pylori.* IARC Monogr Eval Carcinog Risks Hum, 1994. **61**: p. 1-241.
31. Lao-Sirieix, P., C. Caldas, and R.C. Fitzgerald, *Genetic predisposition to gastro-oesophageal cancer.* Current Opinion in Genetics & Development, 2010. **20**(3): p. 210-217.
32. Talley, N.J., *Is it time to screen and treat H pylori to prevent gastric cancer?* The Lancet, 2008. **372**(9636): p. 350-352.
33. Kaaks, R., A. Lukanova, and M.S. Kurzer, *Obesity, endogenous hormones, and endometrial cancer risk: a synthetic review.* Cancer Epidemiol Biomarkers Prev, 2002. **11**(12): p. 1531-43.
34. Cogliano, V., et al., *Carcinogenicity of combined oestrogen-progestagen contraceptives and menopausal treatment.* The Lancet Oncology, 2005. **6**(8): p. 552-553.
35. Million Women Study Collaborators, *Endometrial cancer and hormone-replacement therapy in the Million Women Study.* The Lancet, 2005. **365**(9470): p. 1543-1551.
36. Lowenfels, A.B. and P. Maisonneuve, *Epidemiology and risk factors for pancreatic cancer.* Best Practice & Research Clinical Gastroenterology, 2006. **20**(2): p. 197-209.
37. La Vecchia, C., *Epidemiology of ovarian cancer: a summary review.* European Journal of Cancer Prevention, 2001. **10**(2): p. 125-129.
38. Risch, H.A., et al., *Population BRCA1 and BRCA2 Mutation Frequencies and Cancer Penetrances: A Kin–Cohort Study in Ontario, Canada.* Journal of the National Cancer Institute, 2006. **98**(23): p. 1694-1706.

This document can be made available in alternative formats on request for a person with a disability.