Ontario
Cancer Care Ontario
Action Cancer Ontario

Cancer Risk Factors in Ontario

Diet

DIET

Risk factor/exposure	Cancer	Direction of association	Magnitude of risk*	Strength of evidence[a]
Red meat	Colon and rectum	↑	1.17[b]	Convincing
Processed meat	Colon and rectum	↑	1.18[b]	Convincing
Salt and salted/ salty foods[†]	Stomach	↑	…	Probable
Dietary fibre	Colon and rectum	↓	0.90[b]	Convincing
Vegetables and fruit[‡]	Oral cavity, pharynx, larynx	↓	…	Probable
	Esophagus	↓	…	
	Stomach	↓	…	
	Lung[§]	↓	…	

Sources: [a]WCRF/AICR, 2007; [b]WCRF/AICR, 2011

* Relative risk (RR) estimate for: every 100 g/day increase in red meat consumption; every 50 g/day increase in processed meat consumption; and every 10 g/day increase in dietary fibre intake.
…Magnitude of risk not shown in table if strength of evidence is "probable" or "limited."
[†] Salt refers to total salt consumption (usually measured in g/day), from processed foods, as well as salt added in cooking and at the table. Salted/salty foods refers to the consumption of foods containing salt, including processed and salt-preserved foods.
[‡] Vegetables refer to non-starchy vegetables and allium vegetables (e.g., onions, garlic, leeks), but exclude roots and tubers, such as potatoes, sweet potatoes and yams.
[§] Probable evidence supports only fruit (not vegetables) as protective for lung cancer.

Introduction: food or nutrients?

Dietary factors are thought to account for a large proportion of certain cancers. Individual diets are dynamic and complex, consisting of a large number of factors, including micronutrients (e.g., vitamins and minerals), macronutrients (e.g., proteins, fats, carbohydrates), whole food items and processed foods, present in countless combinations.

This evidence summary takes a food-based approach, which is the most useful from a prevention standpoint because people consume whole foods rather than individual nutrients. Although convincing or probable evidence from good-quality randomized control trials supports supplementation of some micronutrients (e.g., calcium, selenium) for the prevention of certain cancers, it is difficult to determine whether any given constituent of a particular food is causally associated with a decreased or increased cancer risk, or is simply a marker for some other constituent of the food or of the whole food itself.

A food-based approach is still faced with challenges in determining whether a particular food is a causal risk or protective factor for a given cancer, since individuals eat many different kinds of foods, more than one of which can contain similar types of dietary constituents. Dietary fibre, for example, is found in cereals and grains, as well as in vegetables and fruit. Similarly, salt or sodium is found in processed meats, but also occurs in non-meat processed food items.

RED MEAT AND PROCESSED MEAT

Background

» Red meat comes from animals with more red than white muscle fibres and includes beef, pork, lamb and goat. Processed meat generally refers to meats preserved by smoking, curing or salting, or the addition of chemical preservatives (e.g., ham, bacon, sausages, hot dogs), although there is no standard processed meat definition.[24]

» Consumption of red and processed meat is generally highest in high-income countries, such as the United States and Canada.[24]

» Since processed meat is generally red meat, it is difficult to disentangle the cancer risk associated with each of these factors in epidemiologic studies.[24]

- Red meat and processed meat consumption are both convincing causes of colorectal cancer.[24,38]
- Substantial evidence from prospective studies demonstrates a positive dose-response relationship between colorectal cancer risk and both red and processed meat consumption. A World Cancer Research Fund/American Institute for Cancer Research (WCRF/AICR) meta-analysis estimated a 17% increase in risk for every 100 g/day increase in red meat consumption and an 18% increased risk of colorectal cancer for every 50 g/day increase in processed meat consumption.[38] Results of another meta-analysis suggests that elevated risk associated with red meat consumption levels off above 140 g/day.[39]
- The lowest intake of red meat consumption associated with increased risk of colorectal cancer remains unclear.[24]
- The associations with red and processed meat appear to be of similar magnitude for both colon and rectal cancer, although results have more often reached statistical significance for colon cancer.[39] Some studies show a positive association with red meat consumption for men, but not for women.
- Red and processed meat may increase colorectal cancer risk through several plausible biologic mechanisms:[38]
  - Heme iron in red meat can promote the formation of potentially carcinogenic *N-nitroso* compounds and other harmful substances in response to oxidative degradation of fats.
  - Heterocyclic amines (HCAs) and polycyclic aromatic hydrocarbons (PAHs) from cooking red meat at high temperatures can promote colorectal cancer in people with a genetic predisposition.
  - Processed meats frequently contain nitrates and/or nitrite-fortified salts, which can promote carcinogenesis in the stomach via the formation of *N-nitroso* compounds.

SALT AND SALTED/SALTY FOODS

Background

» "Salt," which commonly refers to sodium chloride, is used to preserve and enhance the flavour of many foods.[24]

» Although the use of salt as a preservative has become less common with the increased availability of refrigeration, several traditional diets still include large amounts of salt-preserved foods, including salted meat and fish. Processed foods are the main source of dietary salt intake in most industrialized countries, with only a small amount added during cooking or at the table.[24]

» Assessing salt intake in epidemiologic studies is difficult. Although measuring the amount of sodium excreted in the urine provides the most reliable estimate,[24] most studies rely on self-reported intake of salty, salted and salt-preserved foods.

- The WCRF/AICR concluded that salt and salted/salty foods probably cause stomach cancer.[24]
- Stomach cancer incidence rates are highest in areas of the world where traditional diets are high in salt (e.g., parts of Asia, Latin America).[40]

- Cohort studies show a positive dose-response relationship between total salt intake (from all sources, including processed foods, salt-preserved foods, and salt added during cooking and at the table) and stomach cancer, and several case-control studies have shown increased risk of stomach cancer with high levels of salt intake.[24] The WCRF/AICR meta-analysis of two cohort studies indicates an 8% increase in stomach cancer risk for every 1 g/day intake of salt.[24]
- Intake of salty/salted foods has also been associated with increased risk of stomach cancer. Case-control studies show a positive dose-response, with a 5.2-fold increased risk of stomach cancer for each additional serving of salty/salted food per day.[24]
- Evidence suggests that salt and salty/salted foods may interact synergistically with *Helicobacter pylori* infection, an established risk factor for stomach cancer, to promote stomach cancer.[41]
- High salt intake can damage the stomach lining, which may promote the effect of gastric and food-derived carcinogens and cause inflammatory responses that increase epithelial cell proliferation.[40]

DIETARY FIBRE

Background

» Dietary fibre can be defined as the components of plant cell walls that cannot be digested in the small intestine.[24]

» Naturally occurring dietary fibre is derived from plant foods, including pulses (legumes), cereals (grains) that have undergone minimal processing, vegetables and fruit.

- There is now convincing evidence that foods containing dietary fibre have a protective effect against the risk of cancer of the colon and rectum.[38]
- Colorectal cancer risk shows an inverse dose-response to dietary fibre intake, with a 10% decreased risk of colorectal cancer for every 10 g/day intake of total dietary fibre.[38] Similar findings have been observed for colon and rectal cancer, although the results for rectal cancer have generally not reached statistical significance.[38]
- Colorectal cancer risk is reduced in response to total dietary fibre intake and to fibre derived from cereals (grains) and whole grains. Fibre from other sources (e.g., vegetables and fruit) also appears to be protective, although results are not statistically significant.[38]
- Several biologic mechanisms have been proposed for the protective effect of fibre, including diluting fecal carcinogens, reducing transit time through the colon, increasing stool weight, altering bile acid metabolism, reducing colonic pH and/or increasing the production of short-chain fatty acids, which may induce apoptosis following fermentation of fibre by the gut flora.[42]

VEGETABLE AND FRUIT CONSUMPTION

Background

» Vegetables can be classified as starchy (e.g., potatoes, yams and other root vegetables) or non-starchy (e.g., green leafy, cruciferous and allium vegetables). Fruit are the edible seed-containing part of a plant (e.g., apples, bananas, berries).[24]

» Vegetables and fruit are rich sources of vitamins, minerals, dietary fibre, and other micronutrients and bioactive compounds, such as phytochemicals.

» Epidemiologic studies of vegetable and fruit consumption generally rely on self-reported intake and differ in the definition and groupings of vegetables and fruit.

- Consumption of non-starchy vegetables and of fruit has been classified by the WCRF/AICR as probably protective against of cancers of the oral cavity, pharynx, larynx, esophagus and stomach. Fruit consumption also probably protects against lung cancer.[24]
- Risk reductions of 30%–50% have been estimated for cancers of the mouth, pharynx and larynx with higher versus lower intakes of vegetables and fruit.[24,43,44]
- Evidence suggests an inverse dose-response between vegetable consumption and cancer of the oral cavity, pharynx, larynx and esophagus (raw vegetable consumption) and between fruit consumption and cancers of the esophagus, stomach and lung. Case-control studies but not cohort studies support inverse dose-response relationships with vegetable consumption and stomach cancer and with fruit consumption and cancer of the mouth, pharynx and larynx.[24]
- It is unclear whether all vegetables and fruit confer a protective effect, although it is likely that a few vegetables or fruits have an important effect on certain cancers (e.g., foods containing lycopene probably protect against prostate cancer; foods containing carotenoids protect against cancers of the mouth, pharynx, larynx and lung cancer; and foods containing vitamin C protect against esophageal cancer).[24]
- Vegetables and fruit may confer a protective effect through several generic and cancer-specific biologic mechanisms by, for example, preventing nutrient deficiencies.[45] Fruit and vegetables also contain specific potentially cancer-preventive components, including antioxidants (e.g., carotenoids, vitamin C, lycopene), dietary fibre and phytochemicals (e.g., phytoestrogens). These substances may reduce cancer risk through antioxidant activity, modulation of detoxification enzymes, stimulation of the immune system, antiproliferative activities, and/or modulation of steroid hormone concentration and hormone metabolism.[24]
- The low energy density and high fibre content of vegetables and fruit may indirectly protect against certain cancers by preventing weight gain, overweight and obesity.[24]