Lifestyle Recommendations for Breast and Endometrial Cancer Prevention

Presented by Liz Gorecki, RD, CSO, CD, CNSC
Women’s Health Conference
April 7th, 2017
Objectives

• Discuss evidence from the WCRF/AICR breast and endometrial cancer reports.

• Translate evidence into practice with prevention recommendations for breast and endometrial cancer.

• Review WCRF/AICR cancer prevention guidelines.
### Estimated Cases of US Cancers Preventable per Year by Diet, Activity, and Weight Management

<table>
<thead>
<tr>
<th>Cancer Type</th>
<th>New Cases</th>
<th>Percentage Prevented</th>
<th>*Number of Cases Prevented</th>
</tr>
</thead>
<tbody>
<tr>
<td>Breast, female</td>
<td>246,660</td>
<td>33%</td>
<td>81,398</td>
</tr>
<tr>
<td>Colorectal</td>
<td>134,490</td>
<td>47%</td>
<td>63,200</td>
</tr>
<tr>
<td>Endometrial</td>
<td>60,050</td>
<td>59%</td>
<td>35,430</td>
</tr>
<tr>
<td>Esophageal</td>
<td>16,910</td>
<td>33%</td>
<td>5,580</td>
</tr>
<tr>
<td>Gallbladder</td>
<td>11,420</td>
<td>22%</td>
<td>2,512</td>
</tr>
<tr>
<td>Kidney</td>
<td>62,700</td>
<td>24%</td>
<td>15,048</td>
</tr>
<tr>
<td>Liver</td>
<td>39,230</td>
<td>30%</td>
<td>11,769</td>
</tr>
<tr>
<td>Lung</td>
<td>224,390</td>
<td>36%</td>
<td>80,780</td>
</tr>
<tr>
<td>Mouth, Pharyngeal &amp; Laryngeal</td>
<td>42,760</td>
<td>63%</td>
<td>26,939</td>
</tr>
<tr>
<td>Ovarian</td>
<td>22,280</td>
<td>5%</td>
<td>1,114</td>
</tr>
<tr>
<td>Pancreatic</td>
<td>53,070</td>
<td>19%</td>
<td>10,083</td>
</tr>
<tr>
<td>Prostate (advanced)</td>
<td>23,516</td>
<td>11%</td>
<td>2,587</td>
</tr>
<tr>
<td>Stomach</td>
<td>26,370</td>
<td>15%</td>
<td>3,956</td>
</tr>
</tbody>
</table>

**TOTAL PREVENTABLE (rounded)** 340,400

*Estimated and rounded, based on: AICR/WRCF, Policy and Action for Cancer Prevention 2009; Continuous Update Project reports; American Cancer Society, Cancer Facts & Figures 2016.
Breast Cancer
Breast Cancer Trends, Incidence, and Survival

• Most common cancer in women
• Overall risk doubles each decade until menopause, when the increase slows down or remains stable.
  – However, breast cancer is more common after menopause.
• Predominantly disease of high-income countries.
  – Overall rates are 3 x higher in high-income countries compared to low- and middle-income countries.
• Often detected at relatively early stage.
  – Survival rates >90% to <50%
  – Survival dependent on characteristics of tumor + its size/spread + availability of treatment.
Breast Cancer
Development/Progression

• Hormones play an important role in breast cancer progression
  – Modulate structure and growth of epithelial tumor cells
    • ER/PR+ vs ER/PR– breast cancers
  – Early menarche, late menopause, not bearing children, late (over 30) pregnancy increase risk.
• Family history associate with 2-3 fold higher risk
  – BRCA1, BRAC2, p53 mutations
  – Rare, account for 2-5% of cases
• Radiation
  – Exposure from medical treatment (ie x-rays), especially during puberty increases risk.
• Medication
  – Hormone replacement therapy and oral contraceptives
  – Increased risk disappears after cessation
WCF/AICR
Second Expert Report

  – Research relationship between diet, physical activity, and weight management on cancer risk
  – Interpret accumulated scientific literature
• Started Continuous Update Project (CUP) to keep recommendations current.
Continuous Update Project
The process we use to analyse worldwide research

- Imperial College London collates the worldwide evidence
- Peer reviewers
- CUP Expert Panel (scientists from around the world)
- World Cancer Research Fund network

SECOND EXPERT REPORT 2007 CONTINUOUS UPDATE PROJECT TO DATE
WCRF/AICR Breast Cancer 2010 Report

- Part of the Continuous Update Project
- Randomized control trials, cohort and case-controlled studies
- Identified total of 954 publications

Food, Nutrition, Physical Activity and Breast Cancer (Premenopause) 2010

<table>
<thead>
<tr>
<th></th>
<th>Decreases Risk</th>
<th>Increases Risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>Convincing</td>
<td>Lactation</td>
<td>Alcoholic drinks</td>
</tr>
<tr>
<td>Probable</td>
<td>Body fatness</td>
<td>Adult attained height, Greater birth weight</td>
</tr>
<tr>
<td>Substantial effect on risk unlikely</td>
<td>None identified</td>
<td></td>
</tr>
</tbody>
</table>

Food, Nutrition, Physical Activity and Breast Cancer (Postmenopause) 2010

<table>
<thead>
<tr>
<th></th>
<th>Decreases Risk</th>
<th>Increases Risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>Convincing</td>
<td>Lactation</td>
<td>Alcoholic drinks, Body fatness, Adult attained height</td>
</tr>
<tr>
<td>Probable</td>
<td>Physical activity</td>
<td>Abdominal fatness, Adult weight gain</td>
</tr>
<tr>
<td>Substantial effect on risk unlikely</td>
<td>None identified</td>
<td></td>
</tr>
</tbody>
</table>

1 Adult attained height is unlikely directly to modify the risk of cancer. It is a marker for genetic, environmental, hormonal, and also nutritional factors affecting growth during the period from preconception to completion of linear growth (see chapter 6.2.13 – Second Expert Report).

2 Physical activity of all types: occupational, household, transport and recreational.
Alcoholic Drinks

- Meta-analysis of cohort studies showed the following effects per 10 g ethanol intake
  - 10% increased risk for all-age breast cancer
  - 9% increased risk for premenopausal breast cancer
  - 8% increased risk for postmenopausal breast cancer
- Meta-analysis of case-control data showed
  - 5% increased risk per 5 drinks/week
  - 6% increased risk per 10 g ethanol/day
  - Menopausal status did not significantly alter the association
- Meta-analysis* of 3 cohort and 7 case-control studies assessed alcohol intake and ER/PR defined breast cancer
  - Increase in alcohol consumption of 10 g ethanol per day was associated with sig increased risk for all ER+ (12%), all ER- (7%), ER+PR+ (11%), and ER+PR- (15%), but not ER-PR-.

What’s the connection?

- Reactive metabolites of alcohol (acetaldehyde) may be carcinogenic.
- Production of prostaglandins, lipid peroxidation, and generation of free radical oxygen species.
- Alcohol also acts as a solvent, enhancing penetration of carcinogens into cells.
- High consumers of alcohol may have diets deficient in essential nutrients, making tissues susceptible to carcinogenesis.
- Alcohol interferes with estrogen metabolism and action in multiple ways, influencing hormone levels and estrogen receptors.
Conclusion

• Dose-response relationship is apparent.
• Evidence is convincing that alcoholic drinks are cause of pre and postmenopausal breast cancer.
• No threshold was identified.
Lactation

• Meta-analysis for case-control data showed 2% decreased risk per 5 months of breastfeeding.
• Cohort data showed a non-significant decreased risk.
• Pooled analysis (47 epidemiological studies in 30 countries) showed 4.3 % (statistically significant) decreased risk of breast cancer for each 12 months of breastfeeding.
• Meta-analysis (5 population-based case-control studies) found statistically significantly lower risk of both ER+/PR+ (22%) and ER-/PR- (26%) breast cancers for more than 6 months breastfeeding compared to never breastfeeding.
What’s the connection?

- Lactation is associated with increased differentiation of breast cells and with lower exposure to endogenous sex hormones during amenorrhea accompanying lactation.
- Strong exfoliation of breast tissue during lactation, and the massive epithelial apoptosis at the end of lactation, could decrease risk by elimination of cells with potential DNA damage.
Conclusion

• There is abundant epidemiological evidence from both cohort and case-control studies, which is consistent and shows a dose-response relationship.
• The evidence that lactation protects against both premenopausal and postmenopausal breast cancer is convincing.
Physical Activity

• Meta-analysis of case-control studies showed 10% decreased risk per 7 MET-hours activity (vigorous activity) per week.
• Premenopause
  – Data was inconsistent for cohort studies on physical activity.
  – Most case-control studies showed evidence of decreased risk.
• Postmenopause
  – Nearly all cohort studies showed decreased risk with increased physical activity.
  – Meta-analysis of cohort and case-control data both showed 3% decreased risk per 7 MET-hours activity per week.
What’s the connection?

• Sustained moderate physical activity raises the metabolic rate and increases maximal oxygen uptake.
  – Regular periods of such activity increase the body’s metabolic efficiency and capacity, and reduce blood pressure and insulin resistance.
• It decreases level of estrogen and androgens in postmenopausal women.
• Some trials have also shown decreases in circulating estrogens, increased menstrual cycle length, and decreased ovulation in premenopausal women with a high level of physical activity.
Conclusion

• Premenopause
  – Good amount of evidence from prospective studies, but it is **inconsistent**.
  – There is limited evidence suggesting that physical activity protects against premenopausal breast cancer.

• Postmenopause
  – There is plenty of evidence from prospective studies showing lower risk of postmenopausal breast cancer with higher levels of physical activity, with a dose-response relationship.
  – Little evidence on frequency, duration, or intensity of activity.
  – Physical activity **probably** protects against postmenopausal breast cancer.
Body Fatness

- Premenopause
  - Meta-analysis of cohort studies showed 7-15% decreased risk per 5 kg/m²
  - Meta-analysis of case-control studies showed 8% decreased risk per 5 kg/m²
  - Pooled analysis of four cohort studies (723 cases followed for up to 11 years) showed 14% decreased risk for 5 kg/m².
  - Meta-analysis of 20 cohort studies reported 8% decreased risk per 5 kg/m².
Body Fatness

- Postmenopause
  - Meta-analysis of cohort studies showed 8-13% increased risk per 5 kg/m².
  - Pooled analysis of 7 cohort studies (3208 cases) followed for up to 11 years showed a 9% increased risk per 5 kg/m².
  - Meta-analysis of 31 cohort studies reports a 12% increased risk per 5 kg/m².
What’s the connection?

• Premenopause
  – No single well-established mechanism.

• Postmenopause
  – Body fatness directly affects levels of many circulating hormones (insulin, IL-GFs, estrogens) that create an environment that encourages carcinogenesis and discourages apoptosis.
  – It also stimulates the body’s inflammatory response, which may contribute to the initiation and progression of several cancers.
  – Adjusting for serum levels of estradiol diminishes or destroys the association with BMI, suggesting that hormones are a predominant mechanism.
Conclusion

• Premenopause
  – There is a substantial amount of consistent epidemiological evidence with a dose-response relationship, but the mechanistic evidence is speculative.
  – Greater body fatness probably protects against premenopausal breast cancer.

• Postmenopause
  – There is abundant and consistent epidemiological evidence and clear dose-response relationship with evidence for mechanisms.
  – The evidence that great body fatness is a cause of postmenopausal breast cancer is convincing.
Abdominal Fatness

• Meta-analysis of cohort studies on weight circumference
  – Studies that did not adjust for BMI showed 7% increased risk for 8 cm in waist circumference
  – Those that did showed 4% increased risk.
• Meta-analysis of cohort studies on waist to hip ratio
  – Studies that did not adjust for BMI showed 9% increased risk per 0.1 increment in waist to hip ratio
  – Those that did adjust for BMI showed a non-significant increased risk
What’s the connection?

• General mechanisms which abdominal fatness could plausibly cause cancer
  – Increased levels of circulating estrogens
  – Decreased insulin sensitivity
• Associated with abdominal fatness independent of overall body fatness.
Conclusion

• There is substantial amount of epidemiological evidence, but some inconsistency.
• Abdominal fatness is a probable cause of postmenopausal breast cancer.
Adult Weight Gain

- Meta-analyses for cohort studies showed 3% increased risk per 5 kg gained.
- 5% increased risk per 5 kg gain for case-control studies.
What’s the Connection?

• Body fatness directly affects levels of many circulating hormones (insulin, IL-GF, estrogens) creating an environment that encourages carcinogenesis and discourages apoptosis.
• It also stimulates the body’s inflammatory response, which may contribute to the initiation and progression of cancers.
Conclusion

• There is ample, consistent epidemiological evidence and a dose-response relationship was apparent.
• Adult weight gain is a probable cause of postmenopausal breast cancer.
Total Fat Intake

(postmenopause)

- Meta-analyses showed non-significant increased risk for cohort studies
- 11% increased risk per 20 g per day for case-control studies
- Pooled analysis of cohort studies of more than 7300 cases of breast cancer showed overall non-significant decreased risk with increased fat intake
- For energy from fat most cohort studies reported decreased risk with increasing % energy from fat.
  - One reported a statistically increased risk.
Total Fat Intake
(postmenopause)

- The Women’s Health Initiative Dietary Modification Randomized Control Trial*
  - 655 cases of postmenopausal breast cancer
  - Relative risk of 0.91 for intervention and comparison group after 8.1 years.
  - Design: reduce fat intake to 20% and increase fruit and veggie intake to 5 servings per day and increase grains to at least 6 servings per day

What’s the Connection?

- Higher endogenous estrogen levels after menopause are known cause of breast cancer.
- Dietary fat may also increase endogenous estrogen production.
Conclusion

• Evidence from prospective epidemiological studies of different type shows inconsistent effects, while case-control studies show a significant positive association.

• There is limited evidence suggesting that consumption of total fat is a cause of postmenopausal breast cancer.
Endometrial Cancer
Endometrial Cancer Trends, Incidence, and Survival

- 6th most common cancer in women
- Predominantly disease of high-income countries.
  - Highest incidence in North America and Central/Eastern Europe.
  - Lowest incidence in Middle/Western Africa
- In USA, rates are highest in white women, although mortality rates are highest in black women.
- Risk increases with age. Most diagnosed after menopause.
- Often detected at relatively early stage.
  - Survival rates 69%
  - Lower in middle- to low-income countries
Endometrial Cancer
Development/Progression

• Type 1 tumor
  – Estrogen driven
  – Account for 80% of endometrial cancers
  – Favorable prognosis
• Type 2 tumor
  – Less common, 10% of endometrial cancers
  – More associated with endometrial atrophy
  – Tend to metastasize and have less favorable prognosis
• PCOS and insulin sensitivity (or resistance), which are both components of metabolic syndrome, may play a role in development of endometrial cancer
  – Through hormone disruption?
Endometrial Cancer
Development/Progression

• Life events
  – Not bearing children increases risk
  – Late menopause increases risk

• Medications
  – Oral contraceptives protect against endometrial cancer
  – Estrogen-only hormone replacement therapy is a cause
  – Tamoxifen (hormonal therapy used for breast cancer) can also cause endometrial cancer
**WCRF/AICR Endometrial Cancer 2013 Report**

<table>
<thead>
<tr>
<th>FOOD, NUTRITION, PHYSICAL ACTIVITY AND ENDOMETRIAL CANCER 2013</th>
</tr>
</thead>
<tbody>
<tr>
<td>DECREASES RISK</td>
</tr>
<tr>
<td>Convincing</td>
</tr>
<tr>
<td>Probable</td>
</tr>
<tr>
<td></td>
</tr>
</tbody>
</table>

1. The Panel interpreted BMI (including BMI at age 18-25 years), measures of abdominal girth, and adult weight gain as interrelated aspects of body fatness as well as fat distribution.
2. Physical activity of all types: occupational, household, transport and recreational.
3. The effect is found in both caffeinated and decaffeinated coffee and cannot be attributed to caffeine.

- Part of the Continuous Update Project Report
- Identified total of 159 publications
Glycemic Load

- Meta-analysis of 6 studies (n = 3869) showed 15% increased risk per 50 units (g) glucose per day.
- 3 other meta-analyses of cohort studies
  - All finding significant positive association when comparing the highest glycemic load group to the lowest
  - One also reported significant positive association per 50 units (g) glucose
Carbohydrate

• 4 of 5 studies reported an increased risk for the highest intake group compared to the lowest.
  – Other study reported a non-significant inverse association
• Meta-analyses (n = 2629) conducted per 100 g carbohydrate intake per day
  – 18% increased risk
What’s the connection?

• Long-term consumption of a high glycemic load diet results in hyperinsulinemia, which in turn increased the bioavailability of insulin-like growth factor 1 (IGF-1) and directly promotes cell growth, reduces cell death and stimulates cell division in endometrial cancer cell lines.

• Insulin and IGF-1 are also power negative regulators of sex hormone-binding globulin synthesis in vitro and may therefore stimulate endometrial cancer.

• High glycemic load diets may influence the risk of endometrial cancer by increasing oxidative stress.
Conclusion

• There is substantial amount of generally consistent evidence from cohort studies.
• Glycemic load is a probable cause of endometrial cancer.
Coffee

• Dose-response meta-analysis (7 studies, n = 3571) for coffee and endometrial cancer
  – 7% decreased risk per 1 cup per day
  – Statistically significant
• 2 other meta-analyses of cohort studies
  – Found statistically significant decreased risk when comparing highest coffee drinkers to lowest.
  – One of the studies also reported significant decreased risk per 1 cup per day.
• Dose-response meta-analysis for decaf coffee and endometrial cancer (3 studies, n = 2585)
  – 8% decreased risk per 1 cup per day
  – Statistically significant
What’s the connection?

• Several bioactive compounds have strong antioxidant properties and can prevent oxidative DNA damage, improve insulin sensitivity, and inhibit glucose absorption in the intestine.

• Coffee consumption has been demonstrated to improve insulin sensitivity. Caffeinated and decaf coffee are associated with reduced insulin levels, especially in overweight women.
  – Hyperinsulinemia has been positively associated with endometrial cancer development.
  – Endometrial cancer cell lines express high affinity insulin receptors, which shows direct effect of insulin on the growth of endometrial cancer cells.
What’s the connection?

• Hyperinsulinemia may indirectly impact development of endometrial cancer
  – Up-regulation of free IGF-1 or suppression of sex hormone binding globulin (SHBG), which elevates estradiol activity.
    • Coffee drinking has been associated with higher SHBG levels, which reduce risk through decreased estradiol exposure
  – Caffeine seems to up-regulate hepatic expression of CYP1A2 and CYP3A4 which leads to
    • Increase in clearance of estradiol
    • Stimulate synthesis of estrogen metabolites that inhibit estradiol-mediated carcinogenesis on endometrial cells.

• High coffee consumption (including decaf) has been associated with lower circulating levels of C-peptide and higher levels of adiponectin.
Conclusion

• There is substantial, consistent epidemiological evidence and a dose-response relationship.
• Coffee probably protects against endometrial cancer.
Physical Activity

- Recreational physical activity
  - 8 of 9 studies showed decreased risk of endometrial cancer when comparing the highest vs lowest levels of activity

- Occupational physical activity
  - 5 studies showed decreased risk when comparing highest vs lowest levels of activity

- Walking/biking (mainly for transportation)
  - 5 studies showed a decreased risk when comparing highest vs lowest levels of activity
What’s the connection?

• Sustained moderate physical activity raises the metabolic rate and increases maximal oxygen uptake.
  – Regular periods of such activity increase body’s metabolic efficiency and capacity
  – Reduces circulating insulin levels and insulin resistance.
• Physical activity reduces serum estradiol levels and increased levels of SHBG (binding protein for estradiol).
Conclusion

• There is generally consistent evidence showing lower risk of endometrial cancer with higher levels of physical activity.
• Physical activity probably protects against endometrial cancer.
Body Fatness

- Body Mass Index (BMI)
- BMI at age 18-25 years
- Weight gain (including increase in BMI)
- Waist circumference
- Waist-to-hip ratio
BMI

- 26 studies included in meta-analysis for BMI and endometrial cancer (n = 18,717)
  - Found 50% increased risk per 5 BMI units
  - Evidence of non-linear dose-response relationship with a steeper increase in risk at higher BMI levels

- BMI at age 18-25
  - 7 studies in dose-response meta-analysis for BMI at age 18-25 years and endometrial cancer (n = 3476)
    - All studies reported increased risk
    - 42% increased risk per 5 units BMI
Weight Gain

- 5 studies in dose-response meta-analysis for weight change and endometrial cancer (n = 1971).
  - All studies reported in the direction of increased risk
  - 16% increased risk per 5 kg weight gain
Waist Circumference

• 4 studies in dose-response meta-analysis for waist circumference and endometrial cancer (n = 1641).
  – 13% increased risk per 5 cm
  – Non-linear dose-response relationship with a steeper increase in risk at higher waist circumference
Waist-to-Hip Ratio

- 5 studies in dose-response meta-analysis for waist-to-hip ratio and endometrial cancer (n = 2330).
  - 21% increased risk per 0.1 units
What's the connection?

- Insulin and leptin are elevated in obese people and can promote the growth of cancer cells.
  - Insulin resistance is increased and pancreas compensates by increasing insulin production
  - Hyperinsulinemia increases the risk of cancer (colon, endometrium, and possibly pancreas and kidney)
- Sex steroid hormones (estrogens, androgens, progesterone) are likely to play a role in obesity and cancer.
  - Adipose tissue is the main site of estrogen synthesis
  - Increased levels of estrogens
- Obesity is associated with low-grade chronic inflammation
  - The fat cell produces pro-inflammatory factors
  - Elevated levels of circulating tumor necrosis factor, interleukin 6, and C-reactive protein
Conclusion

• There is ample evidence for an association between various measures of body fatness and endometrial cancer.
• The evidence is generally consistent, and there is a dose-response relationship.
• The evidence that greater body fatness, including abdominal fatness and adult weight gain, is a cause of endometrial cancer is convincing.
Putting research into practice
Guidelines from the American Institute for Cancer Research and World Cancer Research Fund

- Be as lean as possible without becoming underweight.
- Engage in physical activity for at least 30 minutes a day.
- Avoid sugary drinks.
- Eat more of a variety of vegetables, fruits, whole grains and legumes.
- Limit consumption of red meats (beef, pork and lamb) and avoid processed meats.
- If consumed at all, limit alcoholic drinks to 2 for men and 1 for women a day.
- Limit consumption of salty foods and processed foods.
- Do not use vitamin/mineral or herbal supplements to protect against cancer.
- NEW MOTHERS: Breastfeed babies exclusively for up to 6 months and then add other liquids and foods.
- And always remember – do not smoke or chew tobacco.
Be as lean as possible without becoming underweight

• **BMI** = \[ \text{weight (lbs) ÷ height}^2 \text{ (in}^2) \] x 703
  – *Goal: BMI between 18.5 and 25*
  – Overweight = BMI between 25 and 30
  – Obesity = BMI above 30

• Any weight loss is helpful
  – Set small goals
  – 5-10 pounds at a time
Abdominal fatness

• Even if you are in “goal” BMI range you still have to be concerned about a large waist or excessive adult weight gain
• Waist circumference measures “abdominal fatness”
  – Includes subcutaneous visceral fat stores
• Goal:
  – **Men:** No more than 37 inches
  – **Women:** No more than 31.5 inches
Physical Activity Goals

At least 30 minutes of moderate activity daily (150 minutes per week)

- Start slowly and add exercise to your day gradually
  - Take stairs or park car farther away
  - Utilize step-counter
- Walking one of the easiest ways to stay active
- Work out in the water - much easier on joints
- Any amount of regular physical activity is better than none
Eat more of a variety of vegetables, fruits, whole grains and legumes

- Vegetables (including beans), fruits, and whole grains are low glycemic foods.

- Whole grains foods are made from the entire grain seed and are lower in calories, higher in fiber and contain more vitamins and minerals than refined (white) grain products.

- Each contain numerous potentially beneficial vitamins, minerals, fiber, phytochemicals and antioxidants that may help prevent cancer.
Achieving greater vegetable, fruit, legume and whole grain intake.

• Fill ⅔ of your plate with plant based foods
• **Aim for 5 cups of fruits and vegetables every day**
• Aim for 3 cups of whole grains each day
• Eat beans and/or legumes 4-5 times each week
• Snack on plant based foods throughout the day
• Stock up on canned and frozen fruits and vegetables
• Visit your local farmers market
• Try meatless meals 2 times per week
• **Eat the rainbow**
Tips for limiting alcohol consumption

• If you don’t already drink- DON’T START
• If you choose to drink stay within the recommended guidelines
  – 2 drinks for men and 1 for women per day
• Consider non-alcoholic beer or wine as options
• Alternate between non-alcoholic and alcoholic drinks
• Dilute alcoholic drinks
• Sip on sparkling water

One drink equals:
• 5 ounces of wine
• 12 ounces of beer
• 1.5 ounces of 80 proof liquor
Lactation

• AICR recommends that mothers breastfeed exclusively for up to six months and then add other liquids and foods.
Phytoestrogens

• Have weak estrogenic activity
  – Bind to estrogen receptor similarly to estradiol
• Isoflavones are most abundant in commonly consumed foods
  – Include soybeans, clover, alfalfa sprouts, oil seeds
• Studies investigating the effects of different forms of isoflavone supplementation have found conflicting results regarding endocrine function (changes in menstrual cycle, hormone levels, changes in vaginal epithelium, etc).
Flaxseed

• Flaxseed’s possible cancer-protective substances:
  – Lignans – estrogen-like compounds that act like weak estrogens.
  – Alpha-linolenic acid (ALA) – an omega-3 fatty acid that has shown promising health benefits, including offering potential protection from some cancers.
  – Fiber – indigestible type of carbohydrate that helps control body weight and
• Flaxseed may lower breast cancer risk, but there is not enough research to recommend it for cancer protection

• **Does flaxseed increase breast cancer risk?**
  – This was a concern. But studies have shown that flaxseed taken at recommended doses does not increase incidence or recurrence of breast cancer.
• **Is it safe to take flaxseed if patient is on tamoxifen or aromatase inhibitor treatment?**
  – There are no human studies to date (animal studies with tamoxifen suggest it is safe).
Flaxseed

• A moderate amount of flaxseed is safe for most people
  • At this time, it’s not recommended to take more than the 4 tablespoons of ground flaxseed per day
• The following groups of people should discuss taking flaxseed with doctor prior to starting:
  – Pregnant or breastfeeding women, and children (the effect of flaxseed is not known).
  – People taking medications, including diabetes and anticoagulant meds: flaxseed may slow absorption or interact with some medications.
  – People taking fish oil or EPA + DHA supplements: flaxseed and flaxseed oil may increase the effects of these blood thinning supplements.
Soy and Breast Cancer

- **Conflicting reports exist**
  - Lab studies have shown slow growth of breast cancer cells.
  - Some human studies have shown stimulatory effect on breast tissue with soy supplementation.
  - Epidemiological analyses in Asian women with breast cancer found links between higher isoflavone intake and reduced mortality.
  - May protect against hormone-dependent cancers, especially when ingested during childhood or adolescence.
  - No adverse effects noted on recurrence or survival from consuming soy and soy foods, may have positive synergistic effect with tamoxifen.
  - Research has suggested that the estrogen-like effects of isoflavones may reduce the effectiveness of hormone therapies used to treat breast cancer.

Zhang FF, Haslam DE, Terry MB, Knight JA, Andrulis IL, Daly MB, Buys SS, John EM. Dietary isoflavone intake and all-cause mortality in breast cancer survivors: The Breast Cancer Family Registry. Cancer. 6 March 2017 (online)

- Looked at the relationship between dietary intake of isoflavones and death from any cause in 6235 American and Canadian women with breast cancer.
- Over a median follow-up of nine years, women with breast cancer who consumed high amounts of isoflavones (>1.5 mg) had a 21 percent lower risk of dying than women who consumed low amounts (<0.3 mg).
  - The investigators noted that they examined only naturally occurring dietary isoflavones, not isoflavones from supplements
- This decrease was confined to women with hormone receptor-negative tumors and women who were not treated with anti-estrogen therapy such as tamoxifen (which blocks the effects of estrogen).
- In contrast to some previous research, high levels of isoflavone intake were not associated with greater mortality among women receiving hormonal therapy.
Recommendation for Soy Consumption

- Consuming 2-3 servings of whole soy food (or 100 mg isoflavone) daily is likely acceptable
  - Based on usual intake in Asia and amounts consumed in epidemiological studies and clinical trials
  - 1 serving of traditional soyfood provides 35 mg isoflavones
- Recommend whole soy (soybeans, tempeh, tofu) over more processed items (soy milk, soy burgers, soy protein drinks)
  - Processing soy increases hydrolization of isoflavones to a more absorbable form
- Discourage soy dietary supplements
• Examples of whole soy foods with amount of soy isoflavones (mg)
• **Miso (1 tbsp)** 7-10
• **Soybeans, edamame (1/2 cup)** 35
• **Soymilk (8 fl oz)** 23
• **Soy nuts (1/4 cup)** 40-50
• **Tempeh (1/2 cup)** 36
• **Tofu** 39
Questions?