

Cancer Drugs, Supplements, Interactions & Food

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Drug Interaction Overview

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There are approximately 1000 drugs and fixed-drug combinations used in the United States

Almost 400 of these drugs may deplete specific nutrients within the body.

Over 400 of these drugs may interact with food or food components.

Over 300 of these drugs have been shown to interact with dietary supplements, with adverse and beneficial interactions equally common.

Almost half the drugs used in clinical practice have documented nutrient **depleting** effects.

We are learning more every day. These numbers change when we add multiple medications, allergies, and symptoms.

This can be a complicated road to navigate. Make sure you have a practitioner you trust on your team throughout this journey.

Underlying Factors that increase interactions

It is possible to take advantage of positive drug interactions. The negative interactions are usually of more interest because they are often unexpected and may even go undiagnosed. The factors or conditions that predispose or favor the appearance of interactions include:

- **Age:** factors relating to how human physiology changes with age may affect the interaction of drugs.
- **Polypharmacy:** The more drugs a patient takes the more likely it will be that some of them will interact.
- **Genetic factors:** Genes synthesize enzymes that metabolize drugs. The consequence of this would, on occasions, be a greater predisposition towards drug interactions and therefore a greater predisposition for adverse effects to occur. This is seen in genotype variations in the isozymes of cytochrome P450.
- **Hepatic or renal diseases:** The blood concentrations of drugs that are metabolized in the liver and / or eliminated by the kidneys may be altered if either of these organs is not functioning correctly. If this is the case an increase in blood concentration is normally seen.
- **Serious diseases that could worsen if the dose of the medicine is reduced.**
- **Drug dependent factors.**
 - **Narrow therapeutic index:** Where the difference between the effective dose and the toxic dose is small. The drugs digoxin and warfarin are examples of this type of drug.
 - **Steep dose-response curve:** Small changes in the dosage of a drug produce large changes in the drug's concentration in the patient's blood plasma.
 - **Overloaded liver metabolism:** The capacity to metabolize the drug is greatly decreased based on liver and kidney function.

Common Drug Treatments

Figure 5.4: Chemotherapy drugs for early and locally advanced breast cancer

Drug (abbreviation)	Brand name
Carboplatin (C)	Paraplatin
Cyclophosphamide (C)	Cytosan
Docetaxel (T)	Taxotere

Doxorubicin (A)	Adriamycin
Epirubicin (E)	Ellence
Methotrexate (M)	Various brand names
Paclitaxel (T)	Taxol

Figure 5.5: Chemotherapy drug combinations for early and locally advanced breast cancer

Drug (abbreviation)	Drug combination
AC	Doxorubicin and cyclophosphamide
AC—>Paclitaxel (T)	Doxorubicin and cyclophosphamide followed by paclitaxel
AC—> Docetaxel (T)	Doxorubicin and cyclophosphamide followed by docetaxel
TAC	Docetaxel, doxorubicin and cyclophosphamide

TC	Cyclophosphamide and docetaxel
CMF	Cyclophosphamide, methotrexate (Rheumatrex) and 5-fluorouracil
TCH*	Docetaxel, carboplatin and trastuzumab (Herceptin)
TCHP*	Docetaxel, carboplatin, trastuzumab and pertuzumab (Perjeta)
TH*	Paclitaxel and trastuzumab
THP*	Paclitaxel, trastuzumab and pertuzumab
ACTH*	Doxorubicin and cyclophosphamide followed by paclitaxel and trastuzumab

*Only used for HER2-positive breast cancers. TCH, TCHP, TH and THP are not used without trastuzumab.

Figure 5.6: Chemotherapy drugs for metastatic breast cancer*

Drug (abbreviation)	Brand name
Capecitabine	Xeloda
Carboplatin	Paraplatin
Cisplatin	Platinol
Cyclophosphamide (C)	Cytoxan
Docetaxel (T)	Taxotere
Doxorubicin (A)	Adriamycin
Epirubicin (E)	Ellence

Eribulin	Halaven
5-Fluorouracil (5FU or F)	Adrucil
Gemcitabine	Gemzar
Ixabepilone	Ixempra
Liposomal doxorubicin	Doxil
Methotrexate (M)	Maxtrex
Paclitaxel (T)	Taxol
Paclitaxel, albumin bound	Abraxane
Vinorelbine	Navelbine

Types of Interactions

Pharmacodynamic: two substances exhibit pharmacologic actions that support or interfere with each other's actions.

Pharmacokinetic: the absorption, distribution, excretion or enzymatic transformation of one substance is altered by another. Most adverse interactions are of this type.

- Induction of liver enzymes through interactions can raise or lower the serum levels of medications.

The Liver Detoxification Pathway

Cytochrome P450 is a family of enzymes that work within the liver to metabolize different drugs, foods, and supplements. The Human Genome Project has identified 57 human genes coding for the various cytochrome P450 enzymes.

Cytochrome P450 enzymes are present in most tissues of the body, and play important roles in hormone synthesis and breakdown (including estrogen and testosterone synthesis and metabolism), cholesterol synthesis, and vitamin D metabolism. Cytochrome P450 enzymes also function to metabolize potentially toxic compounds, including drugs and products of endogenous metabolism such as bilirubin, principally in the liver.

Nutrient Depletions & Interactions

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Drug	Depletion	Interactions
Platinum Agents Cisplatin, Carboplatin, Oxaliplatin, Paraplatin	L-Carnitine Magnesium Potassium Selenium Vitamin E Zinc	Negative: Antioxidants
Anthracyclines Doxorubicin	Riboflavin Vitamin E CoQ10 (supplement with Carnitine)	Negative: Grapefruit, St. John's Wort, sweet orange
Fluorouracil	Niacin Thiamin Vitamin E	Negative: many chemotherapy drugs * No foods are known to interact
Taxane drugs Paclitaxel, Docetaxel,	Vitamin E	Negative: Grapefruit, St. John's Wort, sweet orange, alcohol

*There are a lot of potential food interactions with chemotherapy drugs. Be aware of your body and get to know what feels right for you. If in doubt, always check with your oncologist or integrative practitioner



GET
Ready For
ACTION!

Before treatment

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This time before treatment is the time to optimize your wellness, address current and future depletions as well as other underlying issues. By doing so, you better your treatment outcome and quality of life.

- Know your chemotherapy and/or radiation protocol
- Understand the need for diet and exercise as it applies to you
- Prepare what you need around the home to make treatment easier
- Lower your toxic burden
- Get your healthcare team figured out

Diet and Exercise

[Chirurgia \(Bucur\)](#). 2017 Jul-Aug;112(4):457-468. doi: 10.21614/chirurgia.112.4.457.

Results of 1-year Diet and Exercise Interventions for ER+/PR±/HER2- Breast Cancer Patients Correlated with Treatment Type.

Artene DV, [Bordea CI](#), [Blidaru A](#).

Abstract

PURPOSE: Many breast cancer patients gain weight during chemotherapy and antiestrogenic treatment increasing recurrence, oncologic specific and all-cause mortality risks. Patients and **Methods:** 165 ER+/PR±/HER2- breast cancer patients under antiestrogenic treatment were randomly assigned to follow an at-home diet based on food naturally high in proteins, calcium, probiotics and prebiotics (D), or this diet and 4' isometric exercises (D+Ex) for 1 year. We measured weight (W), body (BF) and visceral fat (VF) using a multi-frequency bioelectrical impedance scale on the 6th and 12th month and we correlated results with chemotherapy, surgery and antiestrogenic medication type. Results were analysed using the Friedman Test, then with Wilcoxon signed-rank tests if Friedman Test was significant. **Results:** Overall, the patients 1-year results show that both D+Ex and D patients obtained statistically significant weight loss and fat loss. D patients lost 3.3 kg, 3.2% BF and 1% visceral fat. D+Ex patients lost 6.5 kg, 3.3% BF and 2% visceral fat. D+Ex patients obtained statistical significance for W, BF and VF regardless of chemotherapy, surgery or antiestrogenic treatment type. D patients with mastectomy or with aromatase inhibitors lost W, BF and VF. D patients with conservatory surgery, adjuvant or both neoadjuvant and adjuvant chemotherapy and those on Tamoxifen only lost W. D patients with neoadjuvant chemotherapy also lost VF.

CONCLUSION: This diet is effective for ER+/PR±/HER2- breast cancer patients on antiestrogenic medication. Adding at least a minimal exercise protocol improves patients chances of counteracting sarcopenic obesity.

Nutrition



- It is important to keep a low simple carbohydrate diet with limited processed foods. Sugar comes in many forms and is one of the main sources of energy for the cancer.
- Increase your intake and variety of vegetables. They should make up at least half of your plate. Approximately 5-9 servings per day is suggested.
 - Eat the rainbow!
- Increase good fats in the diet
- Eat good quality protein from both meat and vegetable sources to prevent wasting.
 - When eating meat, choose grass fed or pasture raised because the food the animals eat is sprayed with pesticides. Then the animals are given antibiotics and growth enhancers that may resemble estrogen.

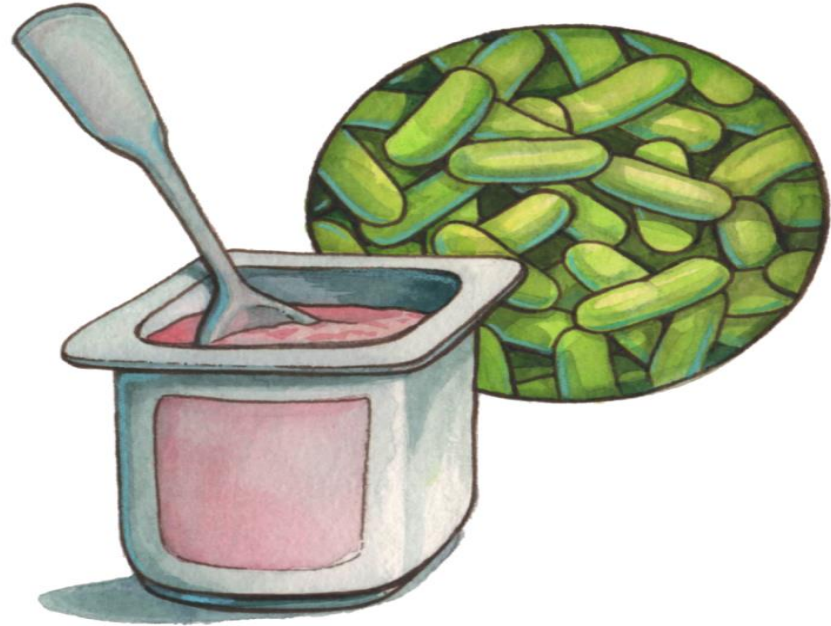
Gut Health

If your gut health is compromised, chemotherapy may not be as effective, side effects may be more severe, and nutrient depletions more rampant.

*Probiotics have no interactions with chemotherapy, so they can be taken during treatment.



Probiotics



Managing Weight

Weight loss/gain is a common part of cancer treatment so keeping your weight stable prior to treatment can help prevent muscle wasting or fat gain.

Cancer requires more energy therefore caloric requirements will change.

Protein needs tend to increase despite caloric needs to prevent any muscle wasting due to the chemotherapy drugs.

*Many doctors have a body composition machine that can help you track the changes in your body. You can also get an inexpensive home scale in order to track body weight trends and adjust your diet as you need.

Managing Glucose Levels

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Making sure that you have balanced and well managed blood sugar levels is an important factor in your outcome.

Normal fasting blood sugar levels are around 80-85. High blood sugar levels can feed the cancer as well as increase inflammation throughout the body.

Markers for glucose levels are hemoglobin A1C, fasting insulin, and glucose levels.

- Reduce sugar intake
- Eat natural sugar foods (low glycemic fruit) combined with fiber and fat.
 - Ex: apple with almond butter

Glucose insult elicits hyperactivation of cancer stem cells through miR-424-cdc42-prdm14 signalling axis.

Nandy SB¹, Orozco A², Lopez-Valdez R¹, Roberts R³, Subramani R¹, Arumugam A¹, Dwivedi AK⁴, Stewart V³, Prabhakaran G³, Jones S⁵, Lakshmanaswamy R^{1,2}.

Author information

Abstract

BACKGROUND: Meta-analysis shows that women with diabetes have a 20% increased risk of breast cancer and also an increased risk for distant metastasis and mortality. The molecular mechanisms for distant metastasis and mortality in breast cancer patients with diabetes are not very well understood.

METHODS: We compared the effect of physiological (5 mM) and diabetic (10 mM) levels of glucose on malignant breast epithelial cell invasion and stemness capabilities. We performed microRNA array to determine the dysregulated microRNAs in hyperglycaemic conditions and performed functional and molecular analysis of the gene targets.

RESULTS: Hyperglycaemia leads to hyperactivation of cancer stem cell pool and enhances invasive ability of breast cancer cells. MiR-424 seems to be a key regulator of cancer cell stemness and invasion. Knockdown of miR-424 in cancer cells under euglycaemic conditions leads to enhanced invasion and stem cell activity, whereas ectopic expression of miR-424 in cancer cells under hyperglycaemic conditions results in suppressed invasion and stem cell activity. Cdc42, a target of miR-424, influences cancer stem cell activity by positively regulating prdm14 through activation of pak1 (p-21-activated kinase 1) and stat5.

CONCLUSIONS: Our findings establish miR-424→cdc42→prdm14 axis as a key molecular signalling cascade that might influence breast cancer progression in diabetic patients through hyperactivation of cancer stem cells. British Journal of Cancer advance online publication, 21 October 2017; doi:10.1038/bjc.2017.335 www.bjcancer.com.

Anti-angiogenic foods are foods that prevent the formation of blood vessels that tumors need to grow.

Dietary Sources of Naturally-Occurring Antiangiogenic Substances

Green tea

Strawberries

Blackberries

Raspberries

Blueberries

Oranges

Grapefruit

Lemons

Apples

Pineapple

Cherries

Red grapes

Red wine

Bok choy

Kale

Soy beans

Ginseng

Maitake mushroom

Licorice

Turmeric

Nutmeg

Artichokes

Lavender

Pumpkin

Sea Cucumber

Tuna

Parsley

Garlic

Tomato

Olive oil

Grape seed oil

Dark chocolate

Others

Toxicity Management

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- This has taken on new meaning since industrialization. While technology has advanced, the cost to the environment has been heavy and little has been done to counteract it.
- A lot of chemicals used currently in America disrupt hormones, are hard on the liver, or can be linked to cancer. Ridding our body of as many as possible is integral to leading a healthy lifestyle, let alone improving cancer treatments.
- Eat as organically as possible. Wash everything well. You can use 2 tablespoons of white vinegar in a sink of water to wash leafy vegetables and scrub the rest.
- Be conscious that toxicity is everywhere... Household cleaning products, dry cleaning fluid, tap water, etc.

REDUCE YOUR TOXIC BURDEN

“Dirty Dozen Endocrine Disruptors” according to the Environmental Working Group

Endocrine Disruptor	Sources
Bisphenol-A (BPA)	Plastic bottle, Canned food, Receipt paper
Dioxin	Animal products (this environmental toxin accumulates up the food chain)
Atrazine	Conventionally grown food (this is an herbicide), Water
<u>Pthalates</u>	Plastic food containers, Plastic wrap, Fragrances, Cosmetics
Perchlorate	Drinking water (reverse osmosis removes it)
Fire Retardants	New furniture and carpets
Lead	Old paint, Unfiltered water
Arsenic	Conventional chicken, Rice, Unfiltered water
Mercury	Seafood
<u>Perfluorinated chemicals (PFCs)</u>	Nonstick pans, Water-resistant clothing, furniture, and carpets
Organophosphate pesticides	Conventionally grown produce
Glycol ethers	Cleaning products



EWG'S 2017

Dirty 12™

1. STRAWBERRIES
2. SPINACH
3. NECTARINES
4. APPLES
5. PEACHES
6. PEARS
7. CHERRIES
8. GRAPES
9. CELERY
10. TOMATOES
11. SWEET BELL PEPPERS
12. POTATOES



EWG'S 2017

Clean 15™

1. SWEET CORN
2. AVOCADOS
3. PINEAPPLES
4. CABBAGE
5. ONIONS
6. SWEET PEAS
7. PAPAYAS
8. ASPARAGUS
9. MANGOES
10. EGGPLANT
11. HONEYDEW
12. KIWI
13. CANTALOUPE
14. CAULIFLOWER
15. GRAPEFRUIT

Monterey Bay Aquarium® Seafood Watch®

The Monterey Bay Aquarium Seafood Watch program creates science-based recommendations that help consumers and businesses make ocean-friendly seafood choices. Carry this guide with you and share it with others to help spread the word.

BEST CHOICES

Arctic Char (farmed)
Barramundi (US & Vietnam farmed)
Bass: Striped (US hook and line, farmed)
Bluefish (US handline)
Catfish (US)
Clams, Mussels & Oysters
Crab: King, Snow & Tanner (AK)
Croaker: Atlantic (beach seine)
Lionfish (US)
Mahi Mahi (US handline)
Prawn: Freshwater (Canada & US)
Prawn: Spot (AK & Canada)
Rockfish (AK, CA, OR & WA)
Salmon (AK & New Zealand)
Sardines: Pacific (Canada & US)
Scallops (farmed)
Seaweed (farmed)
Shrimp (US farmed & AK)
Swordfish (Canada & US buoy, handline, harpoon)
Tilapia (Canada, Ecuador, Peru & US)
Tuna: Albacore (troll, pole and line)
Tuna: Skipjack (Pacific troll, pole and line)

GOOD ALTERNATIVES

Bluefish (US gillnet and trawl)
Branzino (Mediterranean farmed)
Cod: Atlantic (handline)
Crab: Atlantic Rock (Canada & MA)
Crab: Dungeness (Canada & US)
Crab: Jonah (US)
Haddock
Hake (US)
Lobster (Bahamas, Canada & US)
Monkfish (US)
Pollock (Canada longline, gillnet & US)
Redfish (US)
Salmon: Atlantic (ME farmed)
Scallops: Sea (wild)
Shrimp (Canada & US wild, Ecuador & Honduras farmed)
Squid (Chile, Mexico, Peru & US)
Tilapia (China, Colombia, Honduras, Indonesia, Mexico & Taiwan)
Tuna: Albacore (US longline)
Tuna: Skipjack (free school, imported troll, pole and line, and US longline)
Tuna: Yellowfin (free school, troll, pole and line, US longline)

AVOID

Cod: Atlantic (Canada & US)
Crab (Asia & Russia)
Crab: Atlantic Rock (US, except MA)
Halibut: Atlantic (wild)
Mahi Mahi (imported)
Octopus: Common (Portugal & Spain trawl, Mexico)
Orange Roughy
Pollock (Canada trawl)
Salmon: Atlantic (farmed)
Sardines: Atlantic (Mediterranean)
Sharks
Shrimp (imported)
Squid (China, India & Thailand)
Swordfish (imported longline)
Tuna: Albacore (imported except troll, pole and line)
Tuna: Bluefin
Tuna: Skipjack (imported purse seine)
Tuna: Yellowfin (longline except US)

Many seafood items appear in more than one column. Please be sure to check them all.

Best Choices

Buy first, they're well managed and caught or farmed responsibly.

Good Alternatives

Buy, but be aware there are concerns with how they're caught or farmed.

Avoid

Take a pass on these for now, they're overfished or caught or farmed in ways that harm other marine life or the environment.

This guide includes some of our recommendations for popular seafood. **For the full list, visit us online or download our app.**

Environmental Working Group - Skin Deep Cosmetics and Skincare Database



Hazard score key:



Low hazard



Moderate hazard



High hazard

Food preparation during treatment

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Plan meals ahead of time for when you are in treatment.

Freeze meals

Plan to have help cooking

Join a subscription meal service if necessary.

Find a protein shake supplement to have on hand

Lower your environmental toxicity levels.

SUPPLEMENTS AND CANCER:

What can I take?



Timing of chemotherapy and supplements, herbs, etc.

Most chemotherapy is out of the body within a few hours to a few days. This timing should be discussed with your oncologist before adding any supplements back into your treatment. .

Your doctor should be able to give you an exact amount of time depending on your personal treatment plan (drug combination).

Because of the cardiotoxic effect of many chemotherapy drugs, “loading” antioxidants or other supplements prior to or in between treatments may be beneficial but should be discussed with your healthcare team.

- Ex:, CoQ10, Curcumin

The studies on supplement use during chemotherapy are very limited and very specific on the type of chemotherapy being used.

Immune Support

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Stress is a negative interaction with cancer in many different ways.

“In modern lifestyle societies, chronic stress has been associated with the pathogenesis of many diseases, including cancer. Chronic stress results in the activation of specific signaling pathways in cancer cells and the tumor microenvironment, leading to tumor growth and progression.”

- Sleep - Get 7 or more uninterrupted hours of sleep per night
- Nutrition - Healthy well balanced diet is essential for your health and your immune system
- Exercise - Researchers have long observed the positive effects of exercise on the immune system
 - Don't overdo it
- Stress reduction - Chronically high levels of stress hormones (cortisol and adrenaline) suppress the immune system and reduce the body's ability to repair and defend itself

Vitamin C

The use of antioxidants like vitamin C during chemotherapy is controversial. There is concern that antioxidants could reduce the activity of chemotherapy drugs which generate free radicals.

Preliminary data from an animal lymphoma model indicate that vitamin C pretreatment reduces the efficacy of doxorubicin.

Leukemia and lymphoma cell culture studies also suggest that vitamin C pretreatment can reduce the cytotoxicity of doxorubicin, cisplatin, vincristine, methotrexate, and imatinib. Since this list includes drugs which do not generate free radicals, mechanisms other than the antioxidant effects of vitamin C might be involved.

This might include prevention of the mitochondrial membrane depolarization caused by many chemotherapy drugs, which is involved in regulating cell death.

In contrast, some researchers theorize that antioxidants might make chemotherapy more effective by reducing oxidative stress that could interfere with apoptosis (cell death) of cancer cells. Speak with your oncologist or healthcare team before using Vitamin C.

Vitamin D

Vitamin D helps the body absorb calcium, which is essential for good bone health. Vitamin D also helps the immune, muscle, and nervous systems function properly. Most vitamin D is made when an inactive form of the nutrient is activated in your skin when it's exposed to sunlight.

- Smaller amounts of vitamin D are in fortified milk and other foods, fatty fish, and eggs.

As more and more people spend most of their time out of direct sunlight or wearing sunscreen when they are in the sun, vitamin D production from sun exposure is limited.

Research suggests that women with low levels of vitamin D have a higher risk of breast cancer. Vitamin D may play a role in controlling normal breast cell growth and may be able to stop breast cancer cells from growing.

Vitamin D

“Recent evidence shows that vitamin D may have a protective effect against the development of breast cancer and breast cancer progression. Studies have shown that vitamin D is able to inhibit the growth of breast cancer cells in vitro, and epidemiologic studies in the United States have concluded that the risk of breast cancer is inversely proportional to the intensity of local sunlight. ”

Immune Support-AHCC

Cancer Biol Ther. 2017 Sep 8:1-10. doi: 10.1080/15384047.2017.1373211. [Epub ahead of print]

MicroRNA signature in the chemoprevention of functionally-enriched stem and progenitor pools (FESPP) by Active Hexose Correlated Compound (AHCC).

Graham EA¹, Mallet JF², Jambi M², Nishioka H³, Homma K³, Matar C^{1,2}.

+ Author information

Abstract

PURPOSE: Many breast cancer patients use natural compounds in their battle against breast cancer. Active Hexose Correlated Compound (AHCC®) is a cultured mushroom mycelium extract shown to favorably modulate the immune system and alleviate cancer burden. Cancer Stem cells (CSCs) are a subset of highly tumorigenic cancer cells that are thought to be responsible for recurrence. CSCs can be epigenetically regulated by microRNAs (miRNAs). We hypothesized that AHCC may influence CSCs by modulating tumor-suppressor or oncogenic miRNAs.

METHODS: Functionally-enriched stem and progenitor pools (FESPP) were isolated in the form of mammospheres from MDA-MB-231, MCF-7, and 4T1 cells, exposed to AHCC in both regular and primary culture from Balb/c mice, and analyzed by visual counting and flow cytometry. Cell motility was also observed in MDA-MB-231 cells. Profiling and RT-qPCR were performed to determine AHCC influence on miRNAs in MDA-MB-231 mammospheres. Additionally, Balb/c mice were orally gavaged with AHCC, and tumor growth parameters and miR-335 expression were analyzed. MDA-MB-231 cells were transfected with miR-335 and analyzed by western blot.

RESULTS: We demonstrated that AHCC reduced mammosphere growth in three cell lines and in primary culture, prevented cell migration, and upregulated miR-335 expression in MDA-MB-231 cells and mouse tumor samples. Among the differentially regulated miRNAs in CSCs, we focused on tumor suppressor miR-335, known to target extracellular matrix protein Tenascin C (TNC). TNC is involved in CSC immune evasion pathways. In MDA-MB-231, inhibition of miR-335 increased TNC protein expression.

CONCLUSIONS: These results support that AHCC limits FESPP growth, partly by targeting miRNA pathways.

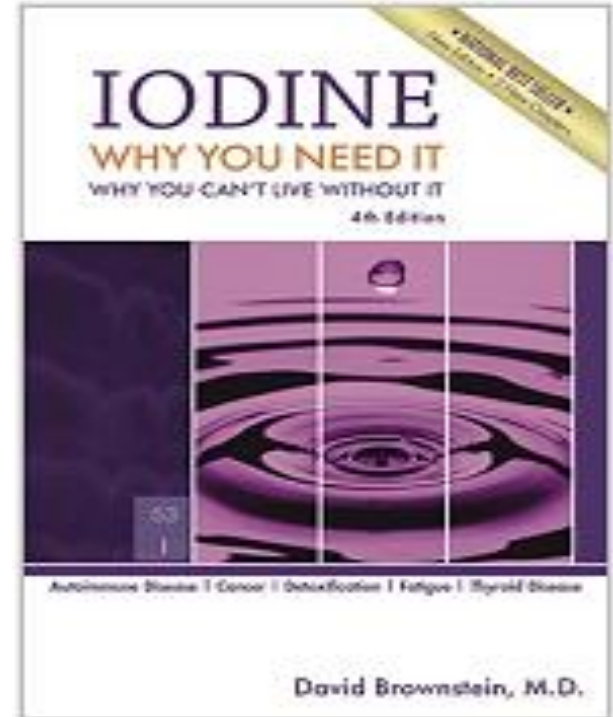
Chemoresistance

The insensitivity of these **cancer** cells causes resistance to chemotherapy or **chemoresistance** that is a major cause of treatment failure with treatment strategies involving chemotherapy (Szakacs et al., 2006). Some of the tumor cells are already resistant to the “achievable” doses of anticancer drugs.

Iodine

Iodine is a crucial ingredient in the delicate balance of the endocrine system, and deficiencies have been implicated in conditions such as diabetes, polycystic ovarian disease (PCOS), fibrocystic breast disease, increased Breast Cancer risk and most commonly goiter.

Normal breast architecture requires adequate Iodine, and in a deficient state, the breasts and thyroid compete for available Iodine.



Iodine and Chemoresistance to Doxorubicin

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[Oncol Rep.](#) 2017 Sep 1. doi: 10.3892/or.2017.5934. [Epub ahead of print]

Molecular iodine impairs chemoresistance mechanisms, enhances doxorubicin retention and induces downregulation of the CD44+/CD24+ and E-cadherin+/vimentin+ subpopulations in MCF-7 cells resistant to low doses of doxorubicin.

[Bontempo A](#)¹, [Ugalde-Villanueva B](#)¹, [Delgado-González E](#)¹, [Rodríguez ÁL](#)², [Aceves C](#)¹.

➕ Author information

Abstract

One of the most dreaded clinical events for an oncology patient is resistance to treatment. Chemoresistance is a complex phenomenon based on alterations in apoptosis, the cell cycle and drug metabolism, and it correlates with the cancer stem cell phenotype and/or epithelial-mesenchymal transition. Molecular iodine (I₂) exerts an antitumor effect on different types of iodine-capturing neoplasms by its oxidant/antioxidant properties and formation of iodolipids. In the present study, wild-type breast carcinoma cells (MCF-7/W) were treated chronically with 10 nM doxorubicin (DOX) to establish a low-dose DOX-resistant mammary cancer model (MCF-7/D). MCF-7/D cells were established after 30 days of treatment when the culture showed a proliferation rate similar to that of MCF-7/W. These DOX-resistant cells also showed increases in p21, Bcl-2 and MDR-1 expression. Supplementation with 200 μM I₂ exerted similar effects in both cell lines: it decreased the proliferation rate by ~40%, and I₂ co-administration with DOX significantly increased the inhibitory effect (to ~60%) and also increased apoptosis (BAX/Bcl-2 index), principally by inhibiting Bcl-2 expression. The inhibition by I₂ + DOX was also accompanied by impaired MDR-1 induction as well as by a significant increase in PPAR γ expression. All of these changes could be attributed to enhanced DOX retention and differential down-selection of CD44+/CD24+ and E-cadherin+/vimentin+ subpopulations. I₂ + DOX-selected cells showed a weak induction of xenografts in Foxn1nu/nu mice, indicating that the iodine supplements reversed the tumorigenic capacity of the MCF-7/D cells. In conclusion, I₂ is able to reduce the drug resistance and invasive capacity of mammary cancer cells exposed to DOX and represents an anti-chemoresistance agent with clinical potential.

Berberine - Triple negative but not with chemo

Biomed Pharmacother. 2017 Aug 18;95:18-24. doi: 10.1016/j.biopha.2017.08.045. [Epub ahead of print]

Berberine activates caspase-9/cytochrome c-mediated apoptosis to suppress triple-negative breast cancer cells in vitro and in vivo.

Zhao Y¹, Jing Z², Lv J¹, Zhang Z¹, Lin J¹, Cao X¹, Zhao Z¹, Liu P³, Mao W⁴.

⊕ Author information

Abstract

Berberine (BBR) is an isoquinoline alkaloid isolated from *Cotridis rhizoma* and exhibits multiple biological roles including anti-microbe, anti-inflammation and anti-tumor activities. In this study, two triple-negative breast cancer cell (TNBC) lines, MDA-MB-231 and BT549, were used to investigate the effect of BBR on growth of TNBC in vitro and in vivo. 3-(4,5-dimethylthiazol-2-yl)-2,5-diphenyltetrazolium bromide (MTT) assay was used to evaluate the viability of cells treated with BBR. After 48h treatments, a 50% inhibitory concentration (IC₅₀) of BBR to BT549 and MDA-MB-231 cells are at 16.575±1.219µg/ml and 18.525±6.139µg/ml respectively. BBR reduced colony formation of BT549 and MDA-MB-231 cells. The wound-healing assay showed BBR decreased breast cancer cell migrations (P<0.01). AnnexinV-PI staining assay confirmed BBR induced cellular apoptosis. The expressions of caspase-3, caspase-9, Bcl-2 and Bax were detected by western blot, which showed BBR activated caspase-3, 9 and Bax, but down-regulated Bcl-2 expression. BBR promoted the release of cytochrome c through the immunofluorescent analysis (P<0.01). We also found BBR increased the level of cellular γH2AX and increased the expression of Ligase4, which suggests BBR induces the double-strand breaks (DSB). These results thus demonstrated that BBR induced DSB, subsequently increased the release of cytochrome c and eventually triggered the caspase9-dependent apoptosis. In addition, we used a MDA-MB-231 mouse-xenograftmodel to evaluate the effect of BBR on tumor growth. BBR suppressed tumor growth and increased caspase-9 levels in xenograft tumors through immunohistochemistry analysis (P<0.01). Taken together, these results demonstrate that BBR activates caspase-9/cytochrome c-mediated apoptosis to inhibit the growth of TNBC breast cancer cells in vitro and in vivo.

Chronic Inflammation

Inflammation is a normal physiological response that causes injured tissue to heal. An inflammatory process starts when chemicals are released by the damaged tissue. In response, white blood cells make substances that cause cells to divide and grow to rebuild tissue to help repair the injury. Once the wound is healed, the inflammatory process ends.

In chronic inflammation, the inflammatory process may begin even if there is no injury, and it does not end when it should. Why the inflammation continues is not always known. Chronic inflammation may be caused by infections that don't go away, abnormal immune reactions to normal tissues, or conditions such as obesity. Over time, chronic inflammation can cause DNA damage and lead to cancer. For example, people with chronic inflammatory bowel diseases, such as ulcerative colitis and Crohn disease, have an increased risk of colon cancer.

Many studies have investigated whether anti-inflammatory medications, such as aspirin or non-steroidal anti-inflammatory drugs, reduce the risk of cancer. However, a clear answer is not yet available. For more information, see [Aspirin to Reduce Cancer Risk](#).

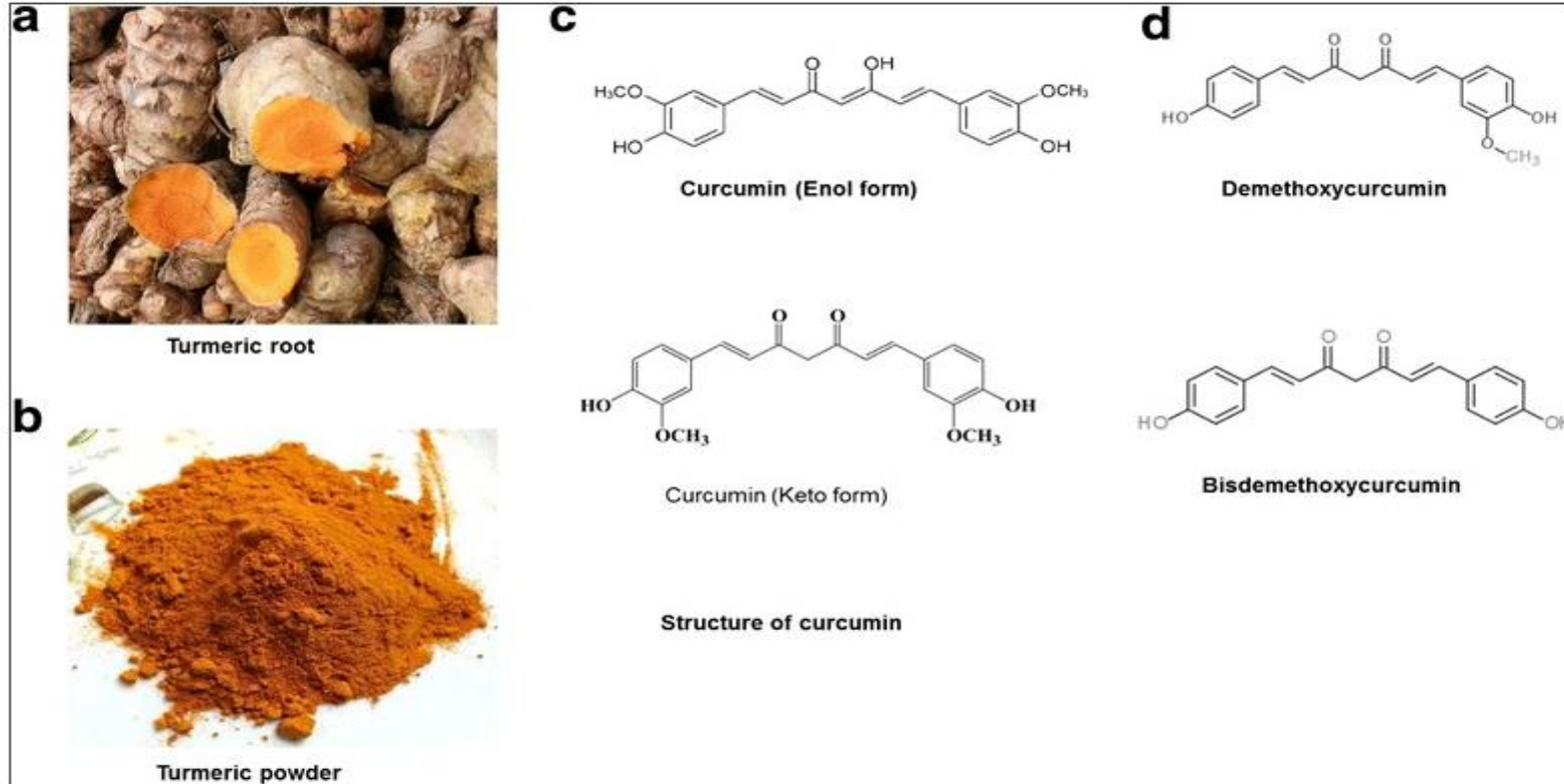
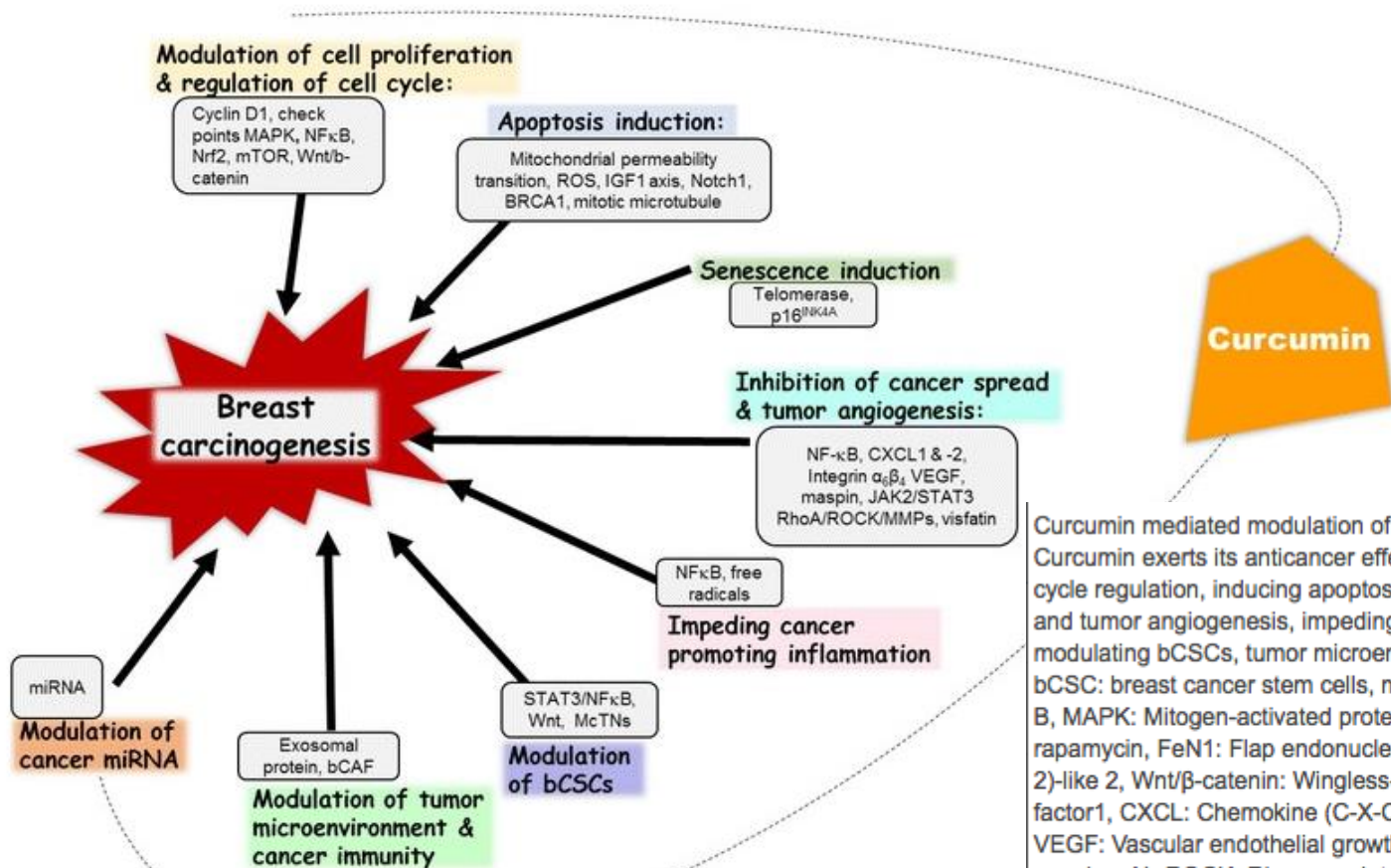


Fig. 2

The source and chemistry of curcumin. **a** Turmeric powder is obtained from the roots of plant *Curcuma longa*. **b** Curcumin is a component of turmeric. **c** The chemical structure of curcumin demonstrates a bis α , β -unsaturated diketone structure that displays keto enol tautomerism, with a predominant keto form in acidic and neutral solutions and a stable enol form in alkaline media. **d** The chemical structure of demethoxycurcumin and bisdemethoxycurcumin

[Curcumin: the spicy modulator of breast carcinogenesis](#)

J Exp Clin Cancer Res. 2017;36:98.



Curcumin mediated modulation of major events in breast carcinogenesis. Curcumin exerts its anticancer effect by modulating cell proliferation and cell cycle regulation, inducing apoptosis and senescence, inhibiting cancer spread and tumor angiogenesis, impeding tumor promoting inflammation and modulating bCSCs, tumor microenvironment, cancer immunity and miRNA. bCSC: breast cancer stem cells, miRNA: microRNA, NFκB: nuclear factor kappa B, MAPK: Mitogen-activated protein kinases, mTOR: mammalian target of rapamycin, FeN1: Flap endonuclease 1, Nrf2: Nuclear factor (erythroid-derived 2)-like 2, Wnt/β-catenin: Wingless-Int/beta-catenin; IGF1: Insulin like growth factor1, CXCL: Chemokine (C-X-C Motif) Ligand, McTNs: microtentacles, VEGF: Vascular endothelial growth factor, RhoA: Ras homolog gene family, member A), ROCK: Rho-associated, coiled-coil-containing protein kinase 1), MMPs: Matrix metalloproteinase-1, bCAFs: breast cancer associated fibroblasts, JAK2/STAT3: Janus kinase 2/Signal transducer and activator of transcription3

[Curcumin: the spicy modulator of breast carcinogenesis](#)

J Exp Clin Cancer Res. 2017;36:98.

Curcumin - The Extract from Turmeric

- [Int J Oncol.](#) 2016 Sep;49(3):1019-27. doi: 10.3892/ijo.2016.3598. Epub 2016 Jul 4.
 - **Curcumin inhibits invasive capabilities through epithelial mesenchymal transition in breast cancer cell lines.**
- [Steroids.](#) 2016 Aug 21;115:80-89. doi: 10.1016/j.steroids.2016.08.014. [Epub ahead of print]
 - **Evaluation of heterocyclic steroids and curcumin derivatives as anti-breast cancer agents: Studying the effect on apoptosis in MCF-7 breast cancer cells.**
- [RSC Adv.](#) 2014 Aug 5;4(66):35242-35250.
 - **Increased chemopreventive effect by combining arctigenin, green tea polyphenol and curcumin in prostate and breast cancer cells.**
 - This is about burdock root, green tea and curcumin

Broad effect of turmeric with platinum based drugs

— — —
[Int J Food Sci Nutr](#). 2014 May;65(3):368-74. doi: 10.3109/09637486.2013.871694. Epub 2014 Jan 20.

Curcumin-enhanced chemosensitivity of FDA-approved platinum (II)-based anti-cancer drugs involves downregulation of nuclear endonuclease G and NF- κ B as well as induction of apoptosis and G2/M arrest.

[Wang YT](#)¹, [Liu HS](#), [Su CL](#).

 **Author information**

Abstract

Curcumin, an active natural compound in turmeric and curry, has been reported to exhibit anti-cancer effect. Cisplatin, carboplatin and oxaliplatin are used to treat various types of cancers. However, acquired resistance and toxicities are observed. Here, the addition of curcumin significantly increased cytotoxicity of the anti-cancer drugs on human colorectal cancer HT-29 cells, producing synergistic (cisplatin and carboplatin) and additivity (oxaliplatin) effects. Treatments in combination with curcumin resulted in a significantly increased induction of apoptosis and occurrence of G2/M arrest. Nuclear apoptosis-inducing factor (AIF), EndoG and NF- κ B were elevated by anti-cancer drugs, suggesting the involvement of AIF and EndoG. The addition of curcumin suppressed nuclear AIF and EndoG and reversed anti-cancer drugs-induced NF- κ B expression, suggesting the association of EndoG and NF- κ B in curcumin-enhanced chemosensitivity. Therefore, the intake of foods rich in curcumin or curcumin-containing supplements should be taken into consideration for patients receiving chemotherapy to optimize the outcome of treatments.

Pre treatment with Curcumin before Doxorubicin

[Mol Cell Biochem](#). 2017 Sep 19. doi: 10.1007/s11010-017-3195-6. [Epub ahead of print]

Mode of treatment governs curcumin response on doxorubicin-induced toxicity in cardiomyoblasts.

Jain A¹, Rani V².

Author information

Abstract

Doxorubicin (Dox) is an effective anti-cancer drug with severe reported cardiotoxicity. Cardiovascular risks associated with present cancer therapeutics demand urgent attention. There has been a growing interest in naturally occurring compounds to improve the therapeutic index as well as prevent non-tumour tissues from sustaining chemotherapy-induced damages. In the present study, the effects of curcumin, a polyphenol isolated from *Curcuma longa* and well known for its anti-oxidative, anti-cancerous and anti-inflammatory properties, was studied in relation to the Dox-induced cardiotoxicity. As literature suggests conflicting role of curcumin in Dox-induced cardiotoxicity, concentration- and time-dependent studies were conducted to study the different curcumin effects. H9C2 cardiomyoblasts were used in the study and cell viability assays were done to study Dox-induced cellular death. Drug uptake assay for Dox was performed followed by cellular growth inhibition analysis by FACS Calibur. Morphological alterations, intracellular ROS levels and mitochondrial integrity were observed by fluorescent-based microscopic studies. Catalases and superoxide dismutase-inbuilt anti-oxidant enzyme activities were studied, and it was observed that Dox-dependent cardiotoxicity occurs through ROS overproduction by exaggerating the inbuilt anti-oxidant mechanism. Expression analysis for cell death and ROS markers-BCI₂, Bax, SOD, catalase-was investigated by semi-quantitative RT-PCR, and the Dox-induced stress on cardiac cells was confirmed. Initiator and effector caspases activity analysis also confirmed these findings. Our study proposes that curcumin exerts time-dependent responses on Dox-induced cardiotoxicity, where parallel treatment potentiates and pre-treatment suppresses the Dox-induced toxicity in H9C2 cardiomyoblasts. In conclusion, pre-treatment of curcumin suppresses the Dox-induced cardiotoxicity and holds a great potential as future cardio-oncological therapeutics.

Doxorubicin CoQ10 and L-Carnitine for Cardiotoxicity

[Tissue Cell](#). 2017 Jun;49(3):410-426. doi: 10.1016/j.tice.2017.03.007. Epub 2017 Apr 2.

Protective role of CoQ10 or L-carnitine on the integrity of the myocardium in doxorubicin induced toxicity.

[Mustafa HN](#)¹, [Hegazy GA](#)², [Awdan SAE](#)³, [AbdelBaset M](#)³.

Author information

Abstract

Doxorubicin (DOX) is a chemotherapeutic agent used for treatment of different cancers and its clinical usage is hindered by the oxidative injury-related cardiotoxicity. This work aims to declare if the harmful effects of DOX on heart can be alleviated with the use of Coenzyme Q10 (CoQ10) or L-carnitine. The study was performed on seventy two female Wistar albino rats divided into six groups, 12 animals each: Control group; DOX group (10mg/kg); CoQ10 group (200mg/kg); L-carnitine group (100mg/kg); DOX+CoQ10 group; DOX+L-carnitine group. CoQ10 and L-carnitine treatment orally started 5days before a single dose of 10mg/kg DOX that injected intraperitoneally (IP) then the treatment continued for 10days. At the end of the study, serum biochemical parameters of cardiac damage, oxidative stress indices, and histopathological changes were investigated. CoQ10 or L-carnitine showed a noticeable effects in improving cardiac functions evidenced reducing serum enzymes as serum interleukin-1 beta (IL-1 β), tumor necrosis factor alpha (TNF- α), leptin, lactate dehydrogenase (LDH), Cardiotrophin-1, Troponin-I and Troponin-T. Also, alleviate oxidative stress, decrease of cardiac Malondialdehyde (MDA), Nitric oxide (NO) and restoring cardiac reduced glutathione levels to normal levels. Both corrected the cardiac alterations histologically and ultrastructurally. With a visible improvements in α -SMA, vimentin and eNOS immunohistochemical markers. CoQ10 or L-carnitine supplementation improves the functional and structural integrity of the myocardium.

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Diindolylmethane and Doxorubicin

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[Free Radic Res.](#) 2017 Sep 18:1-43. doi: 10.1080/10715762.2017.1381694. [Epub ahead of print]

Attenuation of doxorubicin-induced cardiotoxicity and genotoxicity by an indole based natural compound 3,3'-diindolylmethane (DIM) through activation of Nrf2/ARE signaling pathways and inhibiting apoptosis.

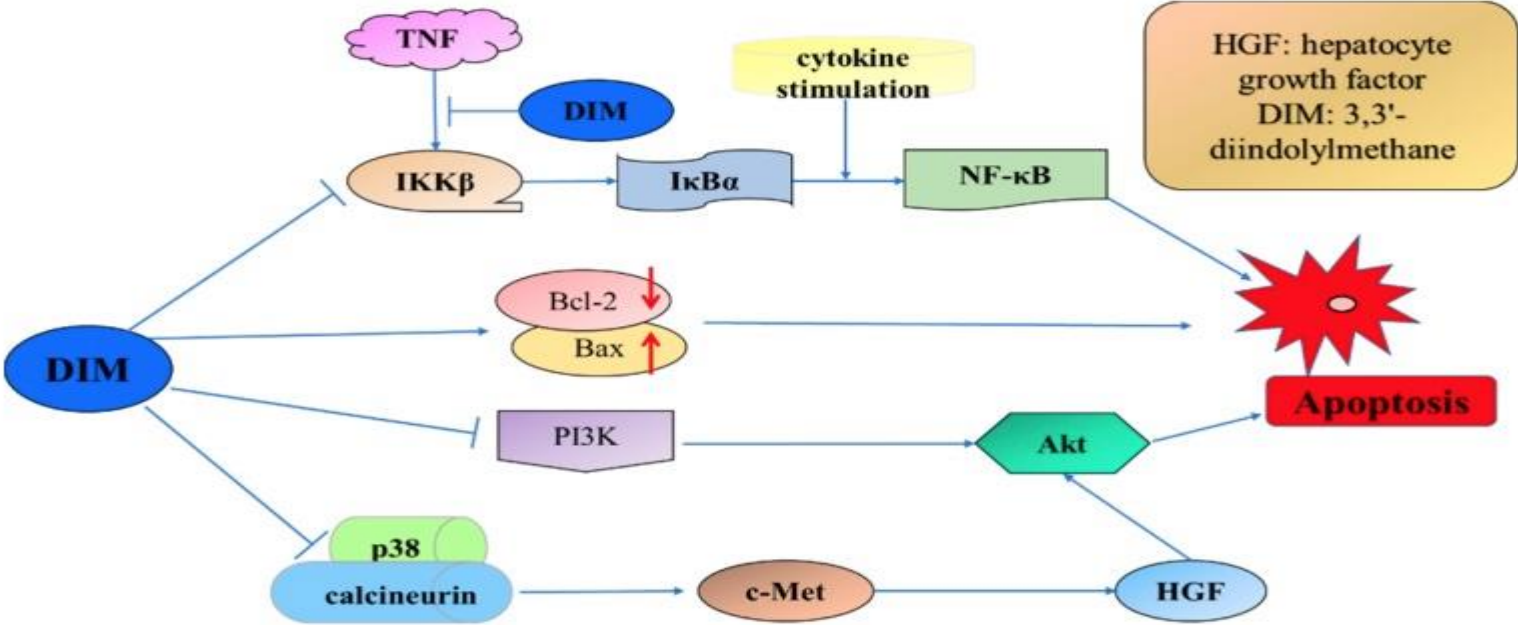
[Hajra S¹](#), [Basu A¹](#), [Singha Roy S^{1,2}](#), [Patra AR¹](#), [Bhattacharya S¹](#).

⊕ Author information

Abstract

The most crucial complication related to doxorubicin (DOX) therapy is nonspecific cytotoxic effect on healthy normal cells. The clinical use of this broad-spectrum chemotherapeutic agent is restricted due to development of severe form of cardiotoxicity, myelosuppression and genotoxicity which interfere with therapeutic schedule, compromise treatment outcome and may lead to secondary malignancy. 3,3'-diindolylmethane (DIM) is a naturally occurring plant alkaloid formed by the hydrolysis of indolylmethyl glucosinolate (glucobrassicin). Therefore, the present study was undertaken to investigate the protective role of DIM against DOX-induced toxicity in mice. DOX was administered (5 mg/kg b.W, i.p.) and DIM was administered (25 mg/kg b.W p.o.) in concomitant and 15-day pretreatment schedule. Results showed that DIM significantly attenuated DOX-induced oxidative stress in the cardiac tissues by reducing the levels of free radicals and lipid peroxidation, and by enhancing the level of glutathione (reduced) and the activity of antioxidant enzymes. The chemoprotective potential of DIM was confirmed by histopathological evaluation of heart and bone marrow niche. Moreover, DIM considerably mitigated DOX-induced clastogenicity, DNA damage, apoptosis and myeloid hyperplasia in bone marrow niche. In addition, oral administration of DIM significantly ($p < 0.05$) stimulated the Nrf2-mediated activation of antioxidant response element (ARE) pathway and promoted expression of ARE-driven cytoprotective proteins, HO-1, NQO1 and glutathione-S-transferase (GST). In connection with that, DIM significantly attenuated DOX-induced apoptosis by upregulation of Bcl-2 expression and downregulation of Bax and caspase-3 expression. Thus, this study suggests that DIM has promising chemoprotective efficacy against DOX-induced toxicity and indicates its future use as an adjuvant in chemotherapy.

DIM(Diindolylmethane) - Apoptosis in breast cancer



Tamoxifen

- Used in pre menopausal women to block estrogen levels.
 - Estrogen levels are higher in menstruating women and decline as women age and go through menopause

DIM (Diindolylmethane) with tamoxifen

Breast Cancer Res Treat. 2017 Aug;165(1):97-107. doi: 10.1007/s10549-017-4292-7. Epub 2017 May 30.

A randomized, placebo-controlled trial of diindolylmethane for breast cancer biomarker modulation in patients taking tamoxifen.

Thomson CA^{1,2}, Chow HHS³, Wertheim BC³, Roe DJ^{3,4}, Stopeck A⁵, Maskarinec G⁶, Altbach M⁷, Chalasanani P³, Huang C⁸, Strom MB⁹, Galons JP^{3,7}, Thompson PA^{5,10}.

Author information

Abstract

PURPOSE: Diindolylmethane (DIM), a bioactive metabolite of indole-3-carbinol found in cruciferous vegetables, has proposed cancer chemoprevention activity in the breast. There is limited evidence of clinically relevant activity of DIM or long-term safety data of its regular use. A randomized, double-blind, placebo-controlled trial was conducted to determine the activity and safety of combined use of BioResponse DIM® (BR-DIM) with tamoxifen.

METHODS: Women prescribed tamoxifen (n = 130) were randomly assigned oral BR-DIM at 150 mg twice daily or placebo, for 12 months. The primary study endpoint was change in urinary 2/16 α -hydroxyestrone (2/16 α -OHE1) ratio. Changes in 4-hydroxyestrone (4-OHE1), sex estrogens, sex hormone-binding globulin (SHBG), breast density, and tamoxifen metabolites were assessed.

RESULTS: Ninety-eight women (51 placebo, 47 DIM) completed intervention; compliance with treatment was >91%. BR-DIM increased the 2/16 α -OHE1 ratio (+3.2 [0.8, 8.4]) compared to placebo (-0.7 [-1.7, 0.8], P < 0.001). Serum SHBG increased with BR-DIM compared to placebo (+25 \pm 22 and +1.1 \pm 19 nmol/L, respectively). No change in breast density measured by mammography or by MRI was observed. Plasma tamoxifen metabolites (endoxifen, 4-OH tamoxifen, and N-desmethyl-tamoxifen) were reduced in women receiving BR-DIM versus placebo (P < 0.001). Minimal adverse events were reported and did not differ by treatment arm.

CONCLUSION: In patients taking tamoxifen for breast cancer, daily BR-DIM promoted favorable changes in estrogen metabolism and circulating levels of SHBG. Further research is warranted to determine whether BR-DIM associated decreases in tamoxifen metabolites, including effects on endoxifen levels, attenuates the clinical benefit of tamoxifen.

TRIAL REGISTRATION: ClinicalTrials.gov NCT01391689.

Green tea and Broccoli sprouts(sulphoraphanes)

[Sci Rep.](#) 2017 Aug 24;7(1):9345. doi: 10.1038/s41598-017-09764-3.

Combinatorial bioactive botanicals re-sensitize tamoxifen treatment in ER-negative breast cancer via epigenetic reactivation of ER α expression.

[Li Y](#)^{1,2,3}, [Meeran SM](#)⁴, [Tollefsbol TO](#)^{5,6,7,8}.

Author information

Abstract

Conventional cancer prevention has primarily focused on single chemopreventive compounds that may not be sufficiently efficacious. We sought to investigate potential combinatorial effects of epigenetic bioactive botanicals including epigallocatechin-3-gallate (EGCG) in green tea polyphenols (GTPs) and sulforaphane (SFN) in broccoli sprouts (BSp) on neutralizing epigenetic aberrations in estrogen receptor- α (ER α) leading to enhanced anti-hormone therapeutic efficacy in ER α -negative breast cancer. Our results showed that this combinatorial treatment re-sensitized ER α -dependent cellular inhibitory responses to an estrogen antagonist, tamoxifen (TAM), via at least in part, epigenetic reactivation of ER α expression in ER α -negative breast cancer cells. Further in vivo studies revealed the combinatorial diets of GTPs and BSp significantly inhibited breast tumor growth in ER α -negative mouse xenografts, especially when combined with TAM treatment. This novel treatment regimen can lead to remodeling of the chromatin structure by histone modifications and recruitment changes of transcriptional factor complex in the ER α promoter thereby contributing to ER α reactivation and re-sensitized chemotherapeutic efficacy of anti-hormone therapy. Our studies indicate that combinatorial bioactive botanicals from GTPs and BSp are highly effective in inhibiting ER α -negative breast cancer due at least in part to epigenetic reactivation of ER α , which in turn increases TAM-dependent anti-estrogen chemosensitivity in vitro and in vivo.

Melatonin and Tamoxifen

— — —

- Melatonin, 20 mg/day at bedtime, with tamoxifen, 20 mg/day at noon, improved clinical status in 28% of patients with metastatic breast cancer unresponsive to tamoxifen alone.
- Melatonin augments sensitivity of breast cancer cells to tamoxifen in tissue culture
- Melatonin interferes with activation of the estrogen receptor by estradiol and also inhibits aromatase

Foods that enhance the effectiveness of Tamoxifen treatment

Arctic char	Carrots	Grapes & grape juice, red	Parsley
Artichokes	Cauliflower	Green tea	Pomegranates & pomegranate juice
Arugula	Celery & celery hearts	Herring	Pumpkins
Bell peppers	Cherries, especially sour or tart	Horseradish & wasabi	Rice, brown, black or purple
Black cumin	Chicken, organic, but not chicken livers	Hot peppers	Salmon, wild
Black pepper	Chives	Kale	Sardines
Blackberries	Cilantro	Kefir	Seaweed, brown
Blueberries & bilberries	Coffee	Lake trout	Strawberries
Bok choy	Collard greens	Lemons	Tomatoes
Broccoli & broccoli sprouts	Cranberries & lingonberries	Limes	Turkey, organic
Brussels sprouts	Currants, black	Mackerel	Turmeric
Buckwheat	Flaxseed & flaxseed oil	Mushrooms	Turnips & turnip greens
Butternut squash	Garlic, fresh	Mustard	Walnuts & walnut oil
Cabbage	Ginger	Mustard greens	Watercress & garden cress
Cantaloupe		Olive oil, extra-virgin & olives	Zucchini
		Onions, green or red	

Foods that decrease the risk of endometrial cancer

--- Tamoxifen acts as an anti estrogen in breast tissue but acts like estrogen in the uterus. This can result in a thickened uterine lining and potentially endometrial hyperplasia or cancer. This risk is low, but still a risk.

Arugula
Bell peppers
Black pepper
Black tea
Brazil nuts
Broccoli
Brussels
sprouts
Carrots
Cherries, sour
Collard greens

Garlic
Green tea
Herring
Honey, (minimally
processed*)
Hot peppers
Kale
Lake trout
Leeks
Lettuce, romaine

Mackerel
Mustard greens
Onions
Pomegranates &
pomegranate
juice
Pumpkins
Raspberries,
especially black
Saffron
Salmon, wild

Sardines
Seaweed
Tomatoes
Turnip greens
Walnut oil
Watercress
Watermelon
Zucchini

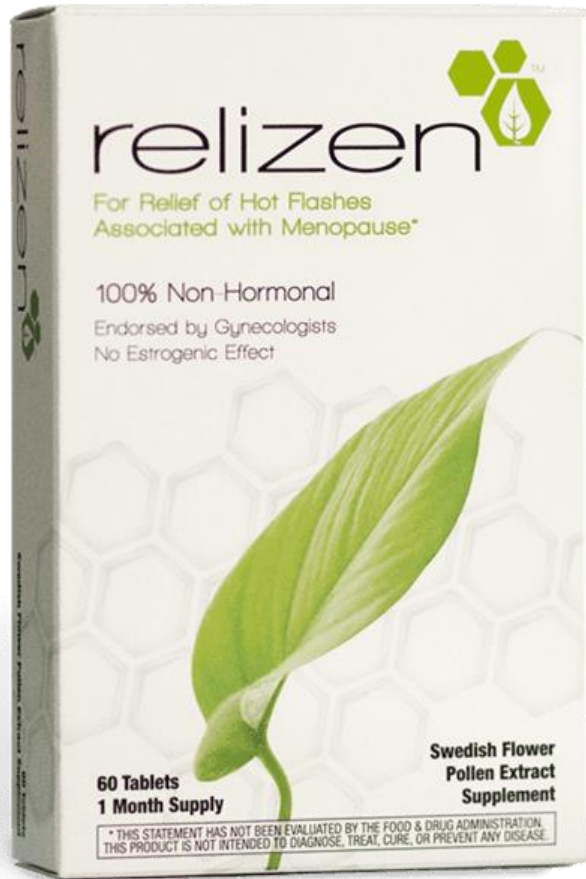
Tamoxifen and Sugar

- Tamoxifen can increase the risk of Type II Diabetes.
- Therefore, sugar and high processed carbohydrates are contraindicated with Tamoxifen use.



Relizen

Swedish flower pollen extract



Does purified Swedish pollen extract, a nonhormonal treatment for vasomotor symptoms, inhibit the CYP2D6 enzyme system?

[Goldstein SR](#)¹, [Espíe M](#), [Druckmann R](#).

RESULTS: Inhibition of CYP2D6 with purified Swedish pollen extract was negligible at all concentrations and ranged from -6.53% to 10.67%. Inhibition of CYP2D6 enzyme with Quinidine increased in a linear dose-related fashion from -7.07% at 2.06 nM to 84.05% at 500 nM.

CONCLUSIONS: Purified Swedish pollen extract is a nonhormonal treatment of VMS that does not show inhibition of the CYP2D6 enzyme. This may have important clinical utility for women using tamoxifen for breast cancer treatment or chemoprevention who experience VMS.

Aromatase Inhibitors

Arimidex (Anastrozole), Femara (Letrozole) , Aromasin (exemestane)

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Aromatase inhibitors are commonly used in menopausal women to reduce the conversion of androgen hormones into estrogen.

Foods that enhance the effectiveness of aromatase inhibitors

— — —

- | | | |
|-----------------------------|--------------------------------------|-------------------------------------|
| Arugula | Collard greens | Olive oil, extra-virgin |
| Beans, dry | Cranberries & ligoberries | Olives |
| Bell peppers | Flaxseed & flaxseed oil,
organic | Parsley |
| Blackberries | Ginger | Pomegranates &
pomegranate juice |
| Blueberries & bilberries | Grapes and grape juice, red | Pumpkins |
| Boysenberries | Horseradish & wasabi | Raspberries |
| Broccoli & broccoli sprouts | Hot peppers | Rice, black, red or purple |
| Brussels sprouts | Kale | Tomatoes |
| Butternut squash | Mushrooms, white button
& related | Turnip greens |
| Cabbage | Mustard | Turnips |
| Carrots | Mustard greens | Walnuts & walnut oil |
| Cauliflower | | Watercress |
| Celery | | Zucchini |
| Cherries, sour | | |
| Cilantro | | |

Foods, supplements and other to avoid while taking aromatase inhibitors

— — —

Alcohol

Citrus peel

Corn oil

Daidzein

Genistein

Goldenseal supplements

Grapefruit & grapefruit juice

Hesperetin or hesperidin supplements

Hormone replacement therapy

Peanut oil

Safflower oil

Soybean oil

Soybean paste

Soy protein isolate

St. John's Wort

Sunflower oil

* Cigarette smoking has also been found to sharply reduce the effectiveness of treatment with aromatase inhibitors.

Foods that safely reduce the side effects of aromatase inhibitors

— — —

Arctic char - bone loss

Mackerel - bone loss

Black tea - bone loss

Olive oil - arthritis

Blackberries - bone loss

Oranges - bone loss

Carrots - bone loss

Plums - bone loss

Cherries, sour - joint pain

Pomegranate - arthritis, bone loss

Dry beans - bone loss

Prunes - bone loss

Ginger - joint pain

Raspberries - arthritis

Green tea - bone loss

Salmon, wild - bone loss

Herring - bone loss

Sardines - bone loss

Kale - bone loss

Walnuts - bone loss

Lake trout - bone loss

Detoxification- Many people like to cleanse but be very careful as toxins leaving are as dangerous as they are coming

How long does it take for the immune system to recover after chemo?

Treatment can last for anywhere from **3 to 6 months**. During that time, you would be considered to be immunocompromised — not as able to fight infection. After finishing chemotherapy treatment, it can take anywhere from about 21 to 28 days for your immune system to recover and sometimes much longer.

However it can take up to **six months** to recover from chemotherapy. Chemotherapy is out of your system a number of hours after you've had it given but the side effects can take up to **about six months**. Your organs would recover a lot quicker than that.

Sulphoraphanes

J Agric Food Chem. 2016 Jul 13;64(27):5515-24. doi: 10.1021/acs.jafc.6b02195. Epub 2016 Jun 29.

Sulforaphene Interferes with Human Breast Cancer Cell Migration and Invasion through Inhibition of Hedgehog Signaling.

Bao C¹, Kim MC¹, Chen J¹, Song J², Ko HW², Lee HJ¹.

Author information

Abstract

Although inhibition of mammary tumorigenesis by isothiocyanates has been widely studied, little is known about the effects of sulforaphene on invasiveness of breast cancer. Here, sulforaphene significantly inhibited the migration and invasion of triple-negative SUM159 human breast cancer cells and suppressed the expression and activity of matrix metalloproteinases 2 and 9 (MMP-2 and MMP-9). The Hedgehog (Hh) pathway, as an upstream signaling modulator, was significantly suppressed by sulforaphene. In particular, ciliary localization of Gli1 and its nuclear translocation were blocked by sulforaphene in a time-dependent manner. Consistently, downregulation of Hh signaling by vismodegib and Gli1 knockdown reduced the cellular migration and invasion as well as the expression of MMP-2 and MMP-9. These results indicate that the suppression of Hh/Gli1 signaling by sulforaphene may reduce the MMP-2 and MMP-9 activities and cellular invasiveness of human breast cancer cells, suggesting the potential efficacy of sulforaphene against breast cancer invasion and metastasis.

Fear is a useless emotion, but it happens to the best of us.

– Once you are over the fear it is time to be pragmatic, do your research and build a plan.

Be gentle on yourself and make time for self care during this time in your life.

Keep good love, spirituality, and joy in your journey

Remember, healthy people get cancer too

“Life is 10% what happens to us and 90% how we react to it.”