

GENOME Privacy

The New York Times

Most White Americans' DNA Can Be Identified Through Genealogy Databases



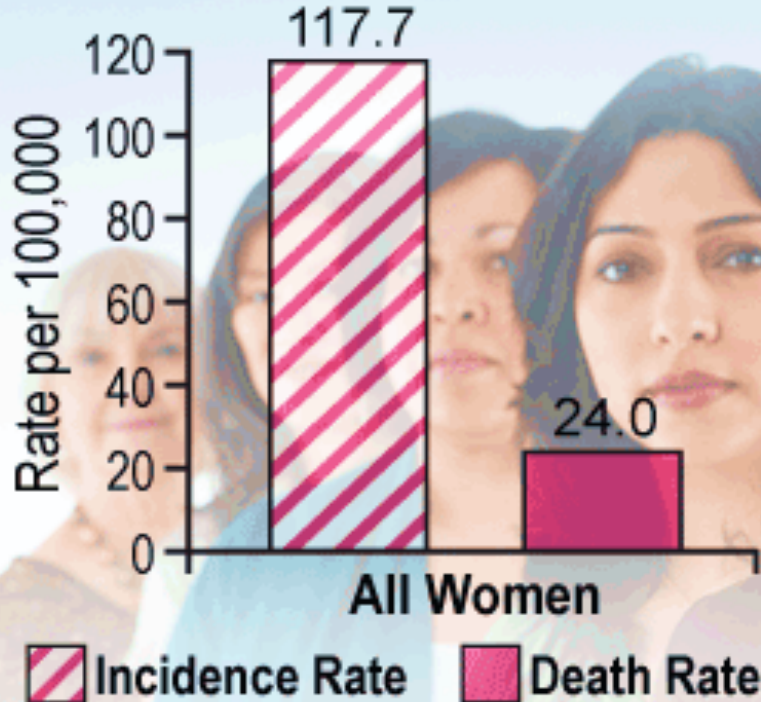
Only two percent of the population needs to have done a DNA test to identify nearly everyone else, researchers found. Leonard Lessin/Science Source



Breast Cancer I

Breast Cancer Statistics

Female Breast Cancer Incidence and Death Rates, U.S.



- ❑ 1 in 8 women in the US (12 to 13%) will develop invasive breast cancer over the course of her lifetime
- ❑ In 2010, ~ **207,090** new cases of invasive breast cancer were diagnosed in women in the U.S., along with **54,010** new cases of non-invasive (in situ) breast cancer.
- ❑ About **1,970** new cases of invasive breast cancer was diagnosed in men in 2010. Less than 1% of all new breast cancer cases occur in men.

Breast Cancer Incidence and Mortality by Race and Ethnicity



Risk Factors for Breast Cancer

Factor	Relative risk	High risk group
Age	>10	Elderly
Geographical location	5	Developed country
Age at menarche	3	Menarche before age 11
Age at menopause	2	Menopause after age 54
Age at first full pregnancy	3	First child in early 40s
Family history:	2	Breast cancer in first degree relative when young
Previous benign disease	4-5	Atypical hyperplasia
Cancer in other breast	>4	
Socioeconomic group	2	Groups I and II
Diet	1.5	High intake of saturated fat
Body weight:		
Premenopausal	0.7	Body mass index >35
Postmenopausal	2	Body mass index >35
Alcohol consumption	1.3	Excessive intake
Exposure to ionising radiation	3	Abnormal exposure in young females after age 10
Taking exogenous hormones:		
Oral contraceptives	1.24	Current use
Hormone replacement therapy	1.35	Use for > 10 years
Diethylstilbestrol	2	Use during pregnancy



What do I look for?

Don't think of a breast self-exam as a hunt to find cancer, but to understand the normal landscape of your breasts. This involves both looking and feeling.

Look in the mirror with your arms up, then again with your hands on your hips. Stand to the side and then face forward. Check for any changes that look like these signs below. →

What does a cancerous lump feel like?

A cancerous lump often feels hard and immovable, like a lemon seed. 80% of lumps are not cancerous, but the only way to know for sure is to examine the cells inside. If you feel a lump ask a doctor to investigate it with you. When breast cancer is caught at early stages the cure rate is over 90%!

When should I do it?

From the age of twenty onwards, do self-exams at least once a season to stay abreast of your health!

The best time to check your breasts is a few days after your period ends when hormones are the most normal and you're least tender (which is also an ideal time to have a doctor's breast exam and mammogram).

If you don't have a period anymore pick any day.

A dozen signs of breast cancer, revealed.

Clinical Breast Exams

begin at age 20 with your doctor every 3 years until you are 40, then go every year. Higher risk patients may visit more, ask your doctor about your risk type.



Mammograms

usually begin when you're 40. It feels like getting your blood pressure taken. Form a screening plan with your doctor to suit your risk type.

If you're too young for a mammogram and suspect something, ask about an ultrasound or biopsy, don't wait it out.

Want to calculate your risk? www.Lemonland.org

Are you 100% screened this year?

Recommended screening paths for Women in the U.S.

Get a mammogram every year.
Risk increases with age.

Mammograms for "high risk" women.

Visit your doctor every year.

Visit your doctor every 1-3 years.

20

Starting age

30

40

50

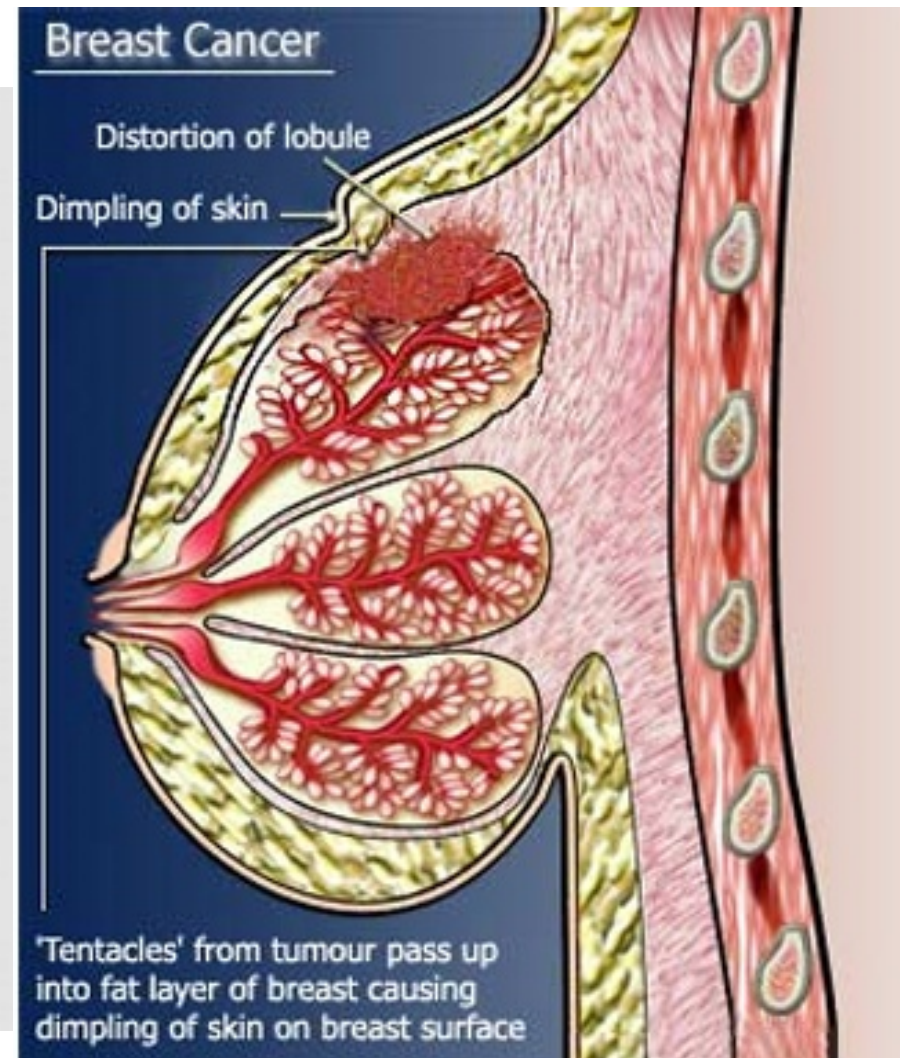
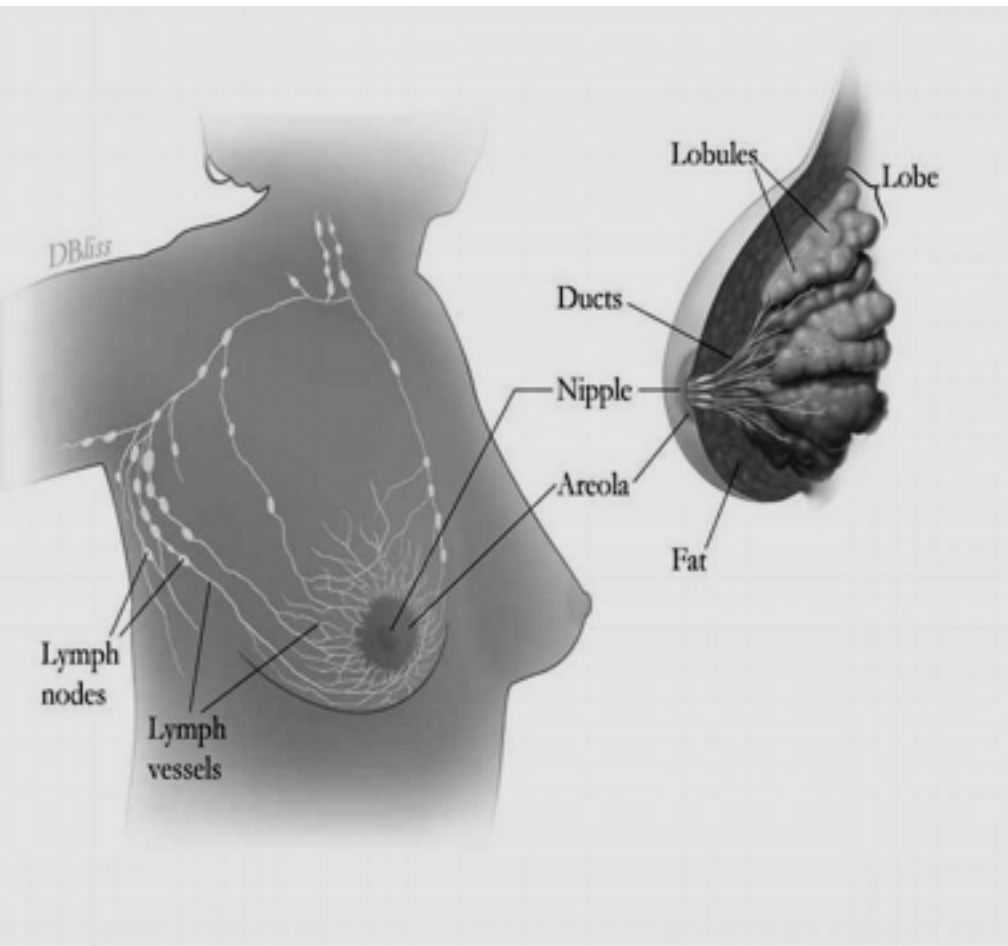
60

70+

You should always
know what's normal.
Look and feel once a season.
If something seems odd, visit your Dr.

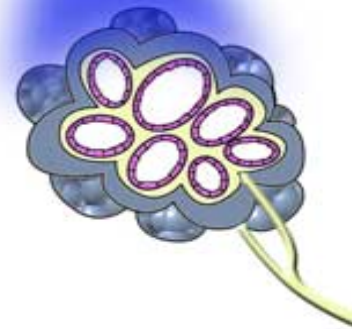
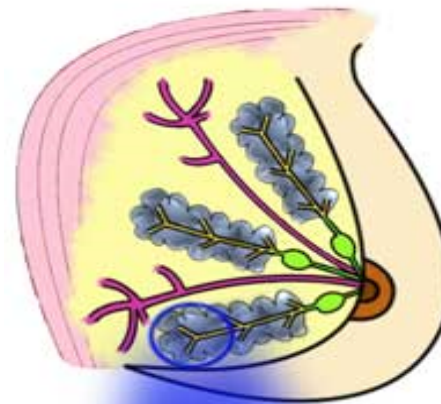
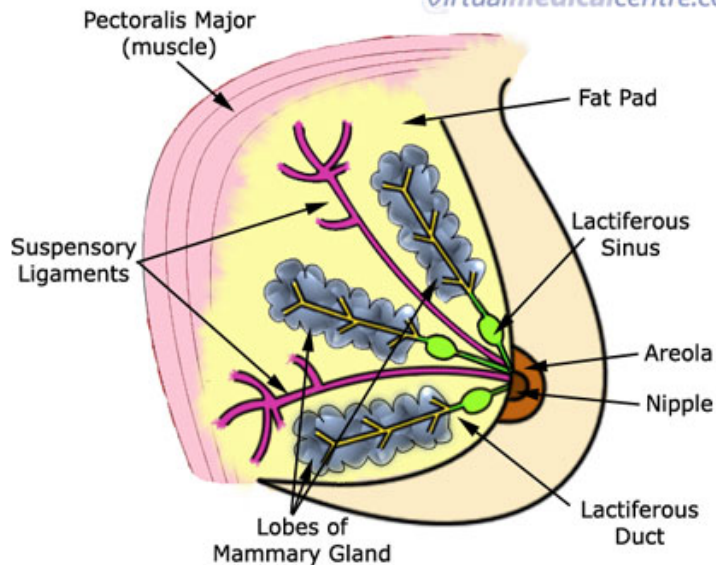
Compiled data from the US National Cancer Institute and other cancer research bodies. Women at "high risk" should consult a physician.
For more information about risk, screening and what you can do, visit Lemonland.org

Anatomy of the Breast/ Breast Cancer

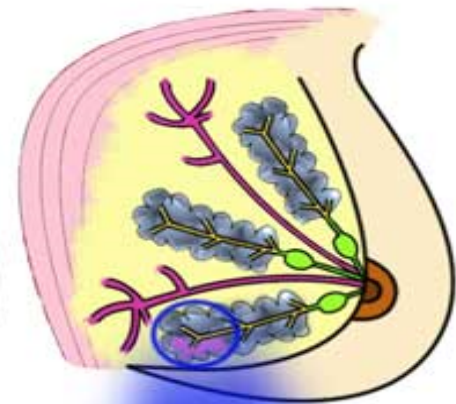


Noninvasive Breast Cancer

virtualmedicalcentre.com®



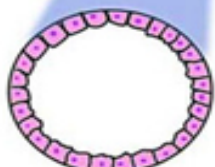
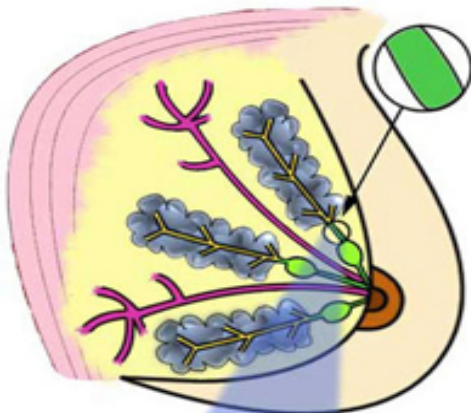
Normal Lobule



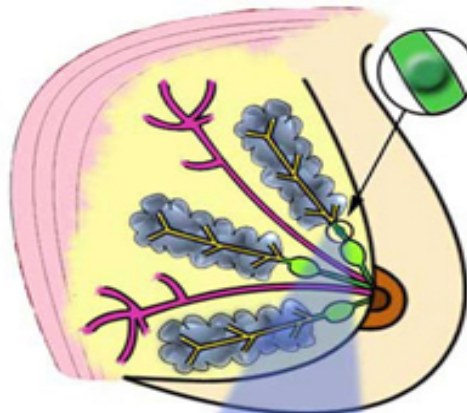
Lobular Carcinoma In Situ

virtualmedicalcentre.com®

For patients with LCIS the risk for developing invasive cancer in the same breast is about 18% -- and 14% in the other breast -- after 20 years



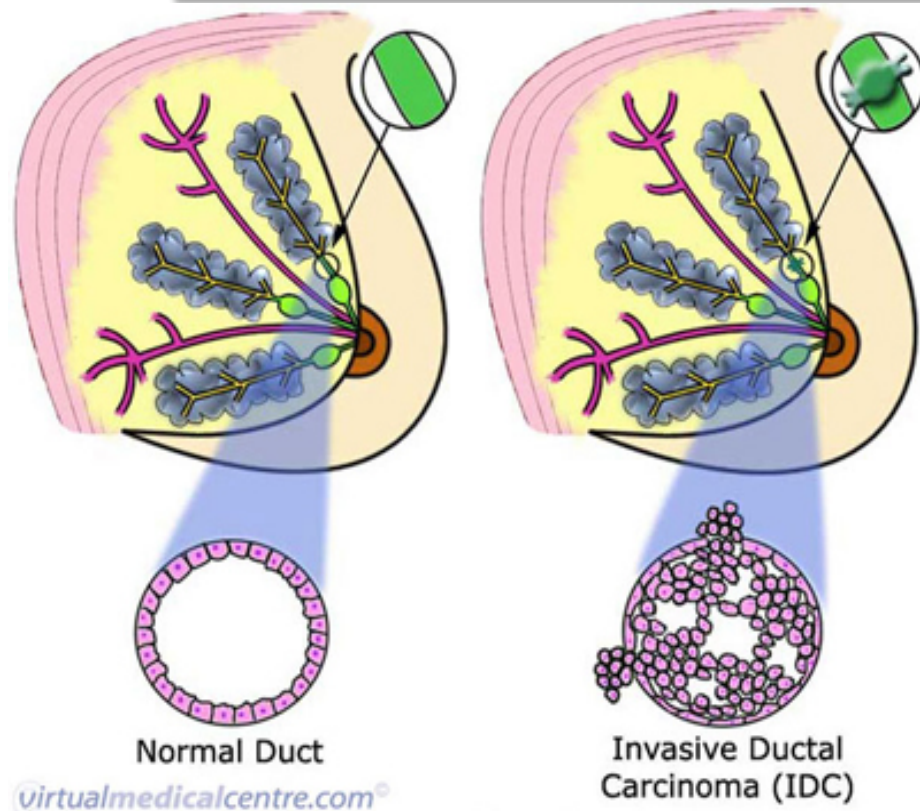
Normal Duct



Ductal Carcinoma In Situ (DCIS)

virtualmedicalcentre.com®

Invasive Ductal Carcinoma (IDC)



❑ **Invasive Ductal Carcinoma (IDC):** This is invasive breast cancer that penetrates the wall of a duct. It comprises between 70 - 80% of all breast cancer cases.

How Breast Cancer Metastasizes

Infiltrating Ductal Carcinoma (IDC) is an invasive malignancy which has spread beyond the ducts. It accounts for almost 80 percent of all invasive breast cancers. Here's how IDC develops in the milk ducts and spreads to other areas of the body:

Lymphatic vessel

Lymph nodes are groups of immune system cells

Milk duct

Nipple

Lobules

Blood vessel

Fatty tissue

MILK DUCT

Ducts are the tiny tubes that carry milk produced from the lobules to the nipple.

Blood vessel

Milk duct

Lymphatic vessel

ABNORMAL GROWTH

Changes in genes cause cells to divide uncontrollably.

Cancer cells

METASTASIS

The cancer cells grow in the milk duct until they break the membrane lining the duct and move into the surrounding tissue.

SECONDARY SITES

Infiltrating Ductal Carcinoma spreads most commonly to these areas.

Brain

Lungs

Liver

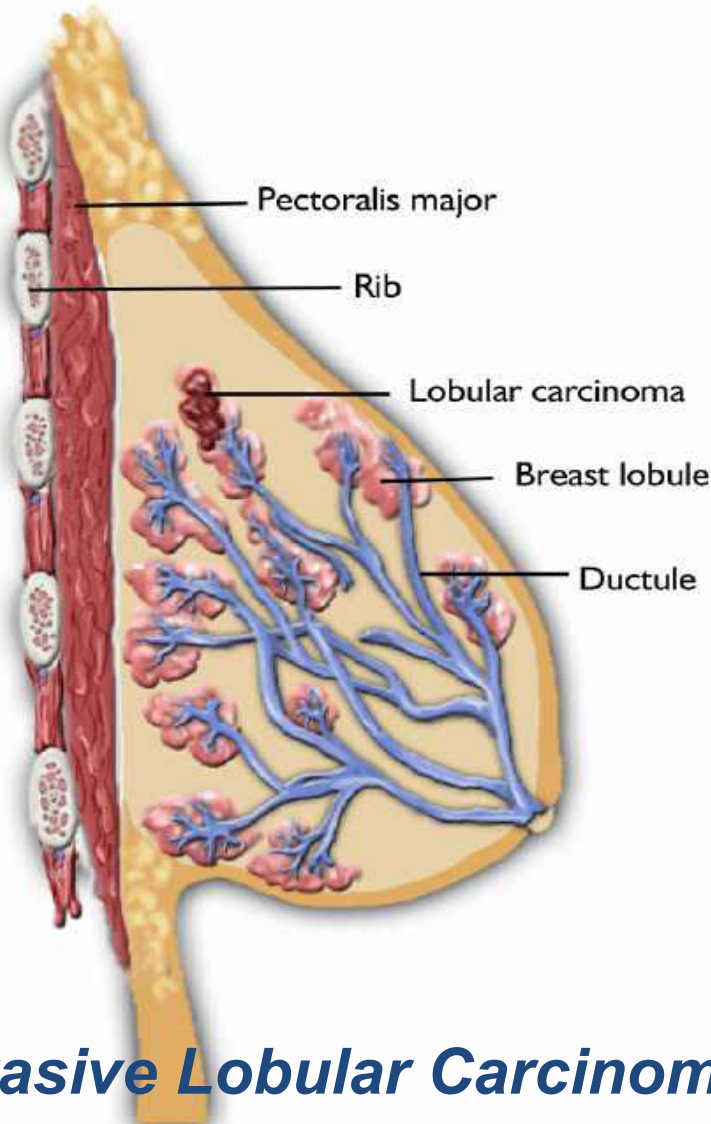
Lymph nodes

Bones

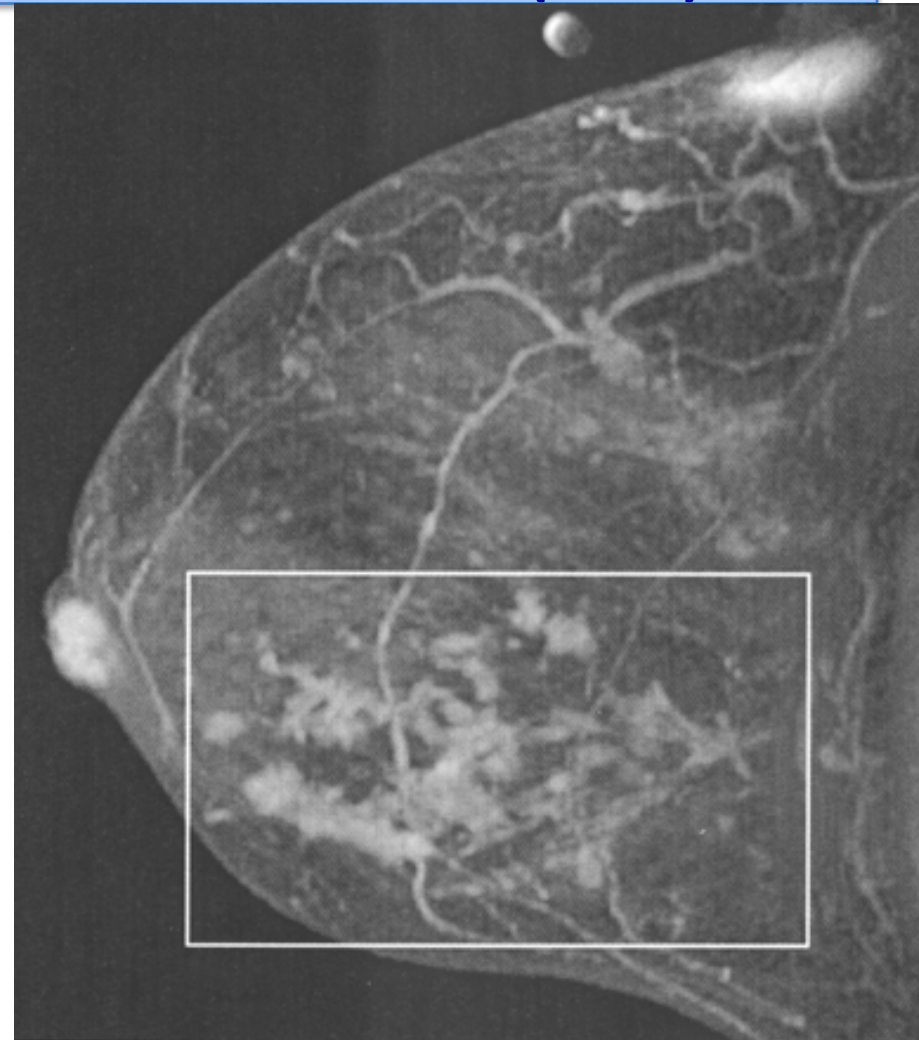
Cancer cells enter the lymphatic vessels and travel to other areas of the body.

Invasive Lobular Carcinoma (ILC)

Lobular carcinoma of the breast



Invasive Lobular Carcinoma (ILC): Accounts for 10 - 15% of all breast cancers.



55-year-old woman with mid-grade invasive lobular carcinoma. Note diffuse enhancement of lesion (*white box*) on maximum-intensity-projection MR image obtained before chemotherapy.

Watchful waiting for DCIS

TIME

10/2/2015 “Basila sat back down, and as their meeting reached the hour mark, she made a choice that humans are practically hardwired not to make in the face of a cancer diagnosis: she decided to do nothing.

Well, not nothing, exactly. She would start taking a drug called tamoxifen that blocks estrogen, which can fuel tumor growth, and she would enroll in a clinical trial involving **active surveillance**: twice-a-year visits in which she would get mammograms alternating with MRIs. As long as there were no worrisome changes, Basila would be spared the standard arsenal in breast-cancer treatment: surgery, radiation and chemotherapy”

Watchful waiting = active surveillance

Tamoxifen = estrogen blocker = antiestrogen

That conversation took place eight years ago. And if it sounds radical today, it was all but heresy back then.

This was before the U.S. Preventive Services Task Force said in 2009 that women should start mammograms at 50, not the previous guideline of 40, because there's insufficient evidence that earlier screening does more good than harm.

Before research showed that for some women with Stages 1 and 2 breast cancer, the absolute survival benefit from preventive double mastectomies is less than 1% after 20 years.

Before the paper in August showing that no matter how a woman is treated for DCIS, the mortality risk is 3%—similar to the average for the general population. And before the news that some women with early-stage breast cancer don't benefit from chemo and can skip it.

- **Screening age effectiveness controversy**
- **Double mastectomies, DCIS same as no treatment**

N Engl J Med. 2002 Oct 17;347(16):1227-32.

Twenty-year follow-up of a randomized study comparing breast-conserving surgery with radical mastectomy for early breast cancer.

Veronesi U¹, Cascinelli N, Mariani L, Greco M, Saccozzi R, Luini A, Aguilar M, Marubini E.

 Author information

Abstract

BACKGROUND: We conducted 20 years of follow-up of women enrolled in a randomized trial to compare the efficacy of radical (Halsted) mastectomy with that of breast-conserving surgery.

METHODS: From 1973 to 1980, 701 women with breast cancers measuring no more than 2 cm in diameter were randomly assigned to undergo radical mastectomy (349 patients) or breast-conserving surgery (quadrantectomy) followed by radiotherapy to the ipsilateral mammary tissue (352 patients). After 1976, patients in both groups who had positive axillary nodes also received adjuvant chemotherapy with cyclophosphamide, methotrexate, and fluorouracil.

CONCLUSIONS: The long-term survival rate among women who undergo breast-conserving surgery is the same as that among women who undergo radical mastectomy. Breast-conserving surgery is therefore the treatment of choice for women with relatively small breast cancers.

Use of and Mortality After Bilateral Mastectomy Compared With Other Surgical Treatments for Breast Cancer in California, 1998-2011

Allison W. Kurian, MD, MSc^{1,2}; Daphne Y. Lichtensztajn, MD, MPH³; Theresa H. M. Keegan, PhD^{2,3}; David O. Nelson, PhD^{2,3}; Christina A. Clarke, PhD^{2,3}; Scarlett L. Gomez, PhD^{2,3}

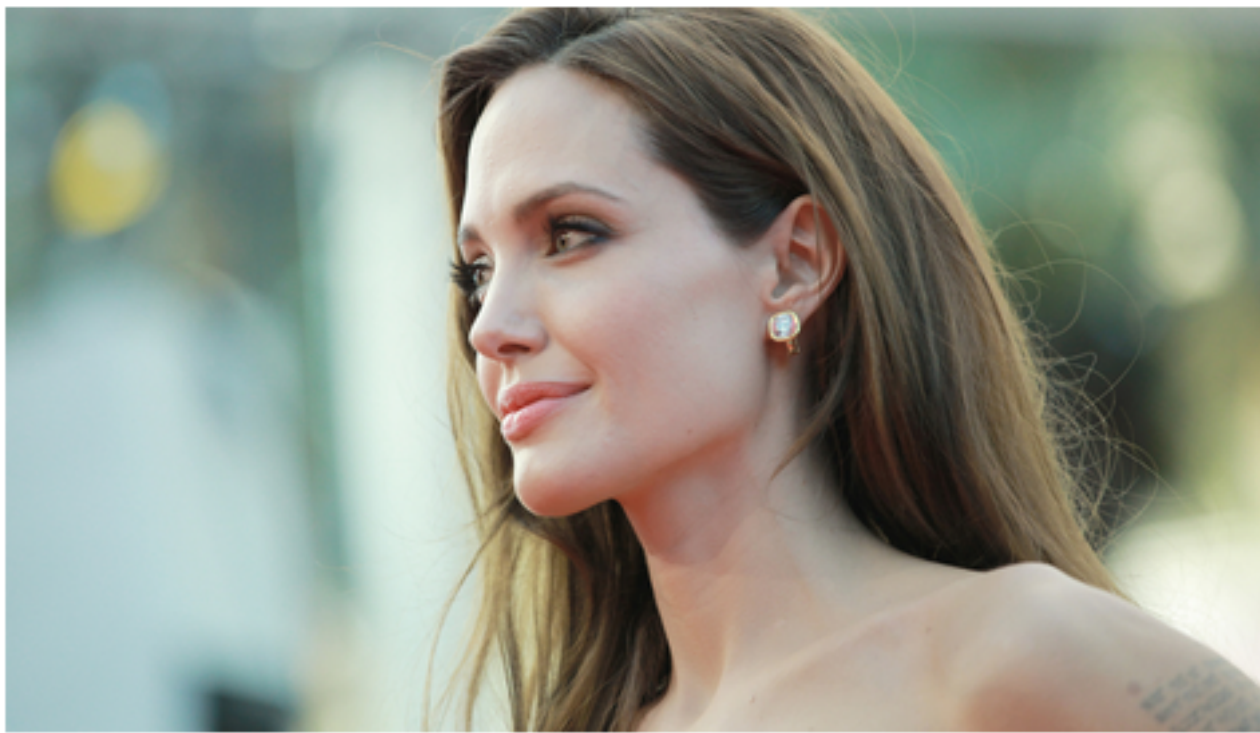
[\[+\] Author Affiliations](#)

JAMA. 2014;312(9):902-914. doi:10.1001/jama.2014.10707.

Text Size: [A](#) [A](#) [A](#)

CI, 36.3%-36.8%)). Compared with breast-conserving surgery with radiation (10-year mortality, 16.8% [95% CI, 16.6%-17.1%]), unilateral mastectomy was associated with higher all-cause mortality (hazard ratio [HR], 1.35 [95% CI, 1.32-1.39]; 10-year mortality, 20.1% [95% CI, 19.9%-20.4%]). There was no significant mortality difference compared with bilateral mastectomy (HR, 1.02 [95% CI, 0.94-1.11]; 10-year mortality, 18.8% [95% CI, 18.6%-19.0%]). Propensity analysis showed similar results.

Conclusions and Relevance Use of bilateral mastectomy increased significantly throughout California from 1998 through 2011 and was not associated with lower mortality than that achieved with breast-conserving surgery plus radiation. Unilateral mastectomy was associated with higher mortality than were the other 2 surgical options.

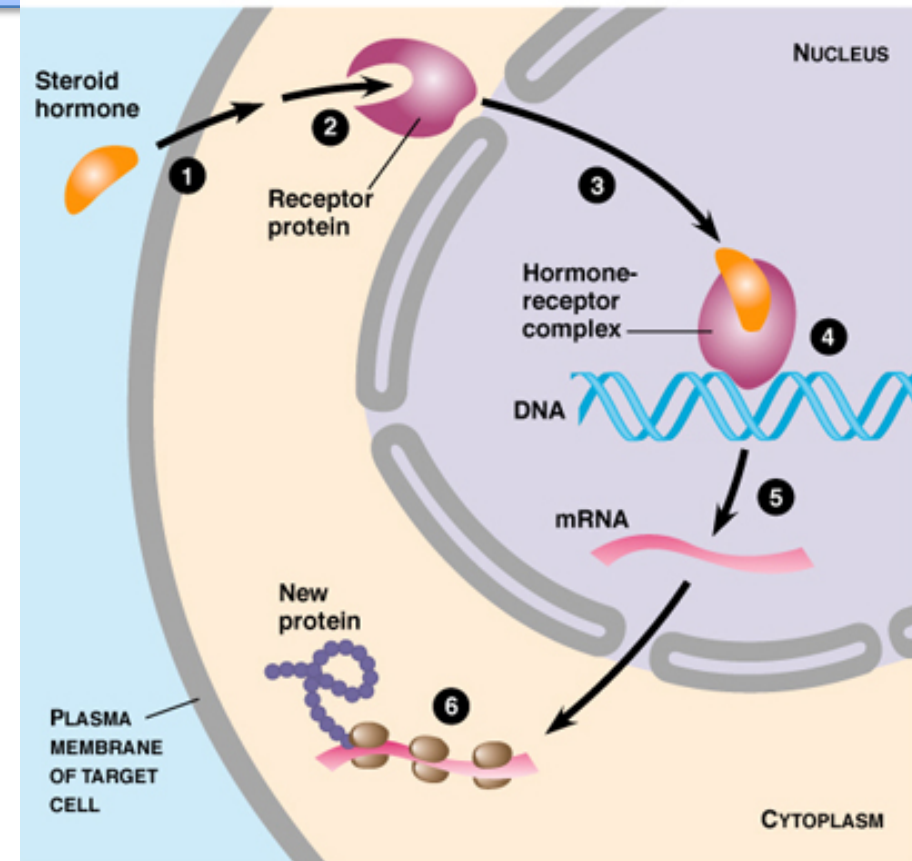
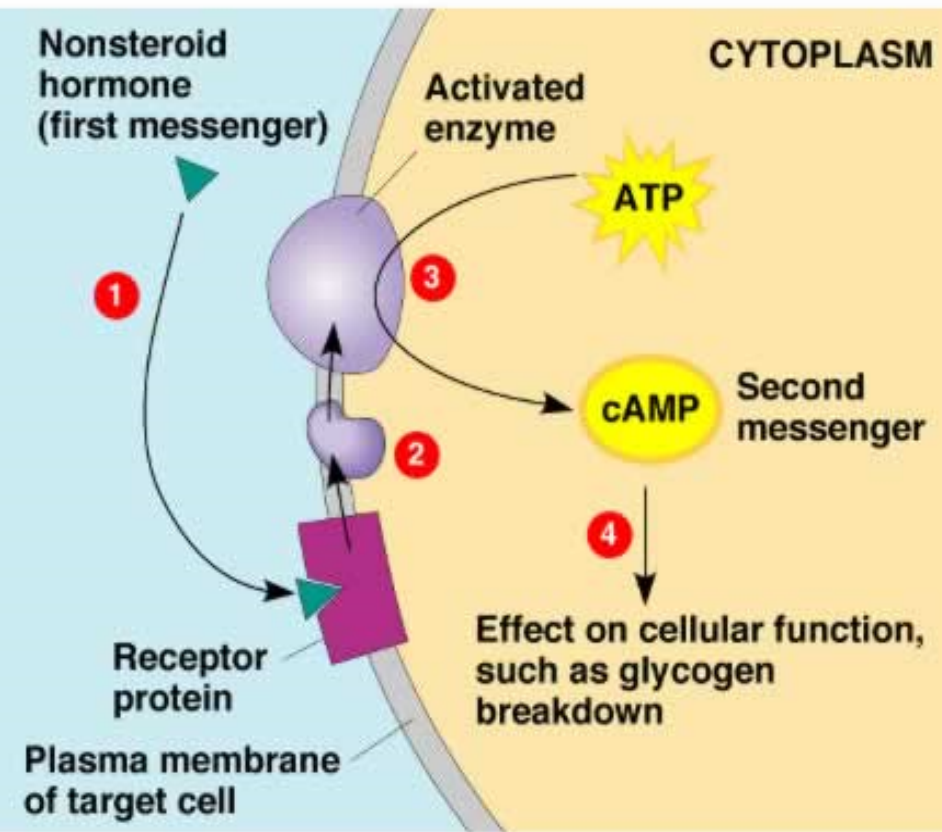


PAN Photo Agency / Shutterstock.com

When Angelina Jolie announced in the *New York Times*, in May of last year, that she'd undergone a **radical mastectomy**, she wrote, "Life comes with many challenges. The ones that should not scare us are the ones we can take on and take control of." Her bravery was widely applauded, and she **became** "the new, gorgeous poster woman" for the procedure, which she chose to have because, like her **mother**, who died at age 56 from cancer, she carries the BRCA1 gene that increases the risks of both ovarian and breast cancer. In the wake of her announcement came a rash of **news stories** about other women—some as **young as 21**—who'd opted for the surgery, along with tweets by breast cancer survivors calling Jolie "so brave" (**Sheryl Crow**) and "admirable," among other accolades.

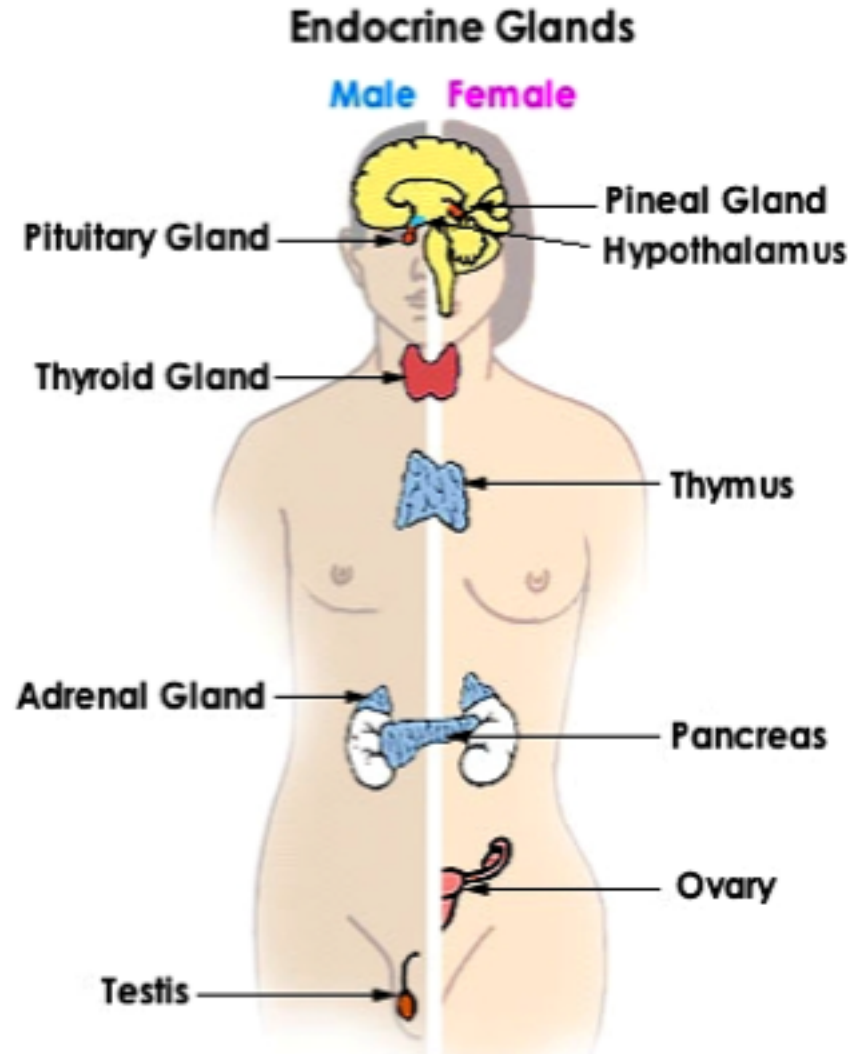
- **Carrier risk 75-82%**
- **Studies confirm the effect on number of women being tested for BRCA mutations has been sustained**

Protein vs Steroid Hormones



What are Hormones?

- ❑ A hormone (from Greek *horman* - "to set in motion") is a chemical messenger from one cell (or group of cells) to another
- ❑ Function as a signal to the target cells
- ❑ Actions can include stimulation or inhibition of growth, induction or suppression of apoptosis, activation or inhibition of the immune system, regulating metabolism and preparation for a new activity or phase of life





Effects of Estrogen

Brain

Estrogen helps to maintain body temperature.

Estrogen may delay memory loss.

Estrogen helps to regulate parts of the brain that prepare the body for sexual and reproductive development.

Heart & Liver

Estrogen helps to regulate the liver's production of cholesterol, thus decreasing the build-up of plaque in the coronary arteries.

Ovary

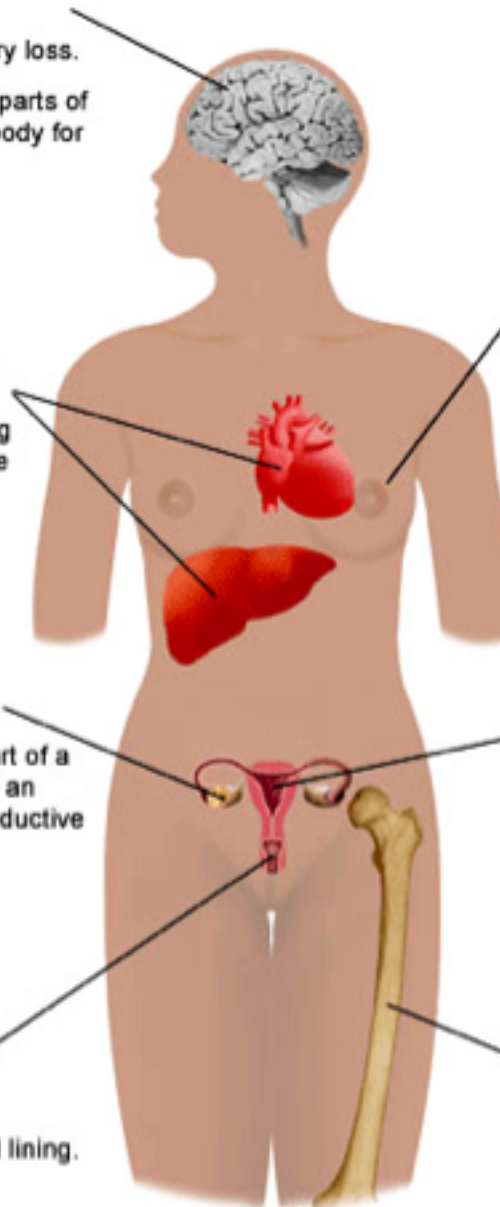
Estrogen stimulates the maturation of the ovaries.

Estrogen stimulates the start of a woman's menstrual cycle – an indication that a girl's reproductive system has matured.

Vagina

Estrogen stimulates the maturation of the vagina.

Estrogen helps maintain a lubricated and thick vaginal lining.



Breast

Estrogen stimulates the development of the breasts at puberty and prepares the glands for future milk production.

Uterus

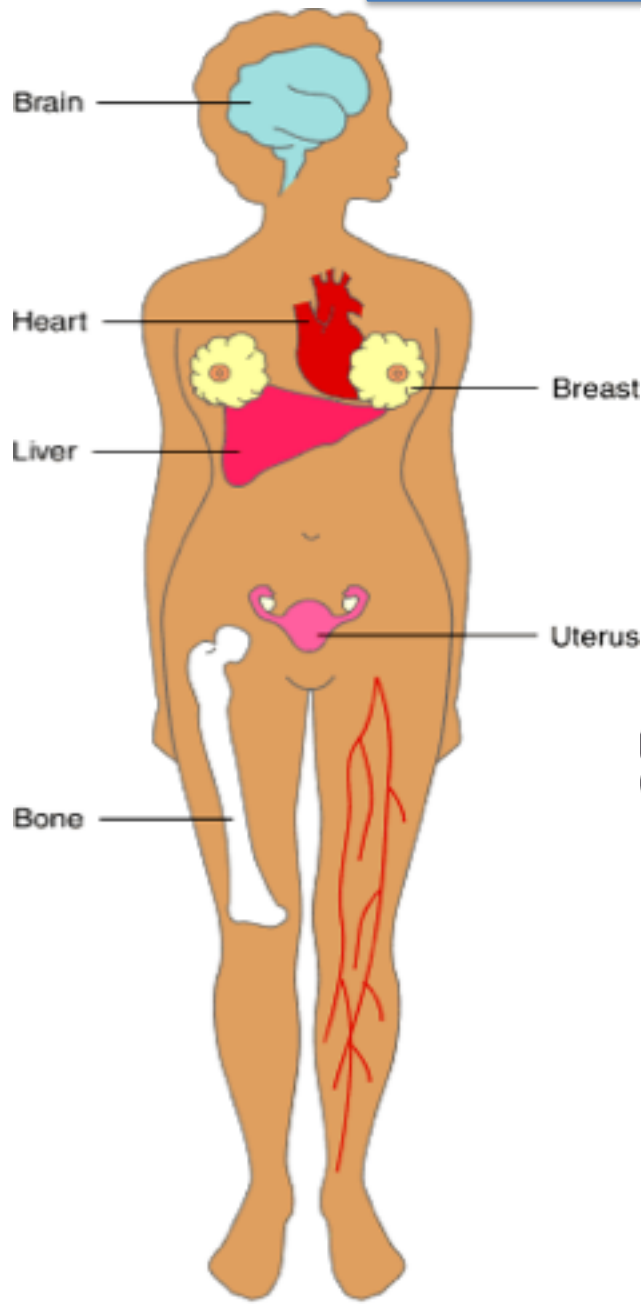
Estrogen stimulates the maturation of the uterus.

Estrogen helps to prepare the uterus to nourish a developing fetus.

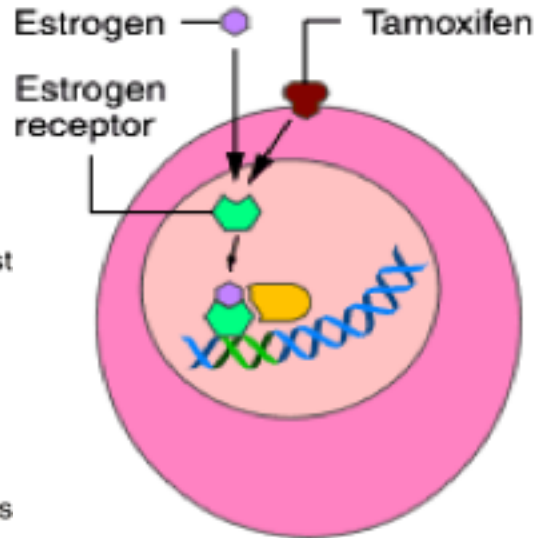
Bone

Estrogen helps to preserve bone density.

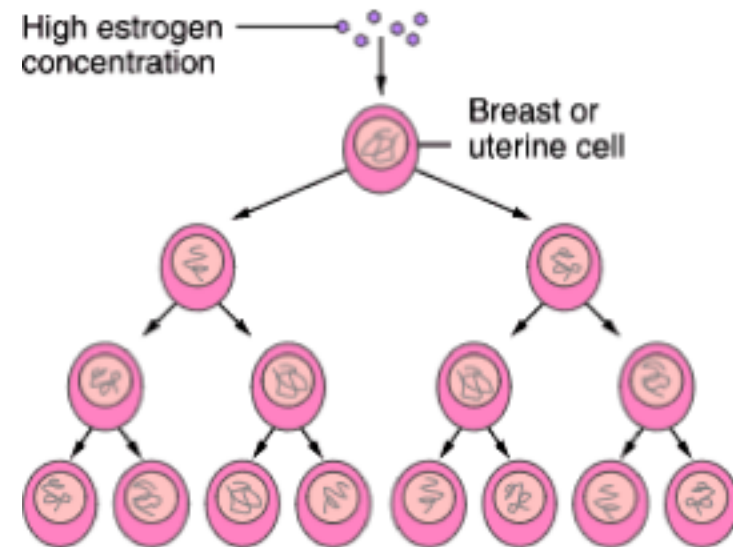
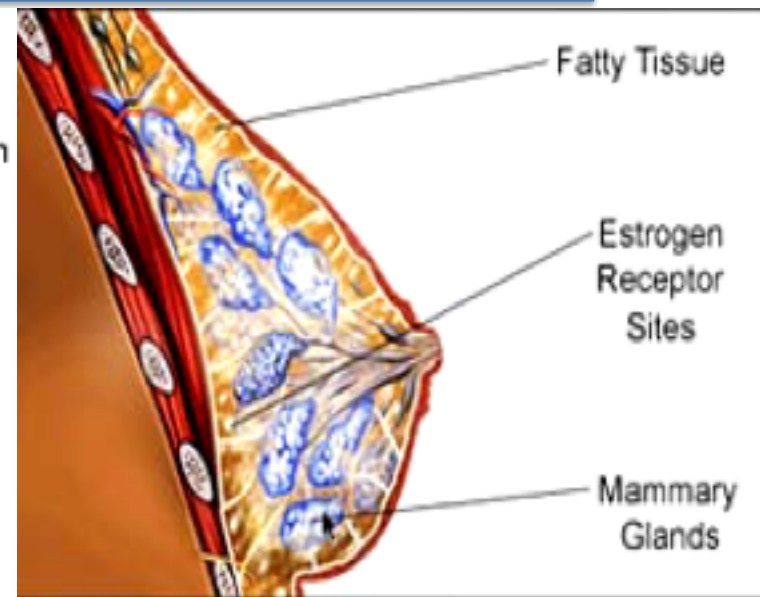
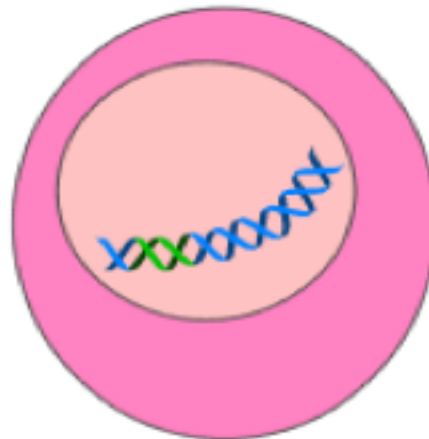
Estrogen Target Tissues



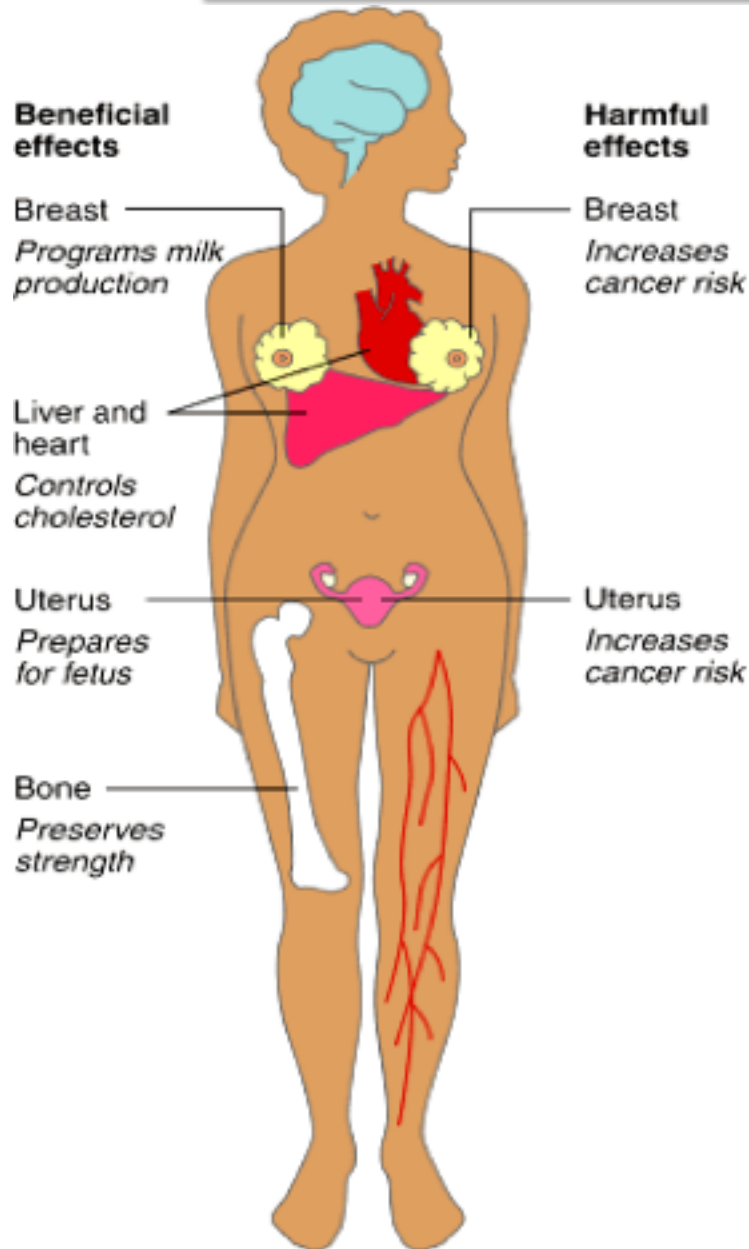
Estrogen target cell
(e.g., breast, uterine lining, liver, etc.)



Non-target cell
(contains no estrogen receptor)

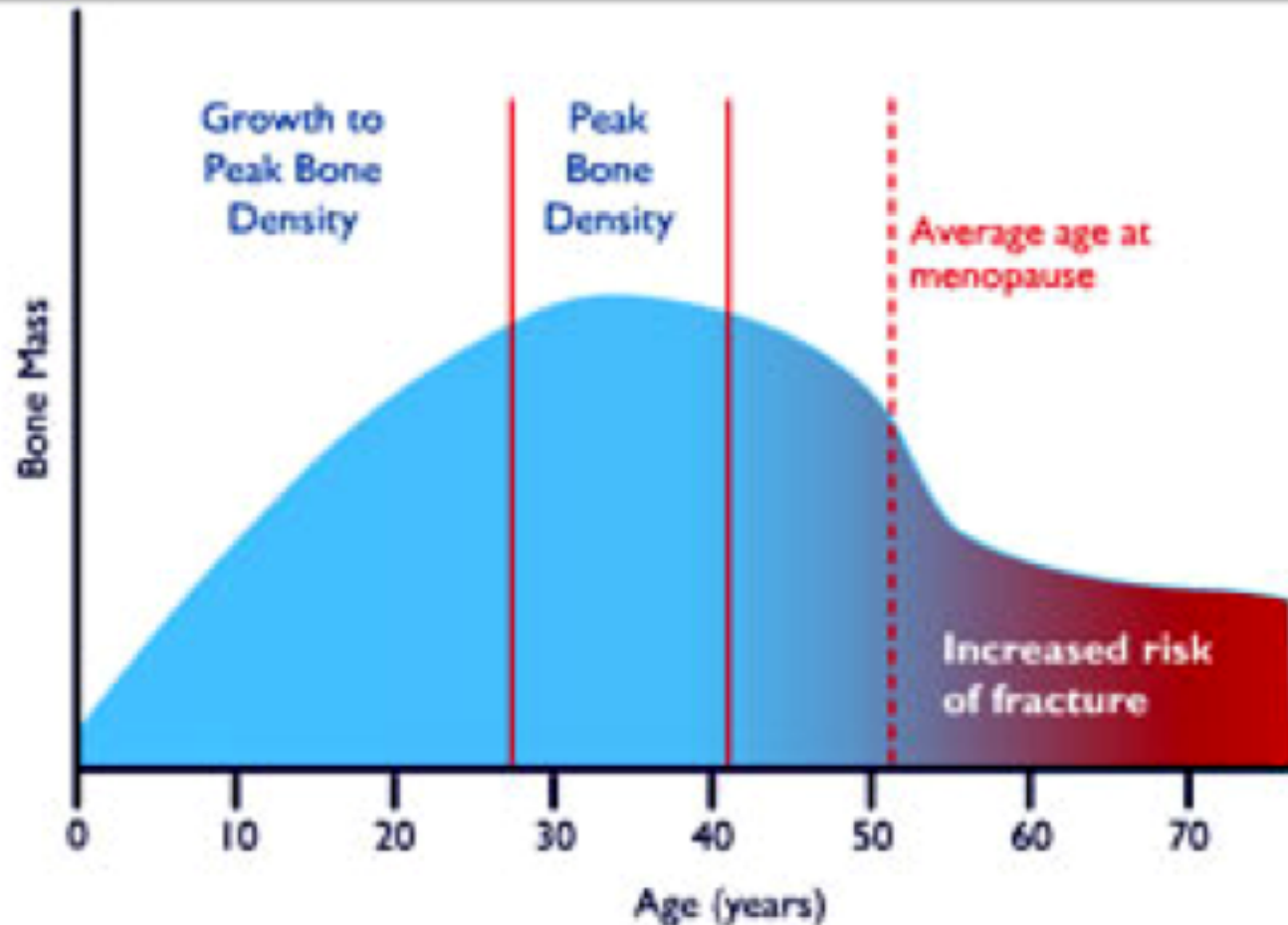


Estrogen and Cancer



- ❑ **The main beneficial effects of estrogen include its roles in**
 - programming the breast and uterus for sexual reproduction
 - safeguarding the heart by controlling cholesterol production in ways that limit the buildup of plaque in the coronary arteries
 - preserving bone strength by helping to maintain the proper balance between bone buildup and breakdown.
- ❑ **Estrogen can also be harmful. The most serious problem arises from its ability to promote proliferation of cells in the breast and uterus**

Hormones, Bone Density & Osteoporosis

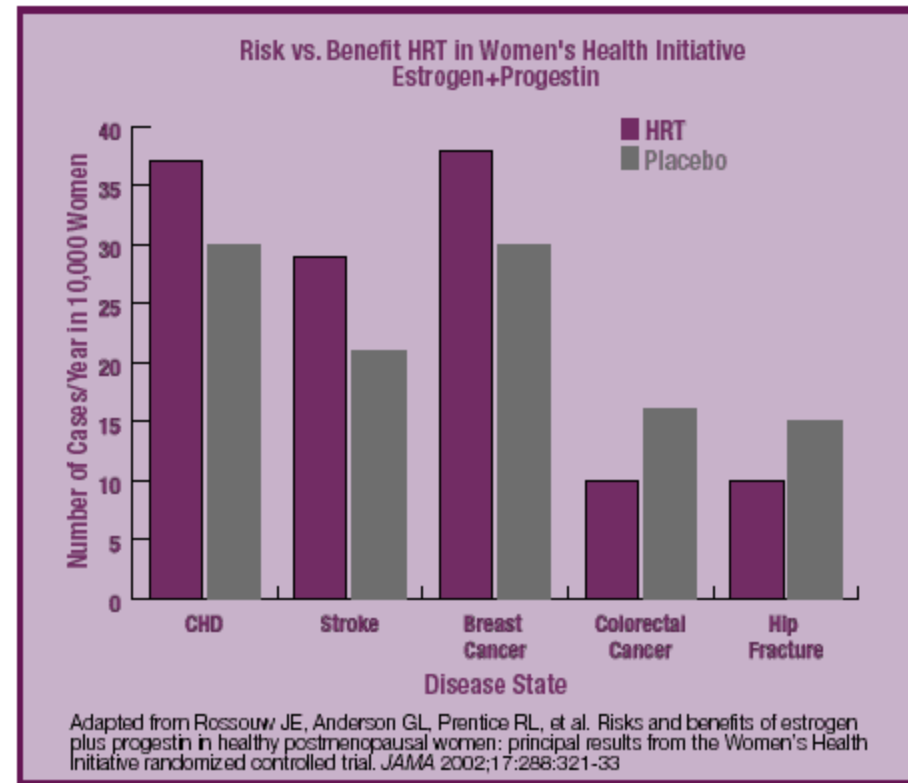


OSTEOPOROSIS

In osteoporotic bones, bone density has fallen to such an extent that the bones have become more fragile, leading to an increased risk of fracture.

What is Hormone Replacement Therapy?

- ❑ In late middle age, a woman has reduced levels of some sex hormones (estrogen and progesterone).
- ❑ Women normally stop having regular periods between their mid-40s and mid-50s
- ❑ This causes unpleasant symptoms such as hot flashes, mood swings, loss of libido.
- ❑ **Hormone Replacement Therapy (HRT)** aims to boost hormone levels to reduce these symptoms.



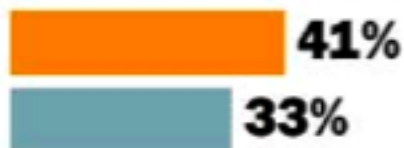
Hormone therapy

AND CANCER

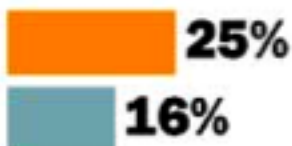
Using combined estrogen and progestin pills to alleviate menopause symptoms may cause breast cancer and make it harder to detect at an early stage.

- Participants who took estrogen and progestin
- Participants who took a placebo

Percentage of invasive breast cancer cases



Breast cancers that had spread by the time they were diagnosed

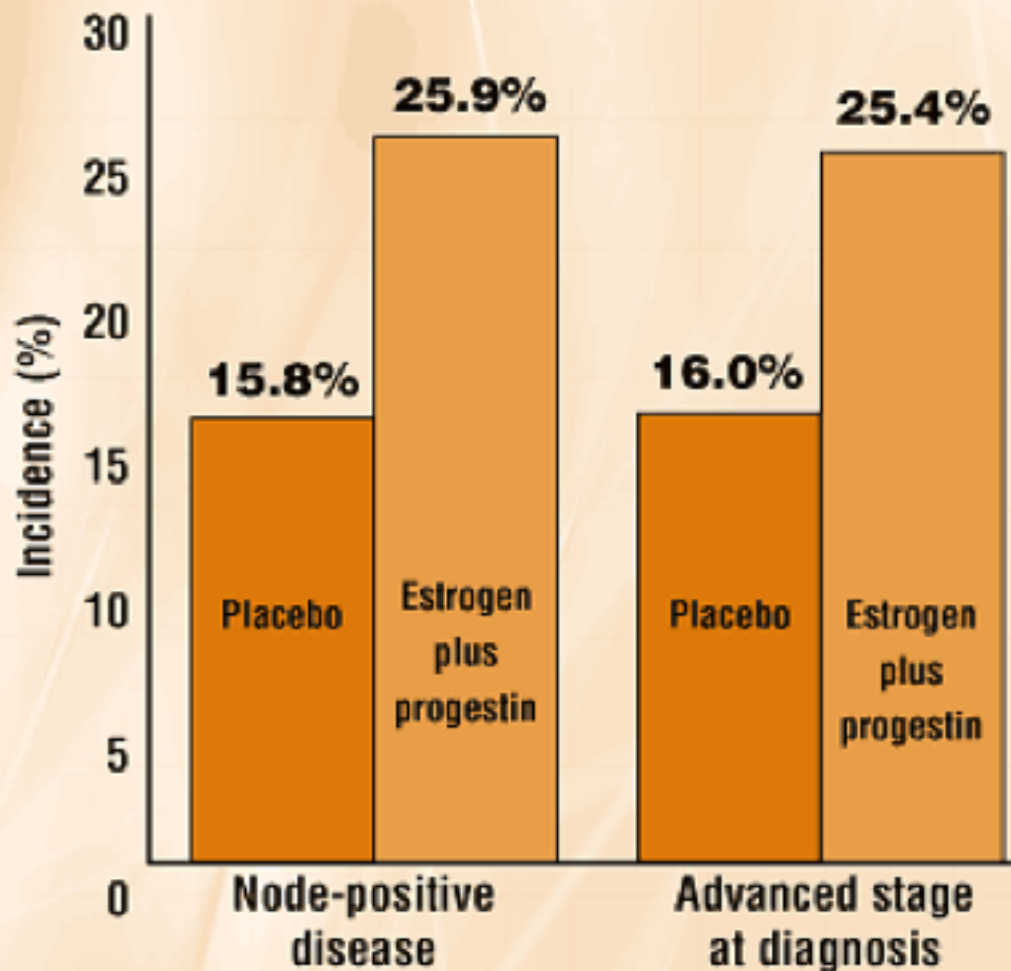


SOURCE: Journal of the American Medical Association

HRT & Breast Cancer

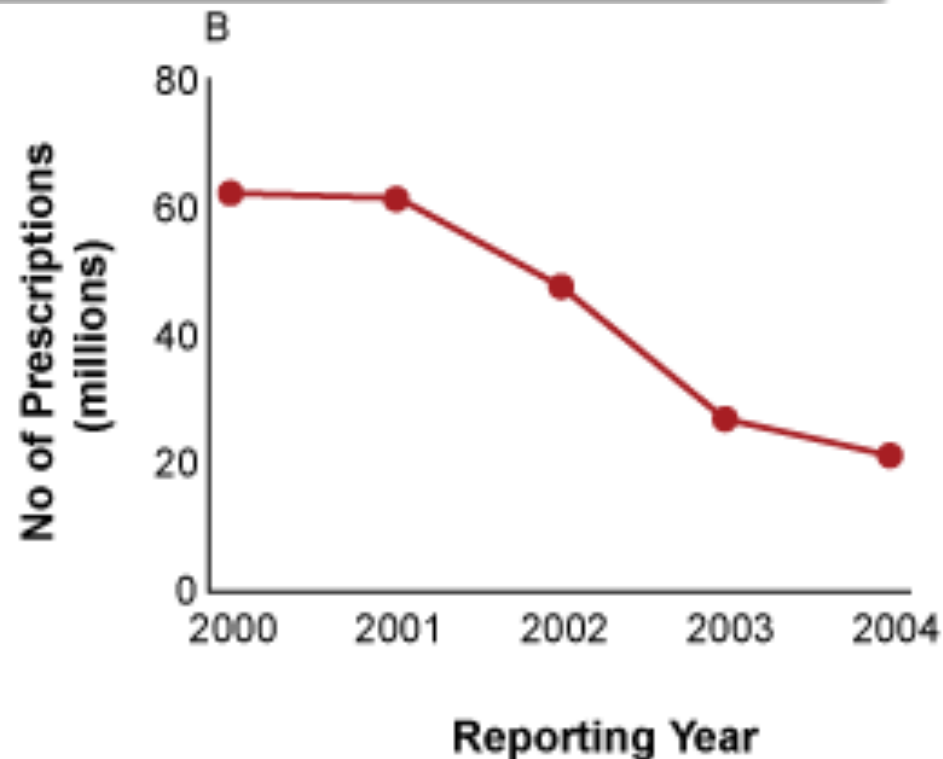
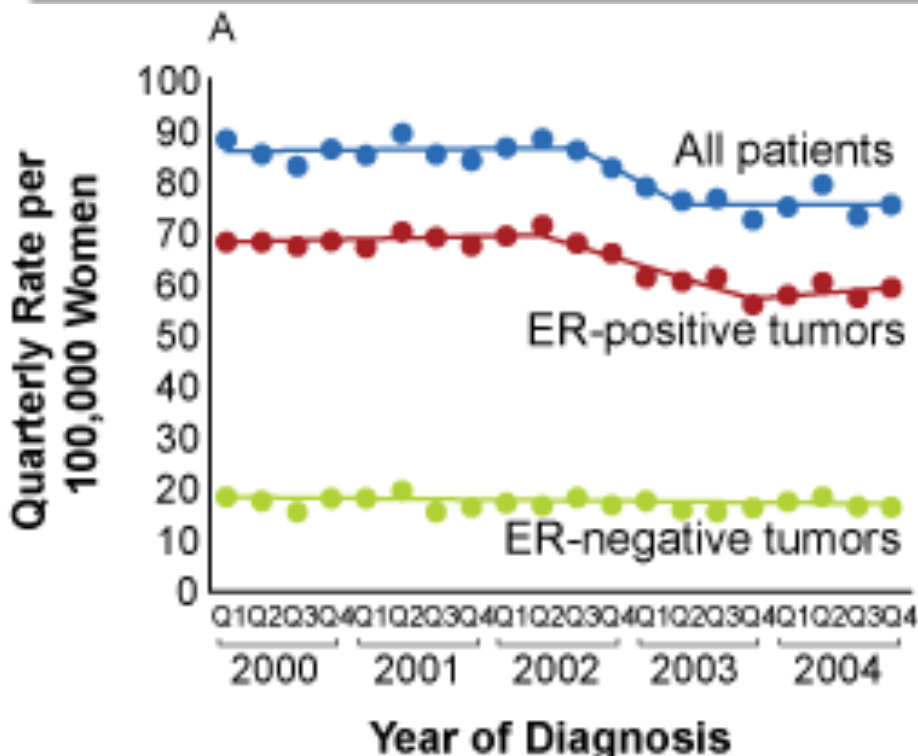
Worse Disease?

Combined hormone therapy increases the risk of advanced, invasive breast cancer



Source: Rowan T. Chlebowski, MD, PhD

Reduction in HRT & Breast Cancer Rates



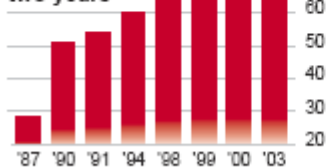
Estrogen has several roles in the risk of developing breast cancer.

1. Stimulates breast cell division during critical periods of breast growth and development.
2. Supports the growth of estrogen responsive tumors.
3. Provides a negative influence on other hormones which may induce the growth of tumors.

Breast cancer rates down

Some experts believe U.S. breast cancer rates dropped sharply in 2003 because many women stopped menopausal hormone use.

Women 40 and older having a mammogram in the previous two years

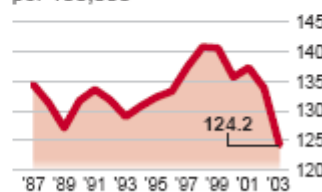


SOURCES: IMS Health; Journal of the American Medical Association; National Center for Health Statistics

Hormone prescriptions dispensed In millions Jan.-March:

Year	Millions
2001	22.8
2003	15.2

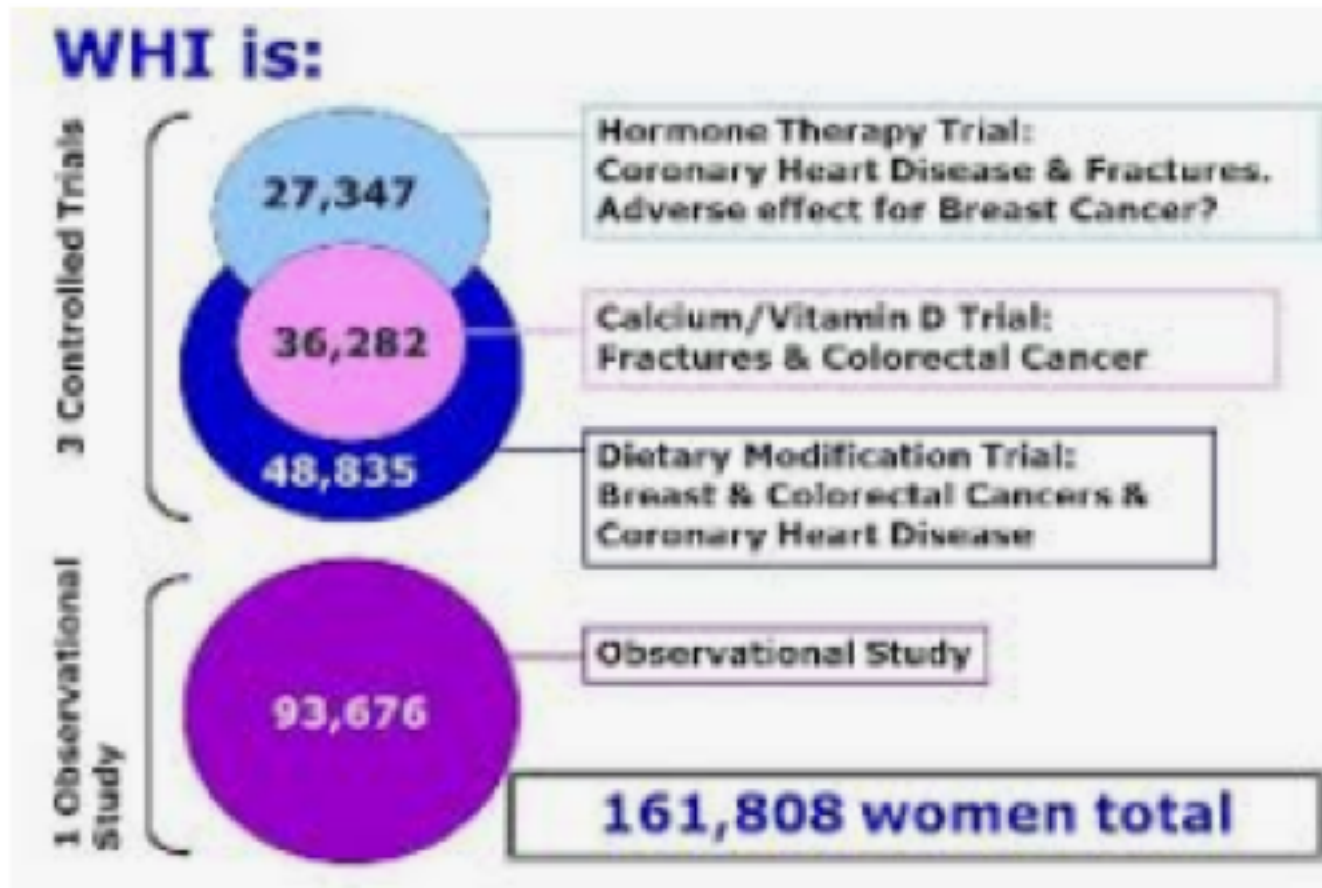
Incidence of breast cancer per 100,000



Million Woman Study

“Combination HRT can cause up to a 200% greater chance of developing breast cancer.”

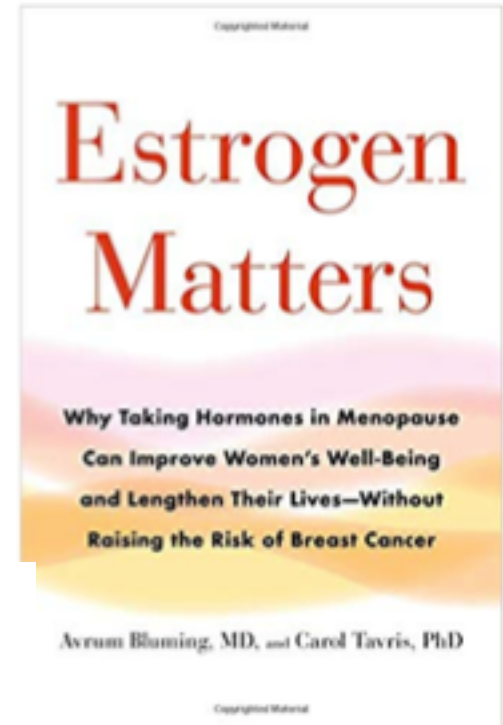
Woman's Health Initiative



Double blind randomized control trial:
neither doctor nor patient knows who is getting treatment or placebo

What did the WHI really show?

The media misrepresented and misinterpreted the WHI findings, giving the impression that HRT was killing women. Not so. Over 10,000 person-years, women on estrogen plus progestin had 7 more coronary events, 8 more strokes, 8 more pulmonary emboli, and 8 more invasive breast cancers than women who didn't take hormones; but they also had 6 *fewer* colorectal cancers and 5 *fewer* hip fractures. In the estrogen-only group, there was a *decreased* risk of coronary heart disease and breast cancer. And in both groups there was ***no increase in overall mortality***. These women were getting more of some diseases and less of others, but they weren't dying any faster.



Hormone replacement therapy (HRT) came in two flavors. Women with an intact uterus were given a combination of estrogen and progestin, since unopposed estrogen was known to cause cancer of the uterus. Women who had had a hysterectomy could safely take estrogen alone (ERT). Estrogen was contraindicated for women who had had breast cancer.

As of the 1990s, the evidence indicated that estrogen reduced the risk of heart disease by 40-50%, hip fractures by 50%, colon cancer by 50%, and Alzheimer's by 35%.

A 1997 article estimated HRT would prolong life by 3 years.

They say, "it is time to relegate the "common knowledge" that ERT and HRT cause breast cancer to the dustbin of discredited ideas along with the theories that radical mastectomy is the best treatment for primary breast cancer, that anger causes peptic ulcers, and that stress causes tuberculosis."

The WHI continued to do follow-up analyses and publish updated findings:

- In 2006 a follow-up analysis of the same cohort of women found *no increased risk* with combined estrogen-progesterone treatment. This time, the news didn't make headlines.
- In 2008, another follow-up found a *nonsignificant* increase in all-cause mortality and *no increased mortality* from breast cancer.
- In 2010, another follow-up showed an increase in breast cancer deaths (2.6 vs. 1.3 per 10,000) in women on HRT compared to placebo. But this was *not statistically significant*.

HRT and breast cancer risk: a realistic perspective.

Stevenson JC, Hodis HN, Pickar JH, Lobo RA.

Abstract

A new analysis from the Women's Health Initiative included data on breast cancer incidence over a 11-year period from the randomized trial of conjugated equine estrogens (CEE) plus medroxyprogesterone acetate (MPA) and a subsequent observational follow-up. The conclusions were that CEE/MPA use was associated with an increase in both breast cancer incidence and mortality. We have concerns over the validity of their statistical analyses, as adjustments for baseline characteristics or for multiple comparisons demonstrate no significant differences in incidence between those allocated to CEE/MPA or placebo. We suspect that the apparent increase in mortality is the result of surveillance and detection bias rather than a true cause and effect. Even if such an effect were true, mortality from breast cancer would still be a very rare event. We also question the clinical relevance and applicability of their findings. The data over the 11 years show no increased risk of breast cancer with CEE/MPA in women who had not previously used hormone replacement therapy (HRT), and the vast majority of women on HRT would not be prior users at initiation. It should be remembered that women using CEE alone showed a significant decrease in breast cancer risk in the WHI trial and follow-up. Even if combined estrogen?progestogen HRT did cause an increase in breast cancer risk, and this is not proven, the magnitude of that risk is small, and less than that risk seen with many lifestyle factors. HRT is a benefit, not a risk, for those women requiring it.

Million Woman Study

“Combination HRT can cause up to a 200% greater chance of developing breast cancer,”

The Million Women Study followed, reporting an increase in breast cancer with both estrogen alone and combined estrogen/progestin. But only in current users, not past users. Bluming and Tavaris ask, “If estrogen is a major risk factor in breast cancer, why would *having taken it for years* not be a problem but *taking it at the time of study* be a risk?” Birth control pills contain the same hormones, and most studies have shown no association with breast cancer. And survival rates for women with breast cancer are higher if they were taking oral contraceptives at the time of diagnosis.

They provide a table of risk factors that have been associated with breast cancer. Estrogen reduces risk (0.77 relative risk), the WHI reported an increase in risk with estrogen and progesterone (1.24 or 1.26 relative risk). Other things that increase risk include fish intake (1.14), multivitamin use (1.19), alcohol (1.26), French fries (1.27), night shift work (1.51), increased carbohydrate intake (2.22), left-handedness (2.41), and electric blanket use (4.90).

Does hormone replacement therapy cause breast cancer? An application of causal principles to three studies. Part 4: the Million Women Study.

Shapiro S¹, Farmer RD, Stevenson JC, Burger HG, Mueck AO.

⊕ Author information

Abstract

BACKGROUND: Based principally on findings in three studies, the collaborative reanalysis (CR), the Women's Health Initiative (WHI) and the Million Women Study (MWS), it is claimed that hormone replacement therapy (HRT) with estrogen plus progestogen (E+P) is now an established cause of breast cancer; the CR and MWS investigators claim that unopposed estrogen therapy (ET) also increases the risk, but to a lesser degree than does E+P. The authors have previously reviewed the findings in the CR and WHI (Parts 1-3).

OBJECTIVE: To evaluate the evidence for causality in the MWS.

METHODS: Using generally accepted causal criteria, in this article (Part 4) the authors evaluate the findings in the MWS for E+P and for ET.

RESULTS: Despite the massive size of the MWS the findings for E+P and for ET did not adequately satisfy the criteria of time order, information bias, detection bias, confounding, statistical stability and strength of association, duration-response, internal consistency, external consistency or biological plausibility. Had detection bias resulted in the identification in women aged 50-55 years of 0.3 additional cases of breast cancer in ET users per 1000 per year, or 1.2 in E+P users, it would have nullified the apparent risks reported.

CONCLUSION: HRT may or may not increase the risk of breast cancer, but the MWS did not establish that it does.



Home

Recommendations

Published Final
Recommendations

Recommendations in
Progress

Copyright Notice

Information for Health
Professionals

Information for
Consumers

Public Comments and
Nominations

Methods and Processes

About the USPSTF

You are here: [Home](#) » [Recommendations for Primary Care Practice](#) » [Published Recommendations](#) » [Recommendation Summary](#) » **Final Recommendation Statement : Final Recommendation Statement**

Final Recommendation Statement

Hormone Therapy in Postmenopausal Women: Primary Prevention of Chronic Conditions

Recommendations made by the USPSTF are independent of the U.S. government. They should not be construed as an official position of the Agency for Healthcare Research and Quality or the U.S. Department of Health and Human Services.

Recommendation Summary

Population	Recommendation	Grade (What's This?)
Postmenopausal women	The USPSTF recommends against the use of combined estrogen and progestin for the primary prevention of chronic conditions in postmenopausal women.	D
Postmenopausal women who have had a hysterectomy	The USPSTF recommends against the use of estrogen alone for the primary prevention of chronic conditions in postmenopausal women who have had a hysterectomy.	D

Menopausal hormone therapy for primary prevention: why the USPSTF is wrong.

Langer RD¹, Simon JA², Pines A³, Lobo RA⁴, Hodis HN⁵, Pickar JH⁶, Archer DF⁷, Sarrel PM⁸, Utian WH⁹.

Author information

Erratum in

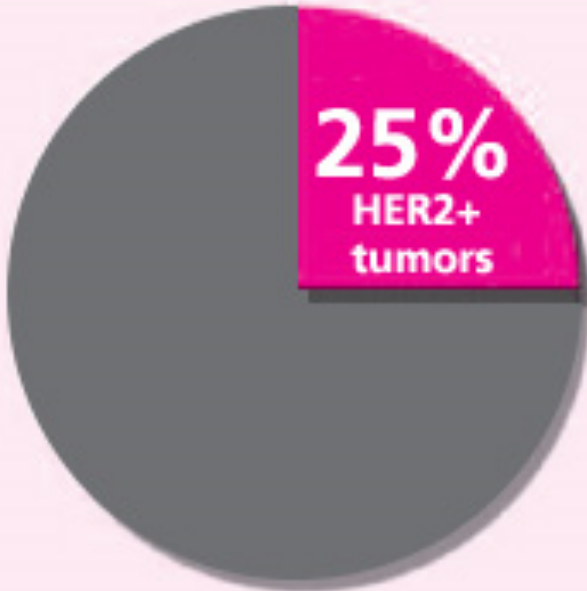
Correction to: Langer et al., Menopausal hormone therapy for primary prevention: why the USPSTF is wrong. [Climacteric. 2017]

Abstract

The US Preventive Services Task Force (USPSTF) Draft Recommendation statement on Menopausal Hormone Therapy: Primary Prevention for Chronic Diseases, released in May 2017, perpetuates a major disconnect between the primary population affected, women within roughly 10 years of menopause, and the data cited. Furthermore, major elements of the evidence relied upon have been misinterpreted or misstated, particularly in regard to coronary heart disease and breast cancer, for which there is no statistically significant evidence of harm. As currently drafted, the recommendations reiterate the USPSTF statements of 2012, 2005 and 2002, and will perpetuate egregious harm to the public health. In an attempt to avoid that outcome and to facilitate a return to rational discourse regarding menopausal hormone therapy, an ad hoc group of experts in menopausal health submitted this comprehensive response to the USPSTF.

HER-2 Receptor and Cell Growth

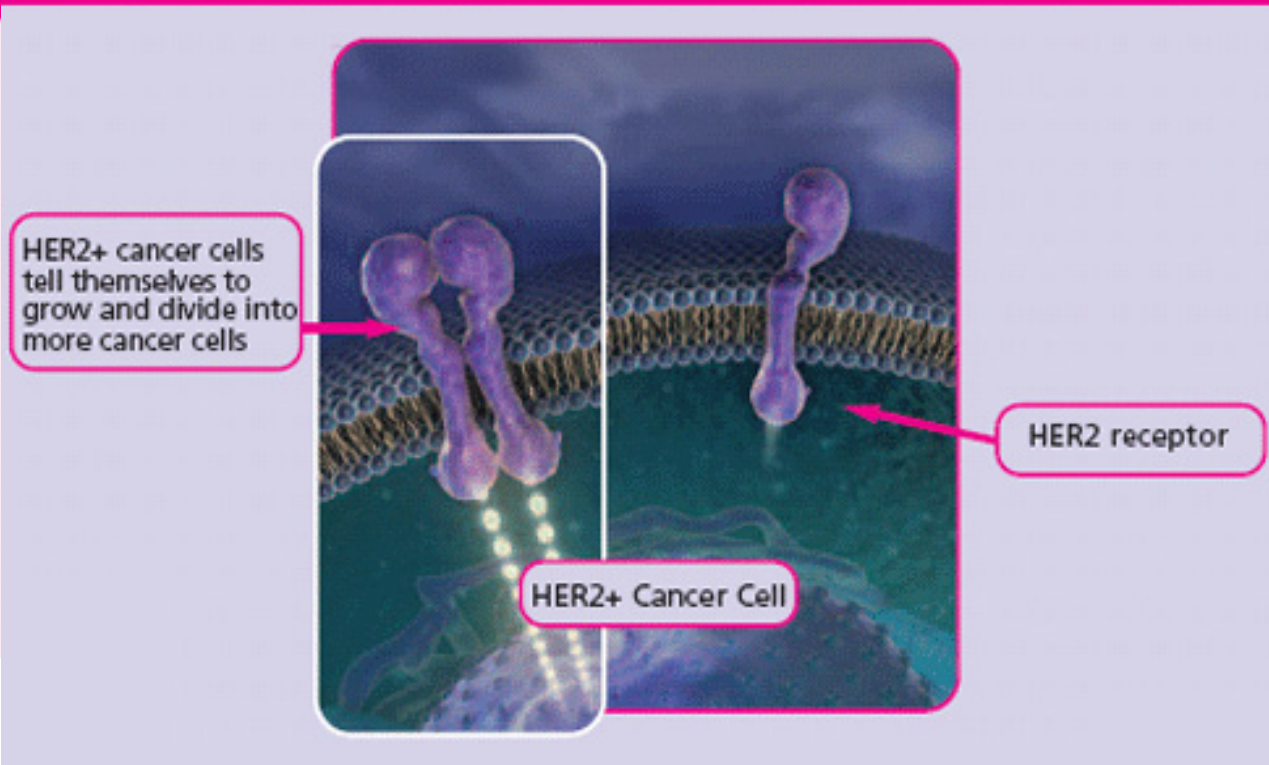
Breast cancer patients



A pie chart with a pink segment representing 25% of the total. The segment is labeled '25% HER2+ tumors'.

25%
HER2+
tumors

HER2+ breast cancer cell



A diagram showing a cross-section of a cell membrane. On the left, two HER2 receptors are dimerized, with a signal transduction pathway (yellow dots) leading into the cell. A text box points to this dimer: 'HER2+ cancer cells tell themselves to grow and divide into more cancer cells'. On the right, a single HER2 receptor is shown. A text box points to it: 'HER2 receptor'. The cell interior is labeled 'HER2+ Cancer Cell'.

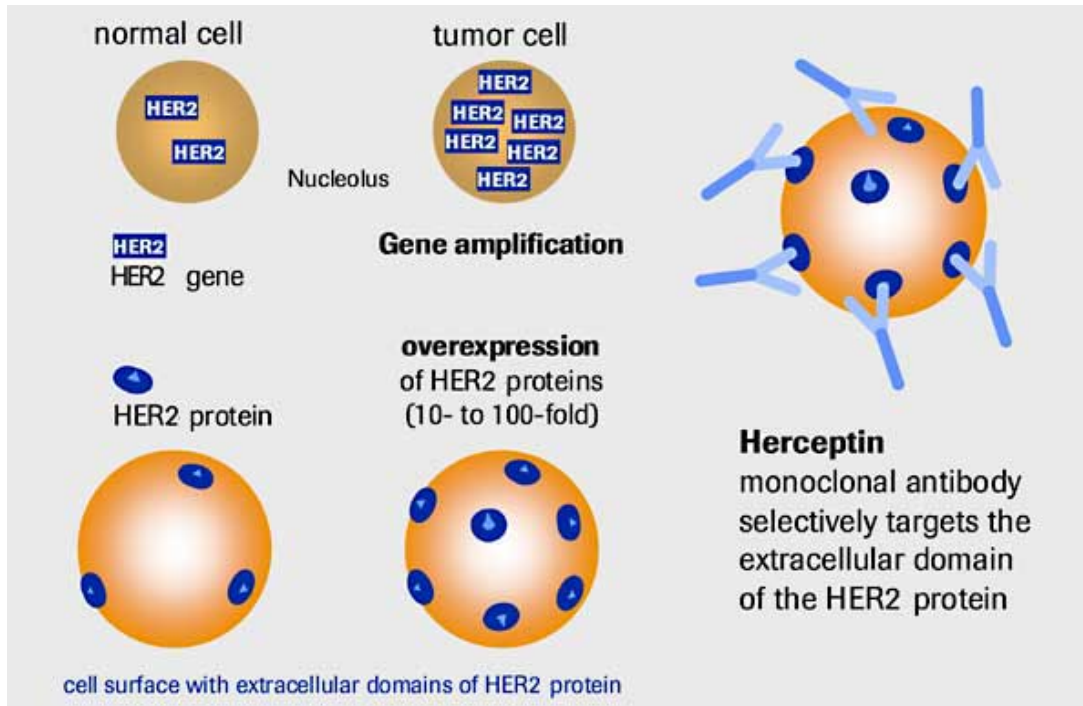
HER2+ cancer cells tell themselves to grow and divide into more cancer cells

HER2 receptor

HER2+ Cancer Cell

- ❑ Human *epidermal growth factor receptor-2 (HER 2)*
- ❑ HER2+ breast cancer is aggressive, so it is important to find out your cancer's HER2 status
- ❑ Higher risk of breast cancer returning (recurrence)

HER-2 Positive Breast Cancer



Breast cancer with amplified HER2 neu oncogene stained with fluorescence in situ hybridization (FISH)

- ❑ HER-2 tumors are less responsive to hormone treatment
- ❑ HER-2 over-expression is also observed in tumors of the lung, intestine, prostate and fallopian tubes.

Her-2 Breast Cancer and Herceptin

Cell from body's
own defense system

1) Herceptin
attaches to a
HER2+ cancer cell
and tells your
body's defense
system to target
the HER2+
cancer cell

Without Herceptin,
the HER2+ cancer
cell keeps telling
itself to grow and
divide into more
cancer cells

2) Herceptin may
also stop the
HER2+ cancer
cell from telling
itself to grow and
divide into more
cancer cells

Without Herceptin

With Herceptin

HER2+ Cancer Cell

