

Breast cancer

Incidence and Epidemiology

Patterns of disease, detection, risk and mortality

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What the epidemiology section explains

- How age, geography and clinical subtype shape the distribution of breast cancer.
- Why observed incidence changes when screening and hormonal exposures change.
- How unequal access, tumor biology and screening limitations influence outcomes.

Focus: Invasive carcinoma; Risk-factor context included where necessary.

Breast cancer became the most commonly diagnosed cancer

2.3 million

Estimated new cases worldwide in 2020

- The global estimate represented almost 12% of all new cancer diagnoses.
- Breast cancer surpassed lung cancer as the most frequently diagnosed cancer overall in 2020.

Incidence and mortality are different endpoints

Incidence

- Number of newly diagnosed cancers in a defined population and interval.
- Affected by both disease occurrence and intensity of detection.

Mortality

- Deaths attributable to breast cancer in a defined population and interval.
- Influenced by stage at diagnosis, biology and care availability.

A population may have higher recorded incidence without better or worse survival.

The worldwide burden includes unequal outcomes

- Breast cancer is the leading cause of cancer death among females worldwide account.
- Survival gains have been achieved mainly in higher-resource settings.
- Earlier diagnosis and more effective adjuvant treatment are identified as major contributors.

Global incidence and mortality do not move in lockstep.

Lifetime risk in the United States

1 in 8

Females living to age 90 years

- A cumulative probability across the life course, not an annual probability.
- The source explicitly conditions the statement on survival to age 90 years.

United States: Estimated invasive cancers in 2023

297,790

Estimated new invasive breast cancer
diagnoses in females

- This is a calendar-year estimate, not a measure of lifetime risk.
- The source reports more than 43,000 breast-cancer deaths for 2023.

Avoid mixing epidemiologic denominators

Lifetime estimate

- Cumulative risk over decades in a defined population.
- Communicates an individual's long-term chance of diagnosis.

Annual count

- Estimated number of diagnoses or deaths in one year.
- Varies with population size and population age structure.

An annual count cannot be directly interpreted as an individual probability.

High-incidence regions identified

- North America.
- Northern and Western Europe.
- Australia and New Zealand.

These are qualitative regional comparisons; No regional rate values were provided.

The burden is rising in many low-resource countries

- The source describes rising breast cancer incidence in many low-resource settings.
- Breast cancer mortality is also increasing in these settings.
- The associated social changes and access constraints differ from those in high-resource regions.

Incidence trends require both exposure and detection context.

Reproductive shifts can increase population risk

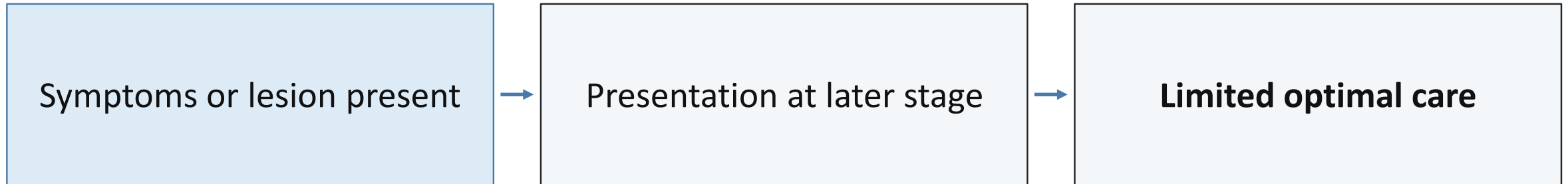
Delayed childbearing

Fewer pregnancies

Reduced breastfeeding

- These social trends as possible contributors to higher incidence.
- They alter the distribution of protective reproductive histories across cohorts.

Limited access can amplify mortality



- The textbook links late presentation and lack of access to worsening outcomes.
- Access acts after disease develops; It is not interchangeable with susceptibility.

Breast cancer is rare before age 25

- The source explicitly characterizes disease in females younger than 25 as rare.
- A breast lesion in this age group cannot be presumed malignant from age alone.
- Population risk differs from the need to investigate a clinically concerning lesion.

Age modifies prior probability; It is not an exclusion criterion.

Incidence rises rapidly after age 30



- A rapid rise after age 30.
- Subtype-specific trajectories diverge as patients age.

Three practical clinical breast cancer groups

Clinical group	ER / HER2 pattern	Epidemiologic significance
Luminal	ER-positive / HER2-negative	Incidence continues rising into later life
HER2-positive	HER2 overexpression; ER may vary	Incidence plateaus by middle age
Triple-negative	ER-, PR-, HER2-	Incidence plateaus by middle age

Clinical subtype labels should not be mistaken for a complete molecular taxonomy.

Luminal breast cancer: Later-life predominance

- ER-positive/HER2-negative tumors are described as the luminal clinical group.
- Luminal cancer incidence continues to rise with age.
- Its incidence peaks later than that of TNBC or HER2-positive cancer.

Cumulative rise changes the older-age case mix.

TNBC and HER2-positive cancers plateau earlier

- The source describes incidence plateaus by middle age.
- This is an age-pattern comparison, not a claim that older adults do not develop these cancers.
- The pattern contrasts with the continued later-life increase of luminal tumors.

No numeric age-specific rates were supplied in the available excerpt.

Aggressive clinical subtypes are prominent in younger females

≈ half

TNBC and HER2-positive together among
young-onset cases

- These two groups as nearly half of young-female breast cancers.
- This is a proportion of cancers in an age stratum, not the absolute population incidence.

Their proportion falls in older females

<20%

TNBC and HER2-positive together among
cancers in older females

- Luminal tumors make up an increasing share with advancing age.
- The lower share in older patients does not imply absence of TNBC or HER2-positive disease.

Absolute age-specific rates differ from case proportions

Subtype incidence

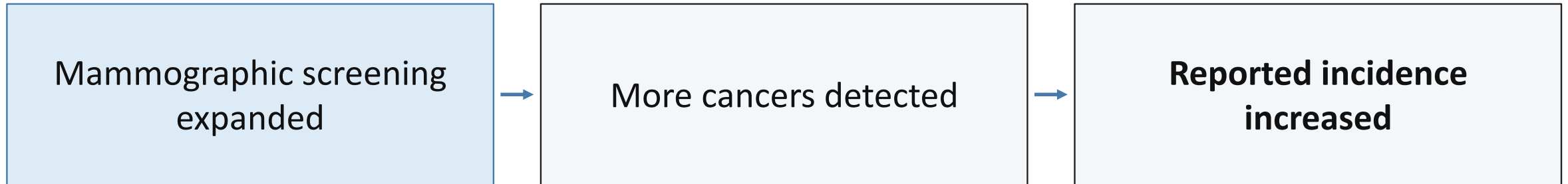
- Rate of new tumors of a subtype per population at a given age.
- The source figure uses rates per 100,000 adult females.

Subtype proportion

- Fraction of all breast cancers in an age group belonging to one subtype.
- Can change because another subtype becomes more common.

Never reconstruct an age-incidence curve from subtype percentages alone.

Two factors shaped the apparent rise in the 1980s



- The source also names increased postmenopausal hormone therapy exposure.
- Recorded incidence reflects both detection practices and disease-associated exposures.

Screening and hormonal therapy act differently

Mammographic screening

- Identifies otherwise asymptomatic cancers.
- Can raise recorded case numbers through earlier detection.

Postmenopausal hormone therapy

- Population exposure increased during the historical period.
- The source identifies its use as a second contributor to incidence change.

Do not attribute an observed incidence increase solely to tumor initiation.

The incidence of older-age cancers fell after 2000

- The source associates the decline with plateauing mammography use.
- Mammography coverage was described as about 65–75% of eligible females.
- Declining use of postmenopausal hormonal therapy was also implicated.

This is a source-era historical account, not a current coverage estimate.

Trends differed by age

Older females

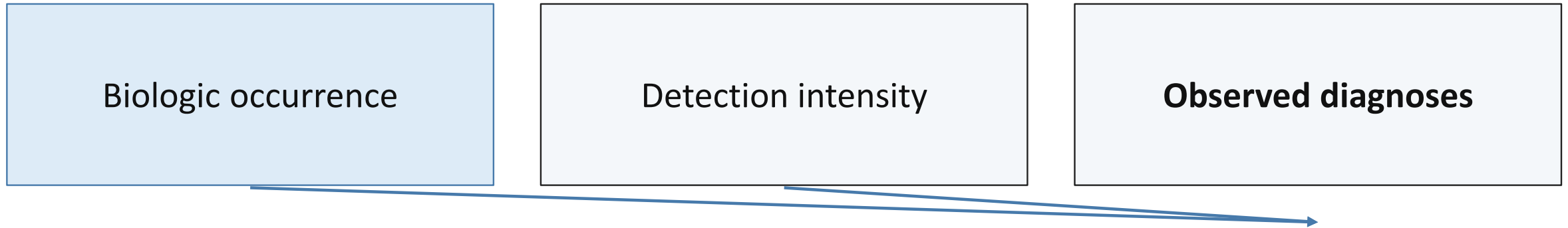
- An apparent screening-era rise was followed by decline after 2000.
- Both screening plateau and hormone therapy changes were cited.

Younger females

- Incidence was described as relatively constant over the same period.
- The age contrast argues against a single uniform population-wide trend.

Avoid applying an older-age trend to every age group.

A framework for interpreting surveillance trends



- A change in mammographic screening alters when and whether a cancer is counted.
- A change in risk exposure may also change the underlying number of new cancers.

Screening seeks clinically occult cancers

- Mammography is used to find asymptomatic, nonpalpable breast carcinoma.
- Mammography as the major screening test.
- A screening abnormality initiates further evaluation; It is not by itself a histologic diagnosis.

Detection and diagnosis are separate steps.

Three principal mammographic signs

Finding	Interpretation	Critical caution
Density	Region of increased attenuation	Not specific for malignancy
Architectural distortion	Disturbed tissue arrangement	Not specific for malignancy
Calcifications	Radiographically visible deposits	Not specific for malignancy

Morphologic and imaging findings require clinicoradiologic assessment.

A density is a finding, not a diagnosis

- A focal or asymmetric density can prompt diagnostic assessment.
- Density among the principal signs of carcinoma.
- The source explicitly states none of the principal mammographic signs is specific.

Do not assign a carcinoma type from density alone.

Architectural distortion can be a detection clue

- Distortion is listed as a principal mammographic sign of carcinoma.
- It indicates abnormal spatial organization rather than a definitive etiology.
- Histologic confirmation may be needed when the finding is suspicious.

Image appearance and tissue diagnosis serve distinct roles.

Calcifications are not inherently malignant

- Calcifications are among the major signs recognized on mammography.
- The source cautions that no single sign is specific for carcinoma.
- The presence of calcification does not determine invasion by itself.

Do not equate “calcification detected” with invasive disease.

Mammography shifts detection toward smaller tumors

≈1 cm

Mean invasive tumor size at mammographic
detection

- The source states mammographically detected invasive cancers are smaller on average than palpated cancers.
- Tumor size at detection is not interchangeable with biologic indolence.

Mammography performs differently across age groups

- Both sensitivity and specificity increase with age.
- Age therefore changes how screening findings should be interpreted.
- A positive image finding is not equally predictive of cancer across populations.

Performance must be considered in the screened population.

Probability that a mammographic lesion is cancer at age 40

10%

Value reported for age 40 years

- A lesion seen on a mammogram does not automatically represent cancer.
- This estimate is age-contextual and should not be exported as universal test performance.

The probability rises after age 50

>25%

Reported probability for females older than
50 years

- The text contrasts this probability with 10% at age 40.
- Age-related prevalence influences the proportion of suspicious findings that are malignant.

Positive predictive value is not sensitivity

Probability a lesion is cancer

- Condition on having a mammographically recognized lesion.
- Depends on age and the population undergoing imaging.

Sensitivity of mammography

- Condition on truly having cancer.
- Describes the test's ability to detect disease.

Do not interpret the age-specific lesion probabilities as sensitivity.

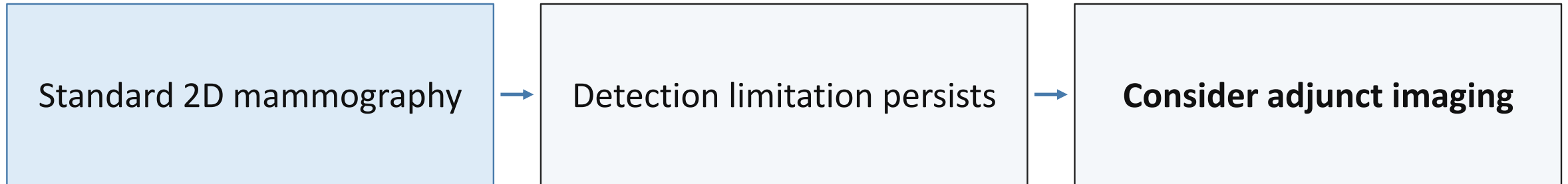
Standard two-dimensional imaging is not infallible

≈10%

Invasive carcinomas not visible on standard
2D mammograms

- A nonvisualized cancer is an important limitation of standard mammography.
- The source names adjunct imaging modalities for selected circumstances.

Adjunct imaging can address selected blind spots



- Digital breast tomosynthesis, ultrasonography and MRI are named adjuncts.
- The excerpt does not prescribe a universal imaging algorithm or testing interval.

Most detected cancers are already invasive

70–80%

Cancers invasive by the time of detection

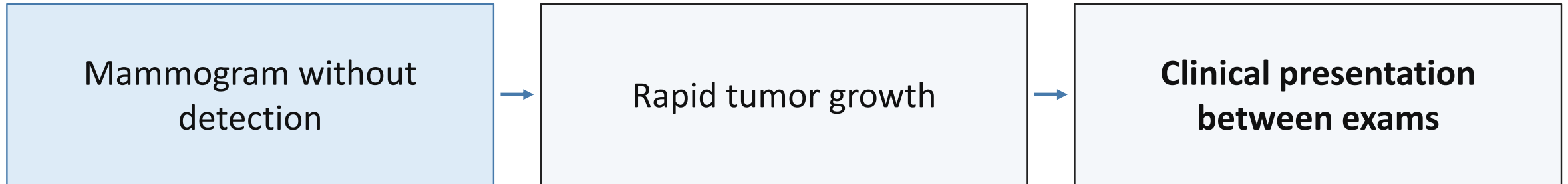
- Earlier diagnosis does not mean that an invasive step has been prevented.
- The source notes that some cancers have already metastasized when detected.

Aggressive disease may arise before screening age

- The text highlights lethal cancers that appear in young, prescreening-age females.
- These cancers cannot be prevented from presenting by screening initiated later in life.
- This is one reason screening has a smaller mortality effect than originally hoped.

Screening eligibility does not determine tumor biology.

Interval cancers challenge periodic screening



- Some aggressive cancers become evident between scheduled mammograms.
- A preceding non-diagnostic mammogram does not guarantee absence of future cancer.

Screening also detects biologically indolent disease

10–30%

Estimated clinically unimportant
mammographically detected cancers

- Such tumors would not cause harm during the individual's lifetime if undetected.
- The figure is an estimate account, not a patient-specific prediction.

Breast screening has two contrasting limitations

Under-detection

- Prescreening aggressive cancers.
- Rapid interval cancers and mammographically occult disease.

Over-detection

- Identification of tumors unlikely to cause harm.
- Potential downstream investigation and treatment of indolent disease.

Assess benefits and harms without treating all detected cancers as equivalent.

Incidence varies across ethnic groups in the source

- The highest incidence among females of European descent.
- Age at presentation and relative subtype composition also differ.
- These are population-level observations, not deterministic predictions for a person.

Ancestry and ethnicity categories are context-dependent descriptors.

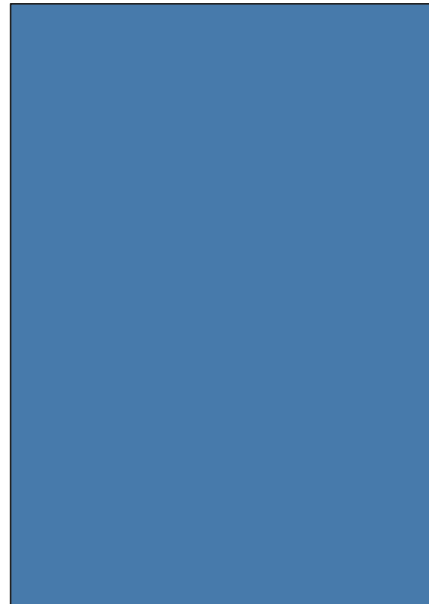
Reported average age at diagnosis by group

Average age at diagnosis (years)

The group means
as 63, 59 and 56
years, respectively.

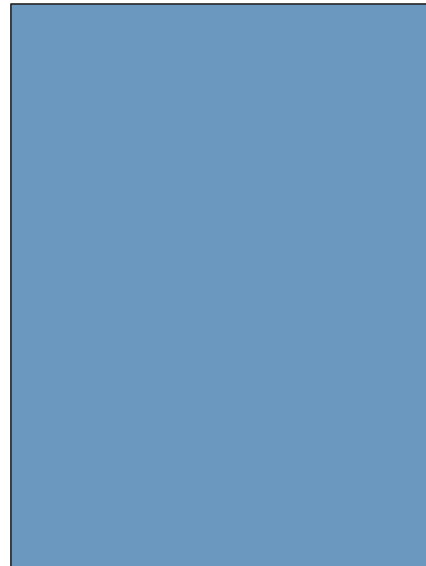
An average is not a
clinical age cutoff
or the age of every
affected individual.

63 years



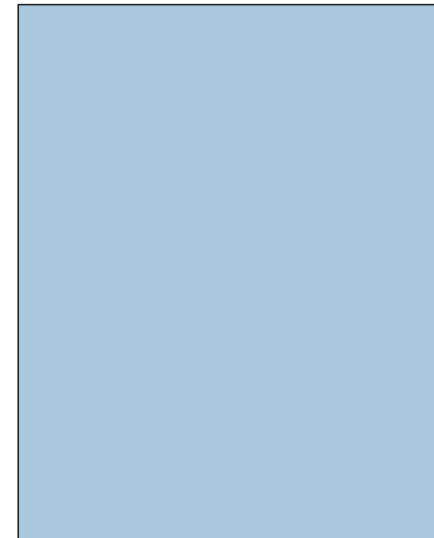
European descent

59 years



African descent

56 years

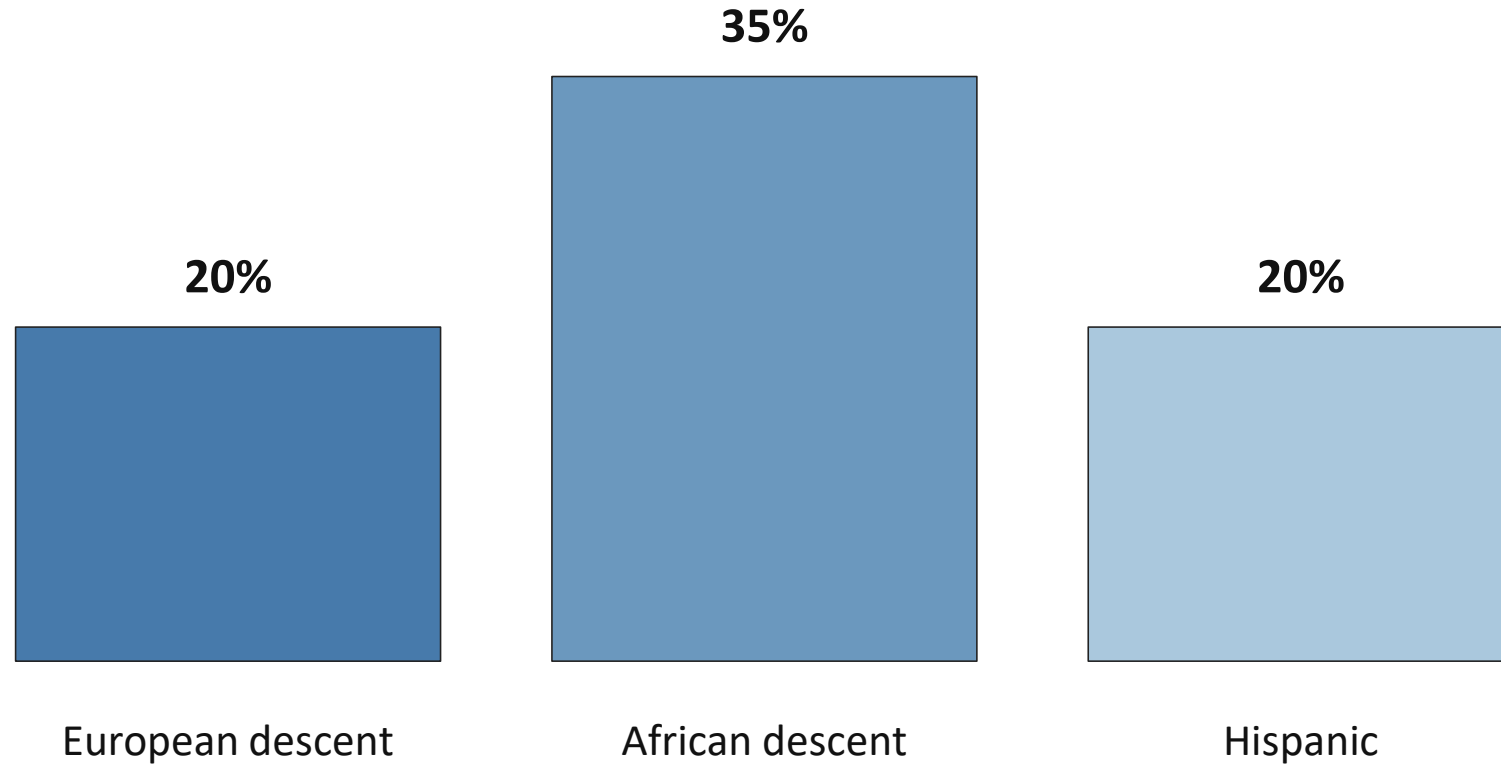


Hispanic

Proportion of diagnoses before age 50

Proportion diagnosed before age 50 (%)

35% before age 50 in females of African descent versus 20% in each of the other listed groups. These are within-group proportions, not population incidence rates.



Luminal cancers contribute to incidence differences

- The source attributes much of the excess incidence in females of European descent to luminal tumors.
- Luminal disease increases more strongly into later life.
- Subtype composition can affect the observed mean age of diagnosis.

Subtype distribution helps explain aggregate epidemiology.

TNBC and HER2-positive proportions also vary

- These groups make up a larger fraction of cases in some non-European-descent populations.
- A higher fraction need not mean a higher absolute subtype rate.
- Do not infer patient-level biomarker status from demographic group membership.

Individual tumors require ER, PR and HER2 assessment.

The reported risk of death has declined

1–2%/year

Recent annual decline reported era

- The decline is described for those diagnosed with invasive breast carcinoma.
- The source links part of mortality improvement to earlier diagnosis and better treatment.

Overall case-associated death risk in the source era

≈20%

Reported overall risk of death after invasive
breast cancer

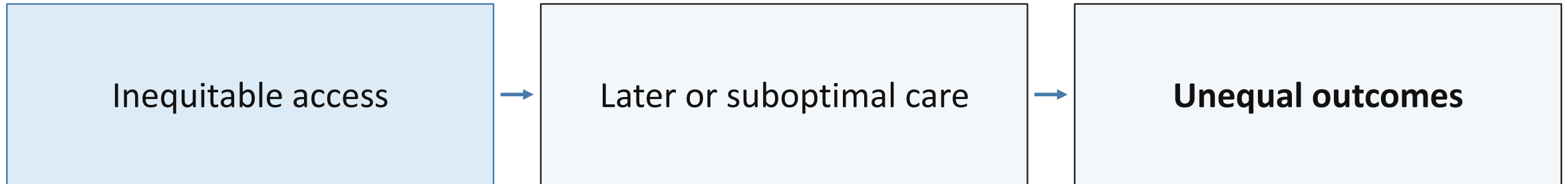
- This is a population summary, not an individualized survival prediction.
- Clinical stage, tumor subtype and access to care influence outcomes.

Mortality gains are not evenly distributed

- That declines in mortality lag in females of African descent.
- This population is described as having the highest breast cancer mortality rate in the cited context.
- The source proposes both disparities in care and differences in tumor subtype as contributors.

Risk disparities are multifactorial.

Care access contributes to mortality disparities



- Access is explicitly identified as a partial explanation for the disparity.
- This framework should not be confused with a biologically fixed causal effect of ancestry.

Subtype mix also affects outcomes

More TNBC in a population



More difficult-to-treat biology



Potential outcome gap

- The source identifies higher proportions of aggressive tumor types among females of African descent.
- Subtype cannot fully explain a disparity without considering care and other determinants.

Population mortality is shaped by two interacting domains

Tumor domain

- ER / PR / HER2 clinical subtype.
- Differences in intrinsic growth and natural history.

Health-system domain

- Timeliness of diagnosis.
- Availability of optimal treatment.

Reducing mortality requires more than a change in screening alone.

Breast cancer risk is multifactorial

- The source highlights sex, age, endogenous estrogen exposure and genetic inheritance.
- Family history adds information about inherited susceptibility.
- Environmental and lifestyle factors are also implicated but contribute variably.

Risk-factor strength must be separated from modifiability.

Female sex dominates the recorded case distribution

99%

People affected by breast cancer who are
female

- The source identifies sex as a major risk determinant.
- Breast cancer in males occurs, but represents a small share of diagnoses.

Age and lifetime estrogen exposure matter

- Incidence increases with age, especially after age 30.
- The table includes early menarche and late menopause among modest risk factors.
- Duration of hormonal exposure is relevant to understanding these associations.

Mechanistic emphasis is hormonal exposure, not a single age threshold.

Higher relative-risk categories

Relative-risk group	Examples	Interpretation
>4.0	High-penetrance germline variants; Strong family history; Prior breast cancer; High density	Large elevation vs reference
2.1–4.0	Moderate-penetrance variants; High-dose chest radiation at young age; One affected first-degree relative	Intermediate elevation
1.1–2.0	Selected hormonal, reproductive and lifestyle exposures	Modest elevation

Table categories are source-era summaries, not individual prediction tools.

Markedly elevated-risk profiles

- High-penetrance germline mutations and strong family history are listed in the >4.0 group.
- Prior personal breast cancer and high breast density also appear in the source's upper band.
- A factor's population frequency differs from its relative-risk magnitude.

High relative risk does not by itself specify absolute risk.

Intermediate relative-risk profiles

- Moderate-penetrance germline mutations appear in the 2.1–4.0 band.
- High-dose irradiation of the chest before age 18 is also in this band.
- A family history involving one first-degree relative belongs to this table category.

Risk strata distinguish strength of association, not diagnosis.

Reproductive and hormone-associated modest risks

- Early menarche (younger than 12) and late menopause (older than 55).
- First pregnancy after 35, nulliparity or absence of breastfeeding.
- Exogenous hormone therapy.

These exposures are listed in the 1.1–2.0 relative-risk band.

Lifestyle factors in the modest-risk band

- Postmenopausal obesity.
- Physical inactivity.
- High alcohol consumption.

These among relative-risk factors of 1.1–2.0.

Modifiable exposures contribute to some cancers

≈20%

Breast cancers attributed to modifiable risk factors

- The source lists obesity, alcohol consumption and low physical activity.
- Attributable fraction is not the percent of individual cases for which causation can be proven.

A suspected exposure is not an established cause

Supported factors

- Obesity, alcohol and inactivity are included as modifiable contributors.
- The source reports an aggregate attributable proportion.

Unproved environmental hypothesis

- Certain pesticides and plastics may have estrogenic effects.
- There is concern but no proof of increased human breast-cancer risk.

Do not translate a hypothesis into a proven etiologic claim.

Early pregnancy and breastfeeding reduce risk

- Pregnancy before age 20 is identified as an important protective factor.
- Prolonged breastfeeding is also described as protective.
- Shifts toward later childbirth and reduced breastfeeding may change population risk over time.

Risk reduction is a population association, not individual certainty.

Risk-reducing mastectomy has a large reported effect

≈90%

Reduction in breast-cancer risk after bilateral prophylactic mastectomy

- The procedure is generally discussed for females at very high risk.
- This is relative risk reduction, not a guarantee of zero residual risk.

Chemoprevention is subtype-specific

- Estrogen receptor antagonists reduce the incidence of ER-positive breast cancers.
- The source does not claim equivalent protection against ER-negative tumors.
- Pharmacologic prevention is described chiefly for women at very high risk.

Biologic subtype determines the expected preventive effect.

High-risk intervention and population prevention differ

Individual high-risk strategy

- Consider family/genetic context.
- Examples: Prophylactic surgery and ER-directed chemoprevention.

Population risk reduction

- Addresses common modifiable exposures.
- Requires attention to reproductive trends and access disparities.

A dramatic relative effect in a selected individual is not an estimate of population impact.

Clinical application: Young patient and subtype

A 34-year-old female is diagnosed with invasive breast cancer.

What can age alone tell you about subtype?

TNBC or HER2-positive disease forms a larger proportion of young-onset cases.

Age cannot establish the tumor's biomarker profile; ER, PR and HER2 require tissue-based evaluation.

Hypothetical teaching scenario — not a patient record.

Clinical application: Screening-negative cancer

A patient presents with a palpable lesion after a recent unremarkable mammogram.

Does the prior mammogram exclude invasive carcinoma?

No. Some invasive cancers are occult on standard 2D mammography.

Aggressive cancers may also become clinically evident between scheduled examinations.

Hypothetical teaching scenario — not a patient record.

Clinical application: Comparing populations

Two groups have different average ages at breast-cancer diagnosis and unequal mortality.

Can the difference be assigned only to inherited biology?

No. The source identifies subtype mix and unequal access to optimal care.

Population-level descriptors do not establish the cause of risk for a particular patient.

Hypothetical teaching scenario — not a patient record.

Integrated teaching conclusions

- Age, reproductive exposure, subtype biology and geography shape incidence.
- Screening changes ascertainment but misses some lethal disease and detects some indolent disease.
- Observed mortality reflects both tumor biology and health-system access.
- Risk-factor magnitude, modifiability and preventive intervention must be interpreted separately.

Epidemiology is the intersection of susceptibility, detection and clinical outcome.

Primary source and scope of estimates

- Robbins, Cotran & Kumar. Pathologic Basis of Disease. 11th edition (2026). Chapter 23, The Breast, pp. 954–956.
- The numerical values dated 2020 and 2023 reflect the historical estimates reproduced in the textbook.
- All diagrams in this presentation are original editable teaching schematics; no clinical images or reconstructed textbook curves are used.
- No current-year incidence, mortality, screening-policy or guideline update was inferred from the textbook excerpt.