

Genetic Testing, Moratoriums, and the Evolving Reality of Risk Assessment



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Genetic Testing, Moratoriums, and Evolving Reality of Risk Assessment

What changed, and the practical implications for
Life & Health insurance.

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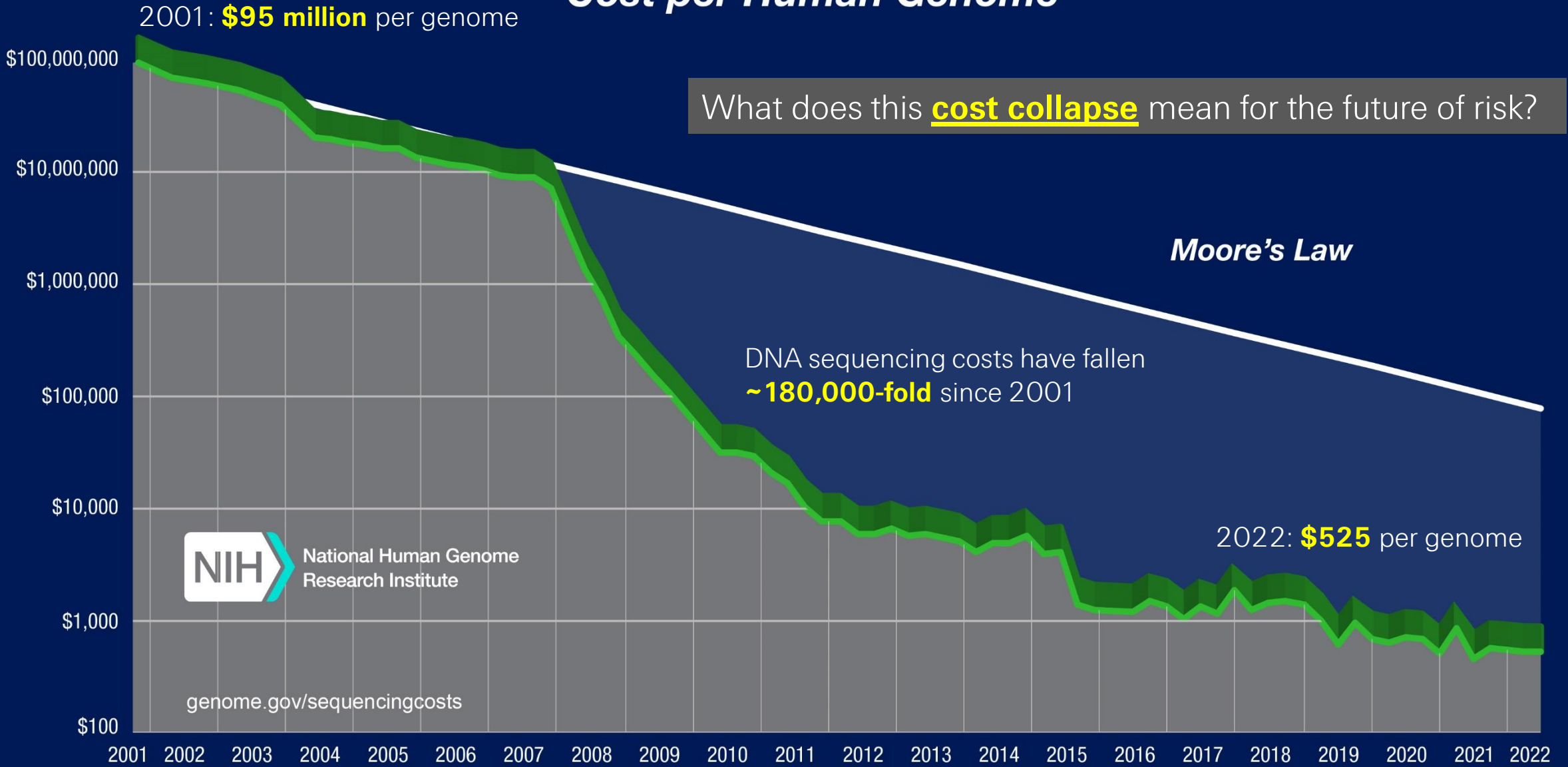


A Quick Reality Check

How many-fold has the cost of sequencing the human genome **fallen over the past 2 decades?**

- A. 10x
- B. 100x
- C. 1,000x
- D. 10,000x
- E. >100,000x

Cost per Human Genome



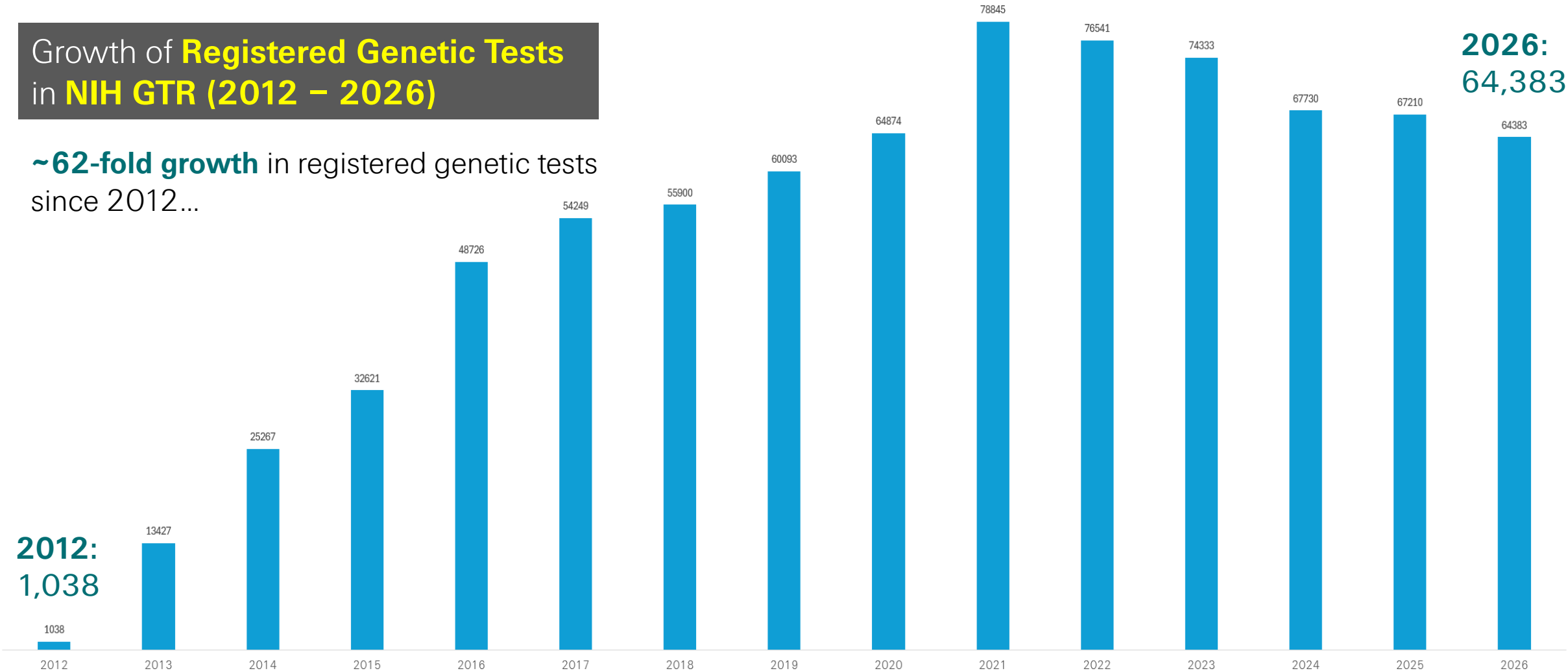
Source: NIH/NHGRI DNA Sequencing Cost Data

As Costs Fell, Genetic Testing Scaled

Lower sequencing cost has accelerated the growth of clinically relevant testing

Growth of **Registered Genetic Tests** in **NIH GTR (2012 – 2026)**

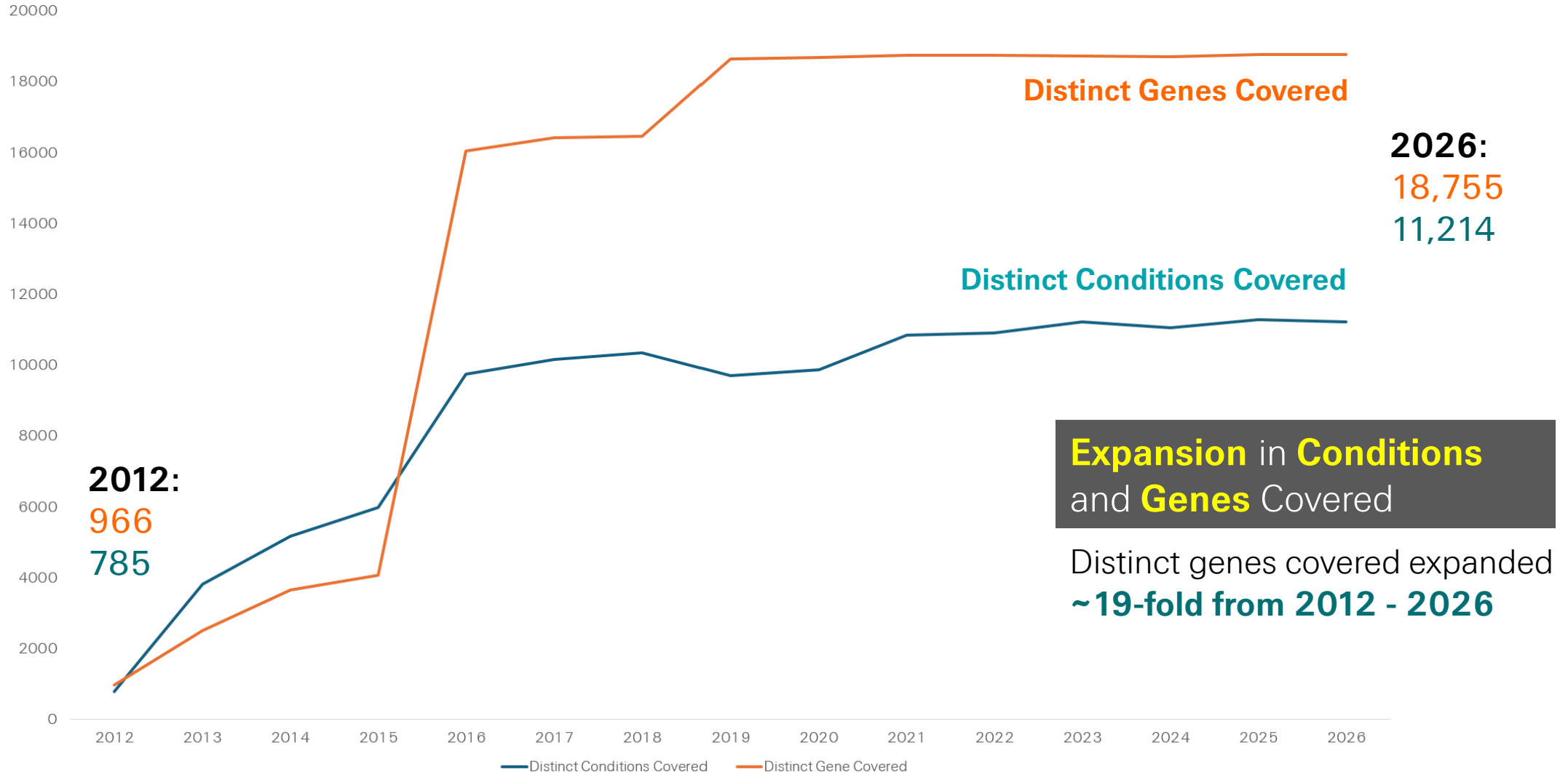
~**62-fold growth** in registered genetic tests since 2012...



Source: NIH GTR (NCBI/NLM), accessed March 2026.

As Costs Fell, Genetic Testing Scaled

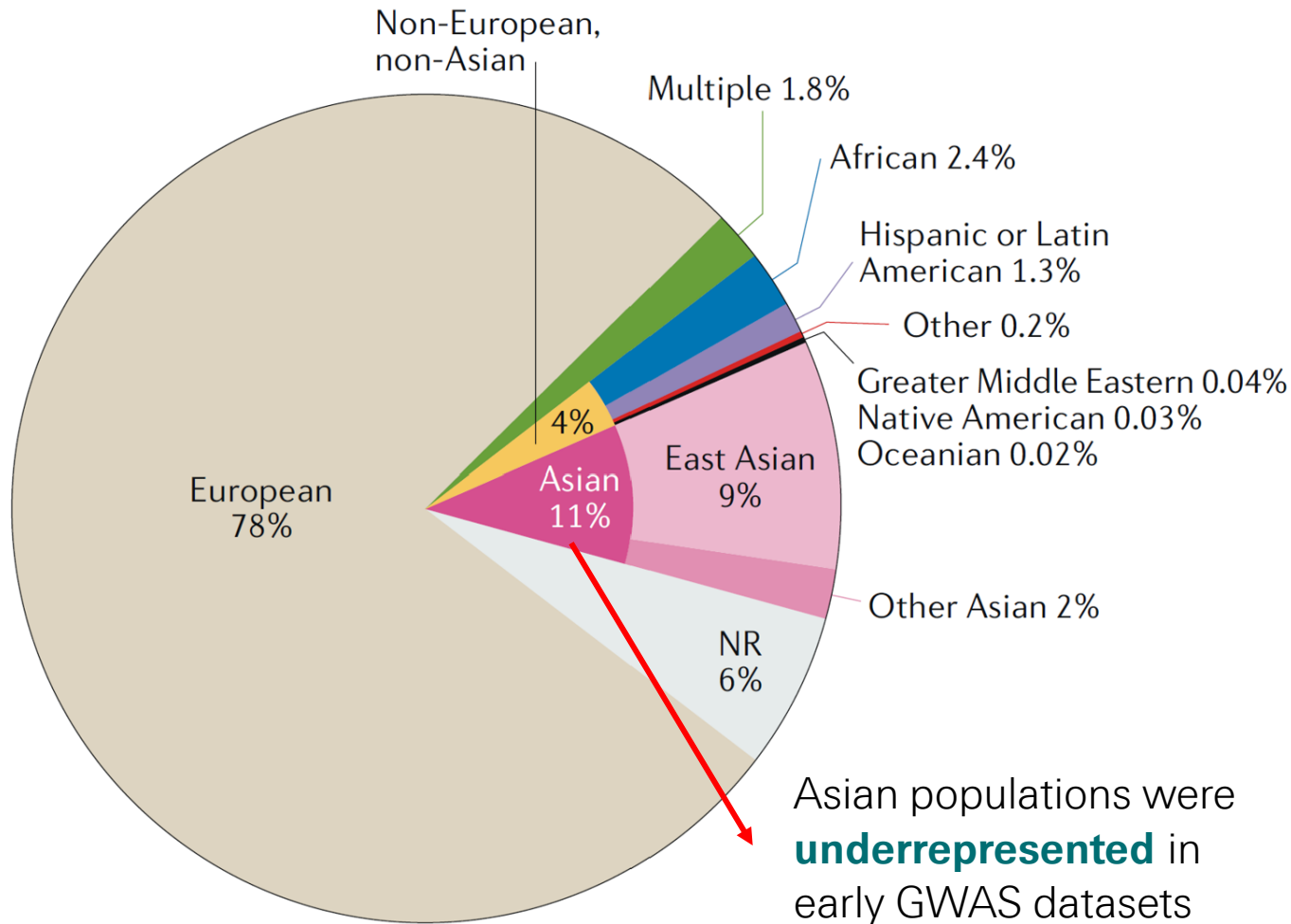
Lower sequencing cost has accelerated the growth of clinically relevant testing



Source: NIH GTR (NCBI/NLM), accessed March 2026.

Why Asia Is Building Genomic Infrastructure

Because **European-led** datasets may not translate reliably to **Asia**



- The **legacy evidence** base is still **heavily skewed away** from **Asia**.
- **Diverse populations** do not just improve fairness, they **improve discovery**.
- **Local genomic resources** are needed for **real clinical translation**.

Source: Genomics of disease risk in globally diverse populations. *Nat Rev Genet.* 2019 Sep;20(9):520-535.

How APAC is Building Genomic Capacity

Precision health is becoming health-system infrastructure

Mainland China

512K+ participants

China Kadoorie Biobank

Population-scale resource with long-term registry linkage.

Hong Kong SAR

60–70K+ participants

HK Genome Project

From project to platform, with expansion underway.

India

10K genomes

GenomeIndia / IBDC

Turning sequencing into a national genomic data asset.

Selected APAC-proof-points



Singapore

100K → 450K+

National Precision Medicine Programme

Scaling toward real-world clinical integration.

South Korea

772K by 2028

Bio Big Data Project

Toward a 1-million-person national genomic resource.

Australia

AUD 500.1m

Genomics Health Futures Mission

Backing genomics integration into healthcare and research.

Source: ¹CCKB ²HKGI/HKSAR Gov ³NPM Singapore ⁴Korea MOHW ⁵Australia Dept Health ⁶GenomeIndia / IBDC

Singapore Clinical Genomic Baseline

Clinically relevant findings may be more common than previously assumed

SG10K
Health



~1 in 30

carried medically
actionable variants

99.7%

had at least one actionable
pharmacogenetic finding

Findings sit in core L&H risk domains



Cardiovascular

Familial hypercholesterolemia

~1 in 140

Premature cardiovascular events



Cancer predisposition

HBOC / Lynch syndrome

~1 in 150 / ~1 in 530

Earlier breast, ovarian, colorectal
or endometrial cancer risk



Cardiac / sudden death

HCM-related variants

~1 in 400

Inherited cardiomyopathy



Neurovascular

CADASIL

~1 in 180

Early-onset stroke and increased
dementia risk

Source: Analysis of clinically relevant variants from ancestrally diverse Asian genomes. *Nature Communications* (2022)13:6694.

Korean Genome Data Shows a Similar Findings

Actionable secondary findings were seen in **3.75%** of Korean genomes, led by **cardiovascular** and **cancer** genes

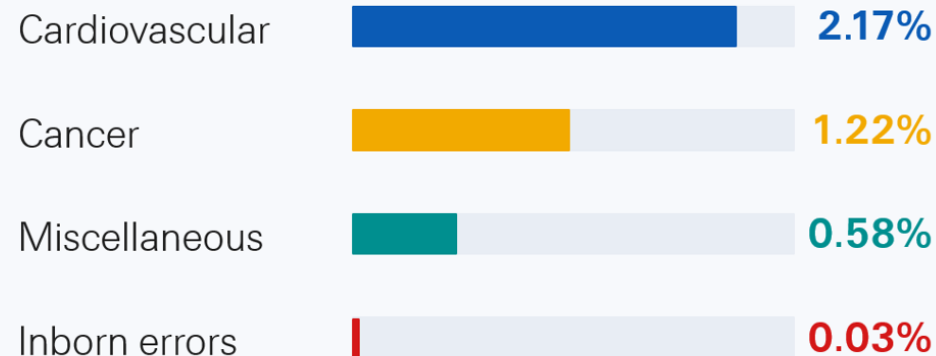
Evidence from 7,472 sequenced Korean participants

ACMG SF v3.1 genes; pathogenic / likely pathogenic variants



About **1 in 27** had an actionable secondary finding

Findings by disease domain



The largest actionable findings sit in cardiovascular and cancer-related genes.



3.12%
**General-
population
KoGES cohort**

Not limited to rare-disease families.

A similar rate was also seen in the general-population KoGES cohort.

Source: Frequency of actionable secondary findings in 7,472 Korean genomes derived from the National Project of Bio Big Data pilot study. *Human Genetics* (2023) 142: 1561-1569.

Why Consumer Trust Matters

Across markets, **concerns** cluster around **discrimination**, **data misuse**, and **loss of control** over genetic information



Discrimination

88%

supported government legislation

Only 4% supported insurer use in underwriting.

Australia survey, n = 367



Data misuse

77%

worried about data breaches

76% concerned about third-party misuse / non-research use.

Korea survey, n = 1,027



Control over use

82%

wanted to know if data would be shared with insurers

54% said sample providers should retain control.

Singapore survey, n = 560

Trust concerns are not abstract: they shape how genetic data can be collected, shared, and used.

1. Community concerns about genetic discrimination in life insurance persist in Australia: A survey of consumers offered genetic testing. *Eur J Hum Genet.* 2024 Mar;32(3):286-294.
2. Conditional trust as a driver of public engagement in Korea's national project of bio-big data. *Front. Genet.* 16:1713598.
3. Who's afraid of genetic test?: An assessment of Singapore's public attitudes and changes in attitudes after taking a genetic test. *BMC Medical Ethics* (2022) 23:5.

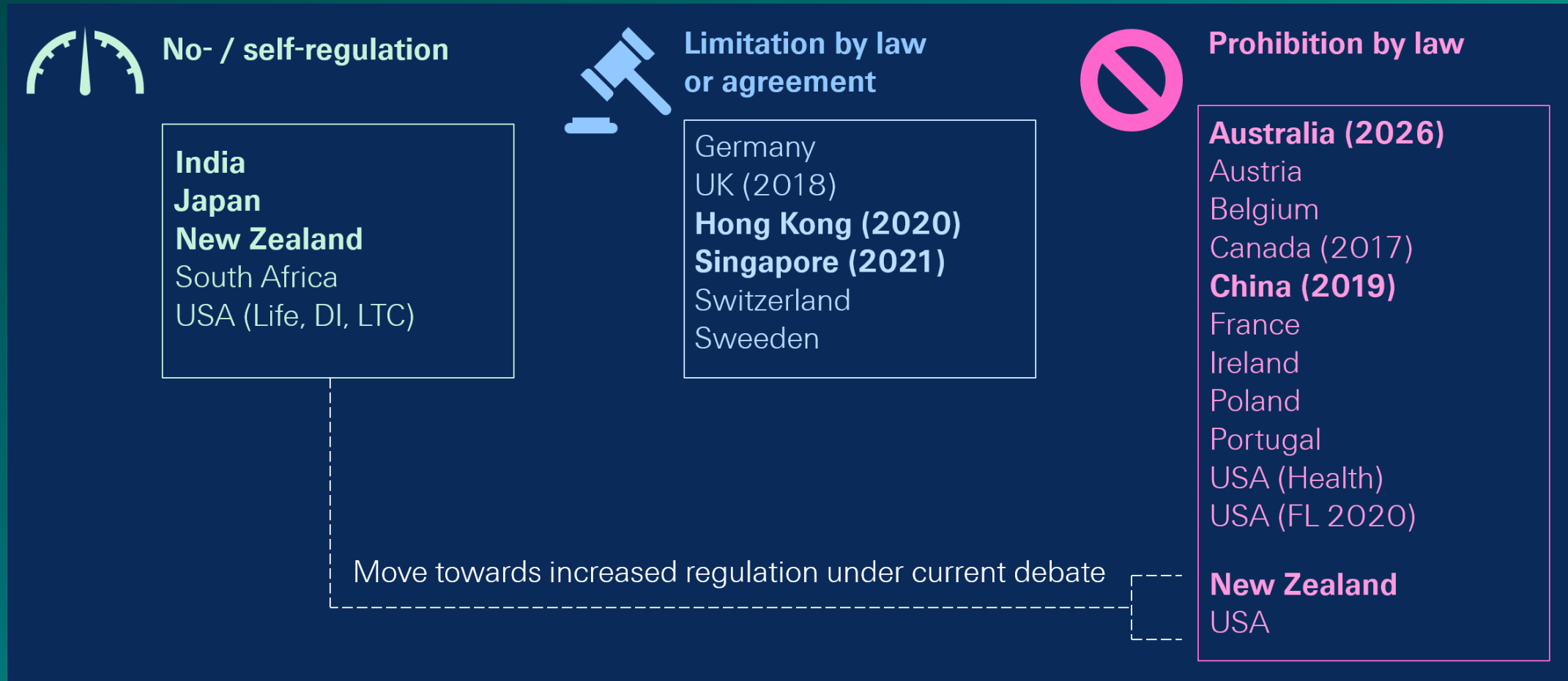
When Regulation Protects Trust

Why moratoriums and legislation
now shape genetic information use



Genetic Information is Becoming More Regulated

Markets are moving from self-regulation towards legal limits and prohibitions on insurer use



APAC already spans the full spectrum, from self-regulation to legal prohibition

Source: Swiss Re regulatory overview (as of Oct 2024; Nov 2024), with Australia updated for 2026 legislation.

How Markets Draw the Line

Hong Kong: Predictive Genetic Test Results Only Above Protection Limits

Position: Limitation by agreement
Framework: HKFI Best Practice
Effective: 1st June 2020

1) Insurers will not

- Require genetic testing for underwriting
- Ask for or use research genetic test results
- Ask for or use a relative's genetic test results

2) Insurers may

- Request diagnostic genetic test results for underwriting
- Use family history information for underwriting

3) Predictive results only above protection limits

Protection limits

- HKD 5M : Life insurance
- HKD 1M : CI / Dread Disease

Specified predictive tests include:

- HBOC
- Lynch Syndrome
- HOCM
- Huntington's Disease
- ADPKD
- Early-onset Alzheimer's Disease

Source: Hong Kong Federation of Insurers. Best Practice on the Use of Genetic Test Results. Effective 1 Jun 2020.

How Markets Draw the Line

Singapore: Moratorium introduced in 2021, strengthened in 2025

Position: Limitation by agreement
Framework: MOH-LIA Moratorium
2025 update: National FH programme explicitly protected

2021: Core moratorium

- Predictive genetic test results are generally protected
- Exceptions applies only if both conditions are met:
 - above approved financial limits
 - approved predictive tests only
- Diagnostic genetic test results may still be used
- Family history remains usable.



2025: What changed

- **New:** Genetic test results under National FH programme are protected
- This protection applies to both **predictive and diagnostic** FH genetic test results
- Moratorium further clarifies clinical vs non-clinical testing
- Threshold-based exception framework is retained and clarified.

BRCA1
BRCA2
Huntington's
Disease

FH: Familial Hypercholesterolaemia

Singapore retains the **predictive-diagnostic framework**, but the 2025 FH update shows that the boundary is becoming **more operationally complex**.

 **Why?**

Source: MOH-LIA. Amended and Restated Moratorium on Genetic Testing and Insurance. Dated 3 Jun 2025; effective 30 Jun 2025

Singapore Familial Hypercholesterolaemia (FH) Prevalence

Higher than Global Average

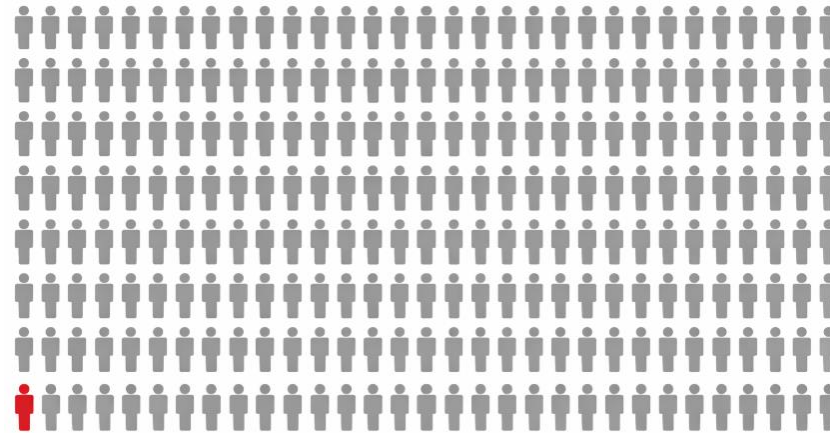


Part of **Singapore National Precision Medicine Programme** (Phase I).

Comprising **10,000 whole-genome sequences** from Chinese, Indian and Malay participants,

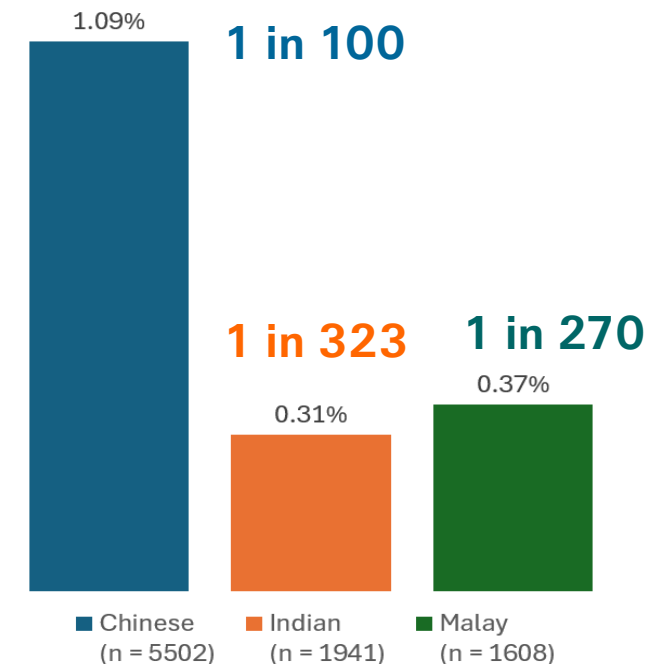
Estimated **prevalence of FH** in **Singapore**

1/140



..and are **at risk** of **premature heart attack** due to these genetic mutations.

From the analysis of the SG10K database, the **adjusted carrier frequency** of **pathogenic or likely pathogenic LDLR** and **APOB** gene :



1. Analysis of clinically relevant variants from ancestrally diverse Asian genomes. *Nature Communications* (2022)13:6694.
2. <https://www.npm.sg/partners/sg10k/>

FH Genetic Tests : Predictive or Diagnostic?

Why **binary classification alone** can be oversimplified

Penetrance describes whether gene carriers of a condition develop features of the condition.

FH penetrance is **> 90%**. About **9 out of 10** carriers of the altered gene will express the phenotype.

Expressivity describes the **degree or severity** with which a specific genotype manifests as a phenotype.

Question:

Are FH Genetic Tests '**Predictive**' or '**Diagnostic**'?

- A. Predictive
- B. Diagnostic
- C. Predictive and/or Diagnostic
- D. Unsure

Penetrance



■ FH carrier : **phenotype present**

■ FH carrier : **phenotype not present**

Expressivity



FH mutation carriers with phenotypic expression of **varying degree of severity**

FH Shows Why the Line Blurs

A diagnosis can be built from family history, LDL-C, clinical signs **or genetic testing**

- FH can be diagnosed without genetic testing, using LDL-C, clinical history, physical signs, and sometimes family history.
- **But** a pathogenic variant from genetic testing can itself support a **definite FH diagnosis**.

Family history		Score
First-degree relative with known premature coronary and/or vascular disease (men aged <55 years and women aged <60 years)		1
or		
First-degree relative with known low-density lipoprotein-cholesterol (LDL-C) above the 95th percentile for age and sex		
First-degree relative with tendinous xanthomata and/or arcus cornealis		2
or		
Children aged <18 years with LDL-C above the 95th percentile for age and sex		
Clinical history		Score
Patient with premature coronary artery disease (ages as above)		2
Patient with premature cerebral or peripheral vascular disease (as above)		1
Physical examination		Score
Tendinous xanthomata		6
Arcus cornealis prior to 45 years of age		4
LDL-C (mmol/L)		
	LDL-C ≥8.5	8
	LDL-C 6.5–8.4	5
	LDL-C 5.0–6.4	3
	LDL-C 4.0–4.9	1
Deoxyribonucleic acid (DNA) analysis: Functional mutation in the low-density lipoprotein receptor (LDLR), apolipoprotein B (APOB) or proprotein convertase subtilisin/kexin type 9 (PCSK9) gene		8

Dutch Lipid Clinic Network Criteria (DLCN):
DLCN considers both **non-genetic** and **genetic routes to diagnosis**.

Physical examination		Score
Tendinous xanthomata		6
Arcus cornealis prior to 45 years of age		4
LDL-C (mmol/L)		
	LDL-C ≥8.5	8
	LDL-C 6.5–8.4	5
	LDL-C 5.0–6.4	3
	LDL-C 4.0–4.9	1
Deoxyribonucleic acid (DNA) analysis: Functional mutation in the low-density lipoprotein receptor (LDLR), apolipoprotein B (APOB) or proprotein convertase subtilisin/kexin type 9 (PCSK9) gene		8
Stratification		Total score
Definite familial hypercholesterolaemia (FH)		≥8
Probable FH		6–7
Possible FH		3–5
Unlikely FH		<3

ApoB, apolipoprotein B; DNA, deoxyribonucleic acid; FH, familial hypercholesterolaemia; LDL-C, low-density lipoprotein-cholesterol; LDLR, low-density lipoprotein receptor; PCSK9, proprotein convertase subtilisin/kexin type 9

RACGP Red Book Appendix 2B; Watts GF et al. *Atheroscler Suppl.* 2011;12(2):221-263.

Has the diagnosis moved earlier?

A DMD case challenging the predictive-diagnostic divide

Question:

For the 3-year-old boy, how should this result be viewed?

- A. Purely predictive
- B. Predictive by moratorium, but biologically closer to pre-symptomatic diagnosis
- C. Purely diagnostic
- D. Unsure

Genetic Test Results:

Indication:

Chromosomal microarray analysis in recent pregnancy showed a male profile with a pathogenic loss of 0.201Mb at Xp21.1, causing deletion of exons 10 to 19 of the *DMD* gene. To check if this was an inherited or de novo copy number loss.

Results:

MLPA analysis showed heterozygous deletion of exons 10 to 19 in the *DMD* gene. This is an out of frame deletion.

Interpretation:

This result shows that [redacted] is a carrier of *DMD* gene deletion which is pathogenic. Her children are at 50% risk of inheriting the X-chromosome with the deletion in the *DMD* gene. Pathogenic variants in *DMD* gene have been associated with X-linked Duchenne muscular dystrophy, X-linked Becker muscular dystrophy and X-linked dilated cardiomyopathy 3B collectively known as dystrophinopathies.

Case

38F applied for USD 350K Life and CI
Same application submitted for her 3-year-old son

At application, both mother and son were asymptomatic

Genetic finding furnished

Mother and son both had a pathogenic DMD variant

The Challenge?

Under the moratorium: predictive at application

Son: molecular diagnosis established, phenotype still pre-symptomatic

Mother: carrier status known, phenotype is more variable

When cause is found before disease is seen

Genomics can move diagnosis earlier, but interpretation still depends on clinical correlation



Molecular Diagnosis

What is the genomic cause?

- Pathogenic variant identified
- Genomic cause found



Clinical Correlation

Does the genomic finding fit the clinical picture?

- Phenotype fit, age/stage, family history
- Shapes interpretation of the finding



Clinical Diagnosis

Is the disease clinically present?

- Symptoms, signs, labs, imaging
- Disease clinically present

1. Phenotypic compatibility and specificity in genomic variant classification. *European Journal of Human Genetics* (2024) 32:471-473
2. Best practices for the interpretation and reporting of clinical whole genome sequencing. *npj Genomic Medicine* (2022) 7:27

How Markets Draw the Line

Australia: Statutory ban from 2026 – no protection limits, broader definition of ‘genetic testing’

Position: Prohibition by statute
Framework: Treasury Laws Amendment
Commences: 8th October 2026
Shift: From moratorium to legislation

1) No financial thresholds

- No sum-assured limits
- No above-limit whitelist
- Applies to all L&H products

2) ‘Genetic Testing’ is broader

- DNA / RNA / chromosome analysis
- Also includes epigenetic tests
- Also proteins, biomarkers, metabolites

3) Protected genetic information

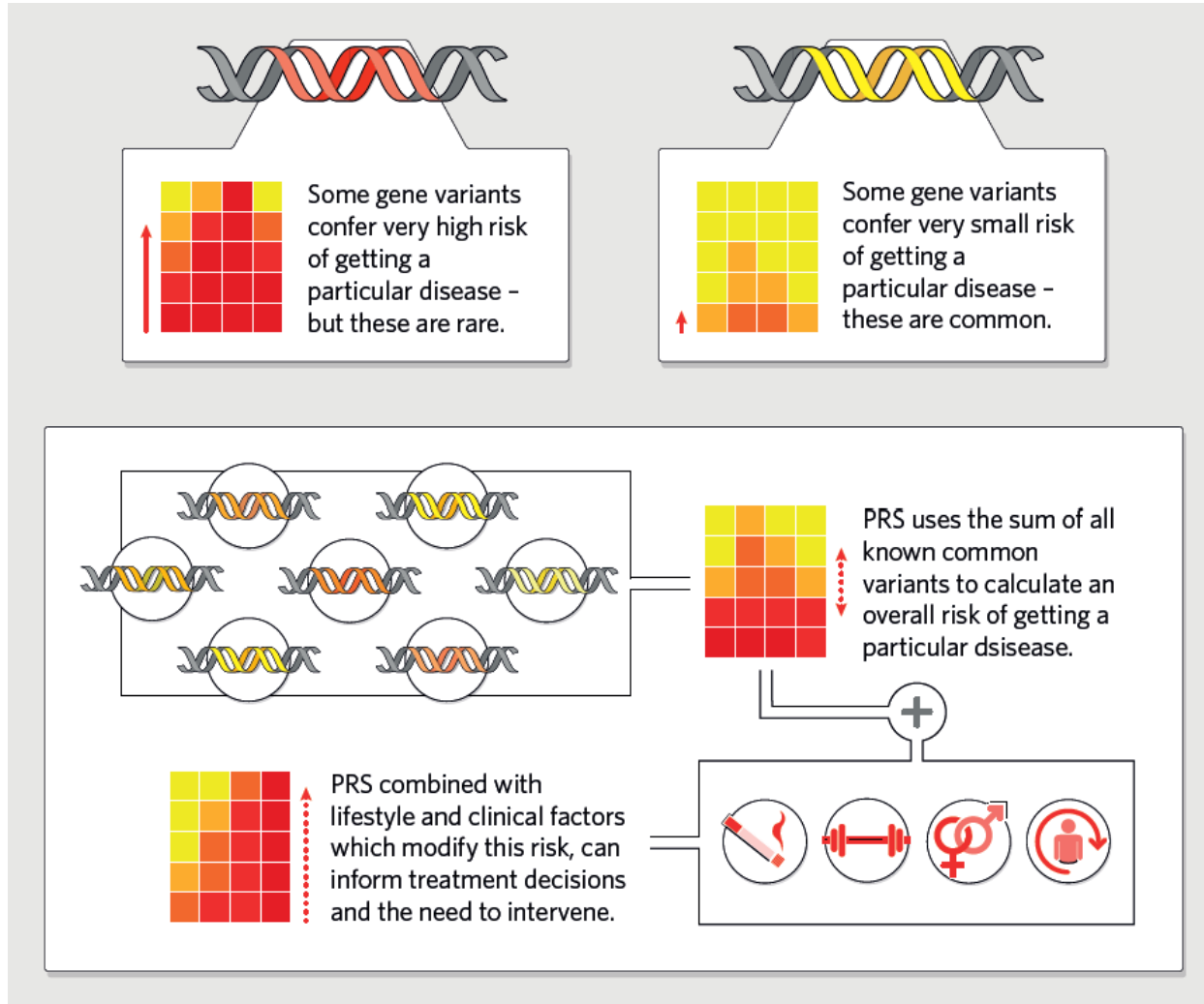
- Covers own and relative’s testing
- Covers actual test results
- Separate from clinically diagnosed disease

Australia replaces ‘threshold-based exceptions’ with a statute that bars soliciting or using protected genetic information.

Source: Treasury Laws Amendment (Genetic Testing Protections in Life Insurance and Other Measures) Act 2026 (Australia), No. 35 of 2026.

From single-gene variants to cumulative genetic risk

Polygenic Risk Score (PRS) adds many small genetic signals into one risk score



Monogenic Risk

Rare variant, large effect.

Polygenic Risk

Many common variants, small effects, added together.

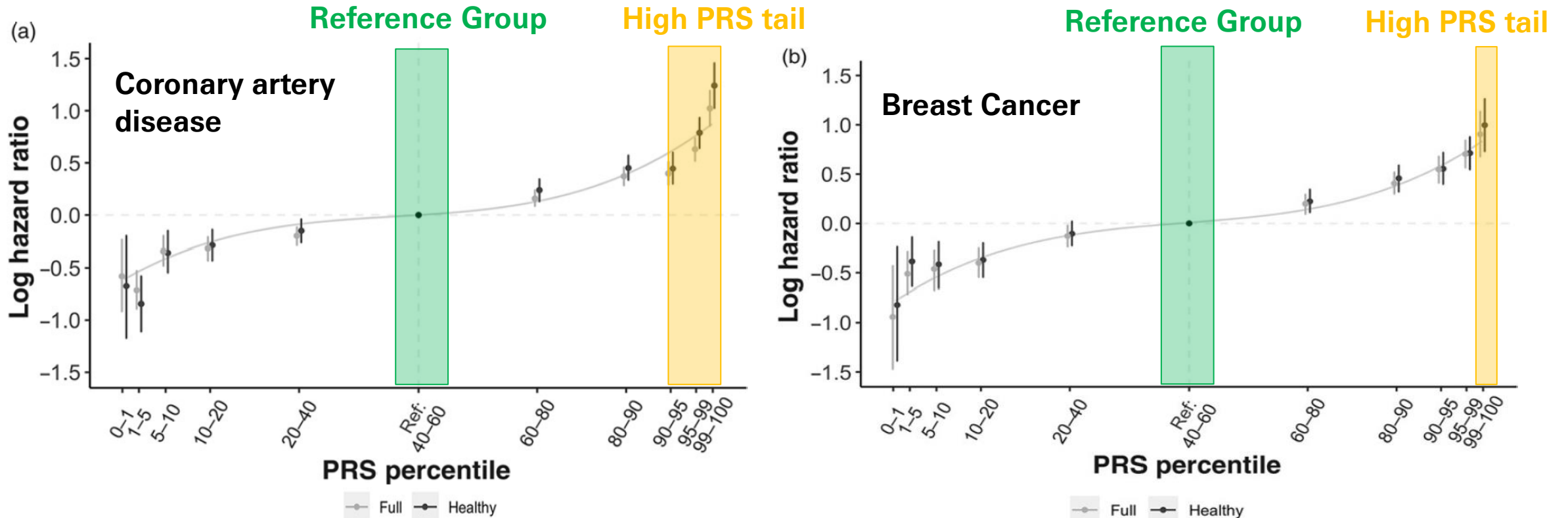
Clinical Meaning

Risk stratification, not disease confirmation.

Visual adapted from Nature Research Custom Media for Illumina, "Polygenic Risk: What's the Score?" Advertisement feature, 2019

PRS creates a measurable risk gradient

Higher PRS, higher future CAD and breast cancer risk, beyond standard risk factors



CAD: Top PRS decile associated with ~2x risk vs average PRS

HR 1.97 [95% CI 1.80 – 2.16] in 'Healthy' cohort

Breast Cancer: Highest PRS bands associated with >2x risk vs average PRS

HR 2.56 [95% CI 2.05 – 3.19] for > 99th percentile; HR 2.05 [95% CI 1.79 – 2.35] for 95-99th percentile.

Source: Maxwell et al. (2021), Annals of Actuarial Science, 15, 488-503.

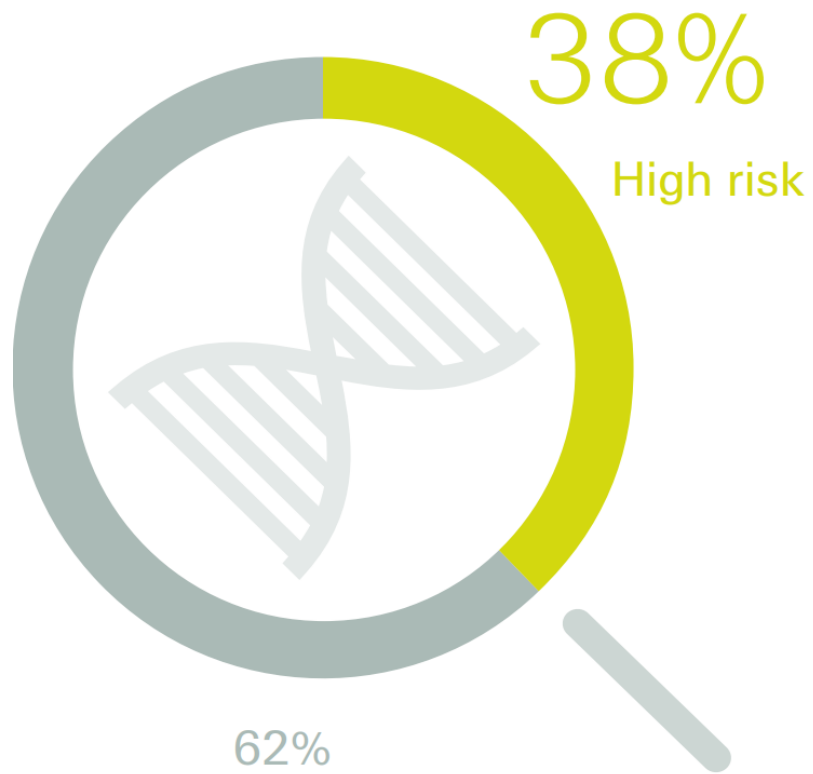
Full: UK Biobank cohort

Healthy: Filtered subgroup approximating **standard-risk** life insurance population.

When Detection Moves Upstream

Why underwriting, products and positioning must now evolve





▶ **4x**

Those who are at high risk are four times more likely to buy insurance



2019 Swiss Re Consumer Survey : Directional Signal

- ~36K respondents across 5 markets **USA, Canada, UK, China and Australia.**
- ~35% said test results influenced **their decision to buy new or additional cover.**
- Among those tested, 38% reported a high-risk result. This group was **~4x more likely** to buy cover.

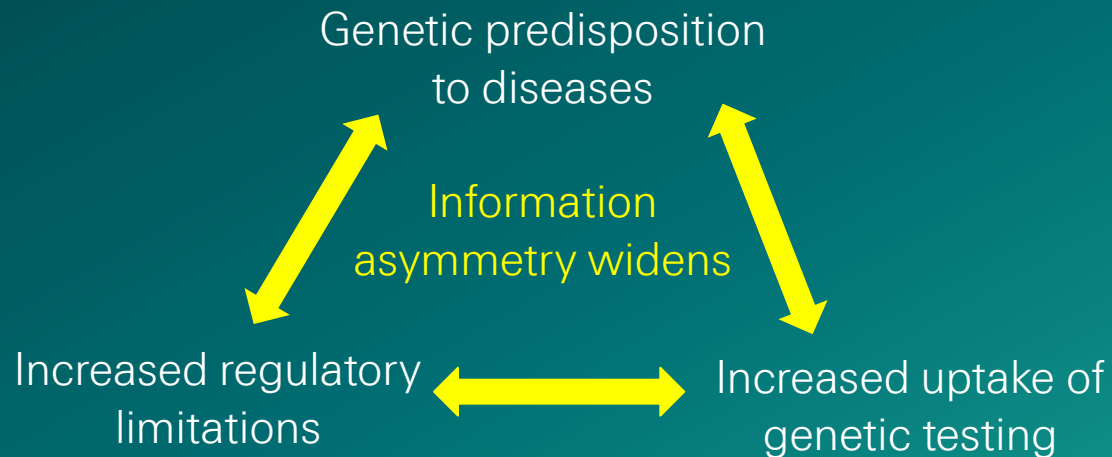
Directional implication:

As consumers gain access to risk information earlier, information asymmetry may also move upstream.

Family History is becoming harder to ignore

As regulators place tighter limits on genetic information, family history may carry greater underwriting relevance

Why it still matters?



Family history may carry greater underwriting materiality

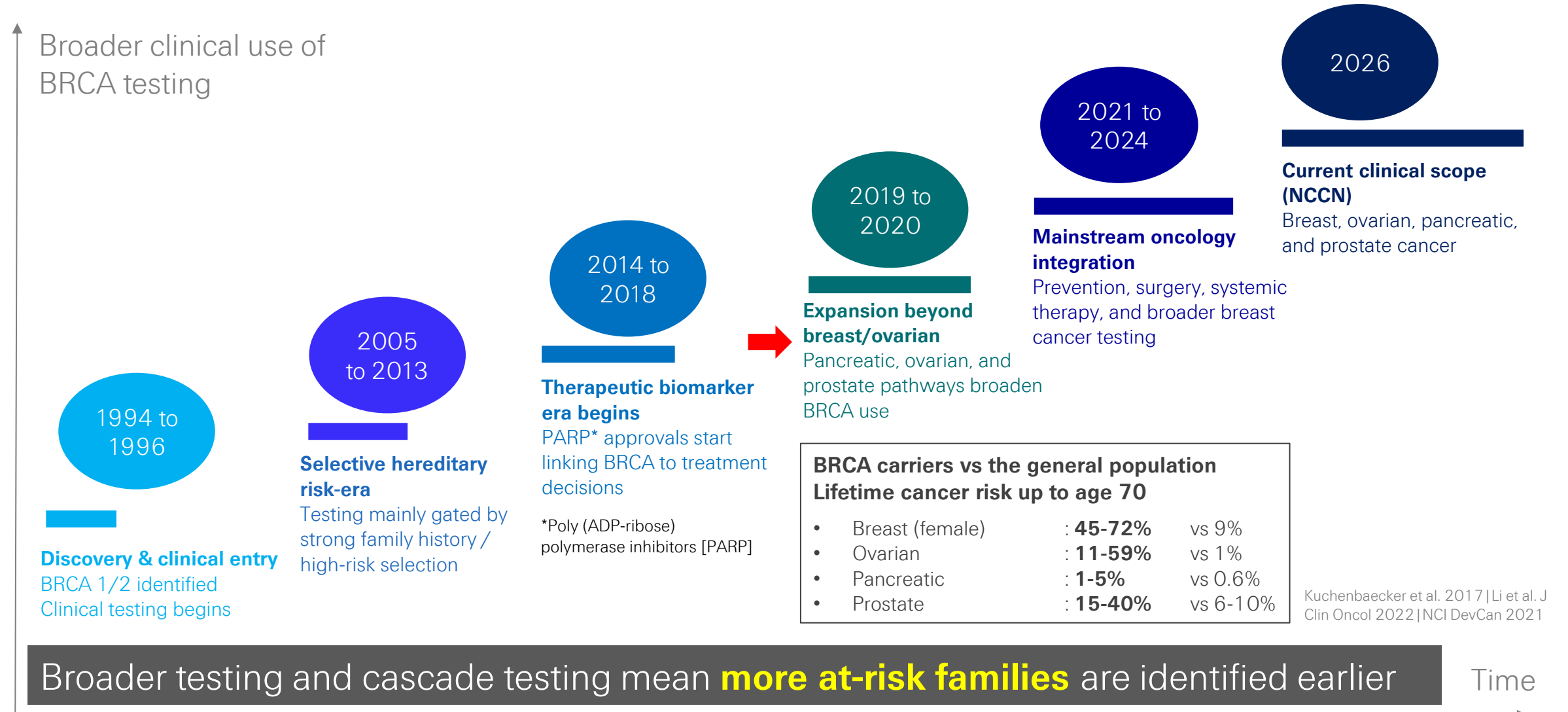
What's challenging then?

- Disclosure may be **incomplete** or **inaccurate**
- **Verification** is often **difficult** in fragmented health systems
- Use is permitted in many markets, but **restricted** in **others** (e.g. Japan and Korea)

Family history may matter more, but it still needs disciplined, market-aware use.

BRCA testing has broadened over time

From selective hereditary-risk testing to mainstream oncology care



Source: Science (1994) | Nature (1995) | USPSTF (2005, 2013, 2019) | NICE (2013) | FDA approvals (2014, 2018, 2019, 2020, 2022) | ASCO (2020, 2024) | NCCN (2026)

**If family history is going to
matter more,**
we need underwriting guidance
that can work with that reality.



How Life Guide approaches family history

Breast, ovarian, and prostate histories are assessed as related patterns, even where genetic test use is restricted.

- **Related family histories** are **not treated** as isolated entities.
- This creates a more **medically coherent** view of **BRCA-linked** family patterns.

Family history of ovarian cancer + breast cancer in 1st degree relatives

Risk classification	Life	ADB	CI	TPD	DI
Family history of ovarian cancer in addition to a history of other cancers in two or more first degree relatives:					
Breast cancer	→	Rate as: <u>Family history of breast cancer</u>	+0		Rate as: <u>Family history of breast cancer</u>

Family history of prostate cancer + breast cancer in 1st degree relatives

Risk classification	Life	ADB	CI	TPD	DI
One or more first degree relatives with prostate cancer of any age, and an additional first degree relative of breast cancer less than age 50					
				→	Rate as: <u>Family history of breast cancer</u>

Family history of breast cancer + prostate cancer in 1st degree relatives

Risk classification	Life	ADB	CI	TPD	DI
Male applicant:					
1 or more first degree relatives with breast cancer equal to or greater than age 50, and 1 or more first degree relatives with prostate cancer of any age	+0	+0	→	Rate as: <u>Family history of prostate cancer</u>	+0

How Life Guide approaches family history

A favourable genetic result can support a more favourable outcome, but only when the result is truly informative.

- Life Guide recognizes a truly **informative negative result** and can support a more favourable underwriting outcome.
- A reported negative result **outside that context** may still need to be interpreted alongside **underlying family history**.

Examples from LG: Family history of breast, ovarian and prostate cancer

Risk classification	Life	ADB	CI	TPD	DI
Gene test result, when use allowed by legislation or industry guidelines:					
Positive for BRCA 1 or 2 gene mutation	+0	+0	Refer Medical Officer, consider prostate and breast cancer exclusion	+0	+0
Negative*	+0	+0	+0	+0	+0

Excerpt from LG: A negative result matters only if it is a **truly informative negative**

*For a gene test to be regarded as negative, it has to be known that the affected relatives have had a specific gene mutation identified and that the applicant has tested negative for that specific mutation. Only in such cases can the exclusion or rating be waived. When a negative gene test is reported other than in this context, the exclusion still applies.

Healthcare is moving upstream

Many APAC health systems are moving upstream, as prevention, prediction, and earlier intervention become more prominent

The dominant legacy model:

Sick → Diagnose → Treat



The emerging direction:

At Risk → Detect → Intervene Early

The same diagnosis may no longer mean the same need

Earlier detection changes what a diagnosis represents and why this becomes a product question

Legacy world

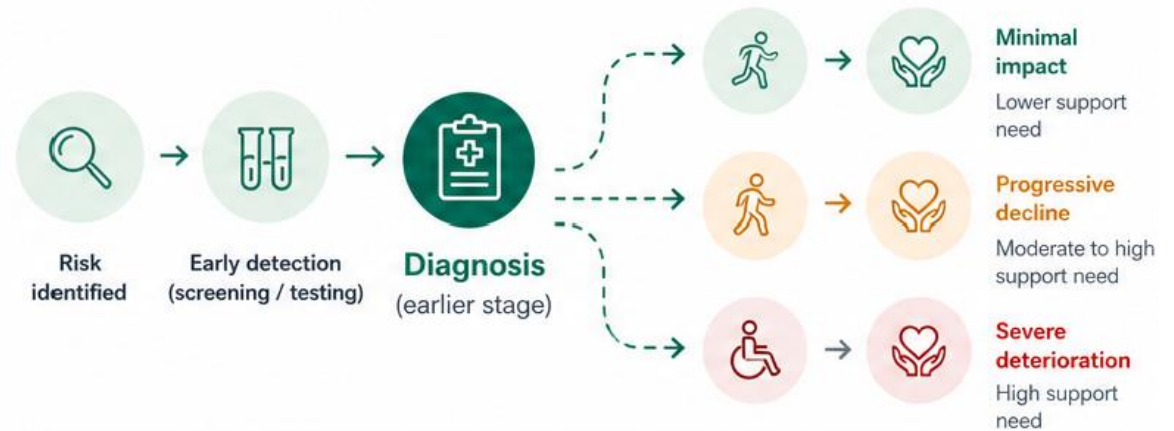
Diagnosis occurs later in the disease pathway



vs.

Upstream world

Diagnosis can occur earlier, before major functional loss



Payout aligns well with real need

Diagnosis is a stronger proxy for severity, function and support need



Payout may misalign with real need

Same diagnosis, but different severity, function and support implications

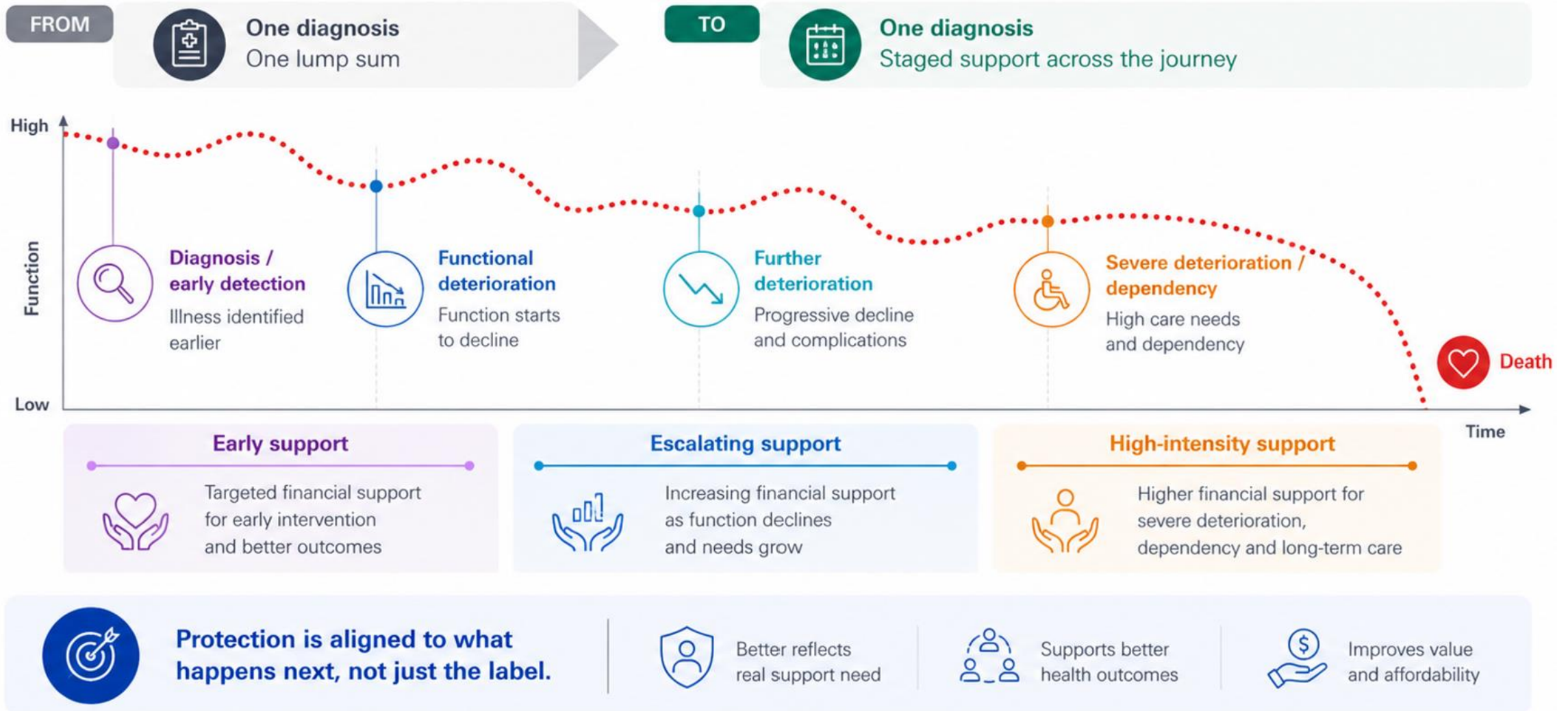


Same trigger.
Different meaning.

As healthcare moves upstream, diagnosis alone may say less about severity, function and real need, making this a **product design question**.

What a more future-ready protection logic could look like

Shifting from a single diagnosis trigger to staged support aligned with function and trajectory



Earlier Risk Visibility Will Reshape L&H

**The legacy
healthcare pathway**

Today's L&H operating model

- Propositions remain reactive and diagnosis-led
- Pricing is calibrated to historical trajectories
- Underwriting and claims rely heavily on declared disease status

1 Risk is becoming visible earlier

Detection is moving upstream, often before symptoms appear.

**The
prevention-
first
healthcare
model**

3 Strategic positioning now matters

L&H must decide how to respond as prevention, prediction, and earlier intervention expand.

2 The legacy model will be tested

Earlier risk visibility may mean today's pricing, product, underwriting, and claims frameworks need to evolve.



Key Takeaways

Three messages to guide our way forward



1 Risk is becoming visible earlier

Precision health and genomic infrastructure are moving detection upstream, often before disease manifests.



2 Genetic limits require credible adaptation

When regulators tighten genetic information use, the industry must communicate transparently about information asymmetry and show it can assess risk responsibly.



3 The next challenge is not just underwriting, but protection design

As diagnosis arrives earlier and means less on its own, protection logic must evolve with better anchors than diagnosis alone.

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