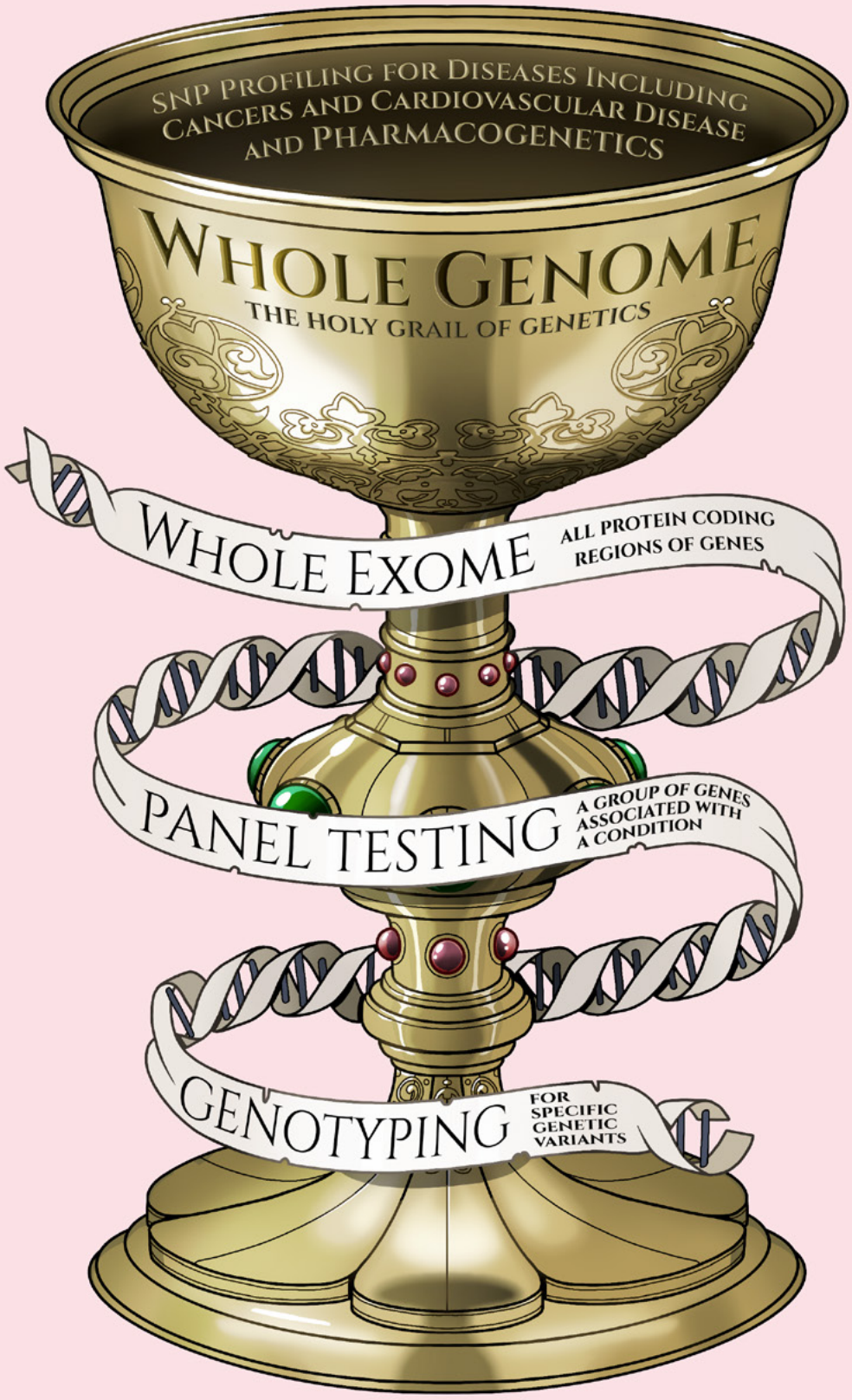


The London Genetics Centre
90 Sloane Street

Veritas Intercontinental

Contents

Our Two Main Genetic Testing Pathways

THE LONDON GENETICS CENTRE
SLOANE STREET 90

Our aim is to save lives and reduce the suffering of illness and treatments. We test for Single Risk Genes and Polygenic Risk Scores to find areas of personal genetic risk where we can significantly lower your risk, and that of your children and grandchildren.

We offer two main genetic testing pathways:

PREVENTIVE SCREENING - Uses whole exome sequencing technology to analyse the following actionable genes: 84 cancer and 81 cardiac genes, 4 other key genes and 13 Polygenic Risk Scores (PRS); 12 for females and 9 for males. This pathway is run by our Genetic Counsellor alongside a Consultant Geneticist who overviews all results and family histories.

WHOLE GENOME SCREENING - This is a more extensive test, including 583 selected actionable genes and 13 PRSs (as above), combined with a full medical, bloods, echocardiogram, ECG and an abdominal/pelvic ultrasound. Our Genetic Consultants review your results in a multidisciplinary meeting, a summary doc - ment is produced, and an appointment with one of the Consultant Geneticists concludes the pathway.

The Psychology of Genetic Testing

For many of us we may choose to avoid things that we fear, burying our head in the sand. But this attitude can be dangerous, putting us at a greater risk and leads to diagnostic delay.

When we do targeted screening, **we are only looking for gene changes where, if we find them, we can do something positive to improve future health.** It does require a philosophical leap, but it is one that will potentially save lives if we can help the world to understand the enormous gains.

The modelling studies show instituting genetic screening and acting on the results should reduce the **cancer mortality** in the general population by nearly a seventh. The sooner genetic screening is actualised, huge numbers of lives could start being saved.

Whole Exome Sequencing vs Whole Genome

WHOLE EXOME SEQUENCING

Our Preventive Screening is done via Whole Exome Sequencing (WES), which is the sequencing of all the **coding parts** of our genetic information.

This targeted approach means we are only analysing **actionable gene changes:** if we find you have any harmful changes in these genes, then we can do something to improve your outlook. It is important to note that some smaller risks associated with these gene changes may **not** be actionable. We **do not** analyse the neurological genes such as those related to Parkinson's disease or dementias currently.

WHOLE GENOME SEQUENCING

Whole Genome Sequencing (WGS) is the sequencing of your **entire genome** (all our genetic information). With Veritas, we have selected a targeted 583 genes to analyse relating to more than 650 conditions, including **116 recessive carrier genes.** Recessive carrier genes will not usually have any implications for your own health but could be relevant to your wider family. WGS also includes **pharmacogenes**, the genes that control how we, as individuals, respond to different medicines.

With our WGS we double check the result by doing WES in addition. WES has the benefit of not missing large gene deletions which can happen on rare occasions with WGS.

The 5 Commandments of Genetic Screening

- **Accuracy** of the highest quality
- **Actionability** – we are only testing for genes you can do something about - we are **not** doing Parkinson's or dementia genes currently
- **Full medical background and family history** known to geneticists
- **Genetic Consultant(s) reviews all results**
- **Informed consenting and feedback** of results by a genetically trained professional

Veritas Intercontinental : Our Partners

Veritas provides the sequencing and interpretation of your whole exome and whole genome and produces a comprehensive report of the information. This information is then further analysed and interpreted by our geneticists, generating a personalised action plan for each of our patients.

Working collaboratively with Veritas over the last three years has given us an exceptional pioneering developmental opportunity. Genetics really is teamwork.

The London Genetics Centre has an exclusive UK agreement with Veritas Intercontinental.

Insurance

In the UK the Association of British Insurers (ABI) has a trade agreement. (www.abi.org.uk).

All members of the ABI are signed up to the code on genetic testing and insurance. They will not ask for or take into account the result of a predictive genetic test, with the only exception being Huntington's disease. If you are doing genetic testing related to a condition you are affected with or develop in the future, they can ask for the genetic results associated with that specific condition.

Requirements for insurance companies outside of the UK may differ.

The Preventive Screen - What Does It Test?

Making genetics more affordable.

Combines single gene changes & Polygenic Risk Scores - our genetic risk comes from both these areas

84 Cancer Genes
81 Cardiac Genes

4 additional health genes

Iron overload (Haemochromatosis)
Factor V Leiden and F2 Prothrombin (clot risk)
Alpha 1 Antitrypsin Deficiency (emphysema and cirrhosis)

Polygenic Risk Scores

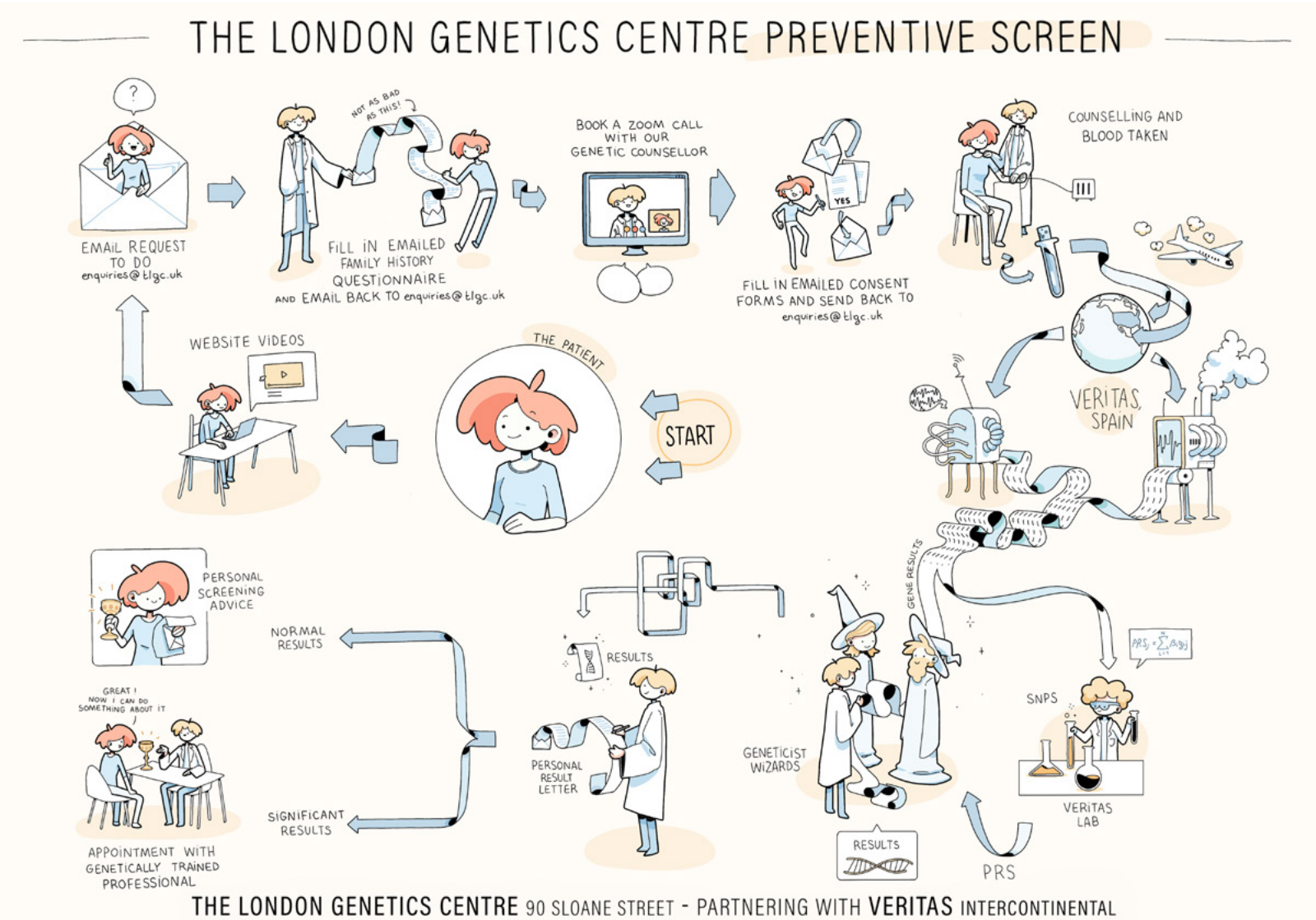
Cancer PRS
Breast cancer
Prostate cancer
Colorectal cancer
Ovarian cancer
Kidney cancer
Melanoma

Cardiovascular PRS
Coronary Artery Disease
LDL cholesterol
Hypertension

Other conditions
Osteoporosis
Early menopause
Coeliac disease
Type 2 Diabetes

High quality genetic testing combined with a team of over 185 years of genetic expertise.

The Preventive Screen Pathway



Cancer and Cardiac Conditions - Examples

Cancers

20-30% are at least due to hereditary factors depending on the type of cancer.

***BRCA* gene changes and Lynch Syndrome:** less than 10% of people with these genetic changes know that they have them, so they are utterly unaware of their risk profile. If they have genetic knowledge, however, their outlook is vastly improved compared to those who are unaware.

***BRCA* gene changes occur in 1 in 250 people,** (increasing to 1 in 40 in the Ashkenazi population). They confer a 60-85% chance of breast cancer and 20-60% chance of ovarian cancer in a lifetime. Prophylactic ovary and fallopian tube removal is a day case procedure and reduces ovarian cancer risk by 95%.

Lynch syndrome affects 1 in 250 people and causes several cancer types, particularly bowel and endometrial (uterine) cancer. Knowing it is present improves the mortality rate by 25%. Taking aspirin daily reduces colon cancer by 40% in these patients, and this gene alteration also means that they respond exceptionally well to immunotherapy.

Pre-natal Genetic Testing (PGT) is one of the ways these conditions can be prevented. PGT can be used to avoid over 600 conditions as stated by The Human Fertilisation and Embryology Authority (HFEA), a professional body in the UK which regulates the use of embryos in fertility treatment and research.

Sudden cardiac deaths

20% are due to genetic abnormalities related to cardiac muscle and cardiac rhythm disorders, or aortic aneurysms.

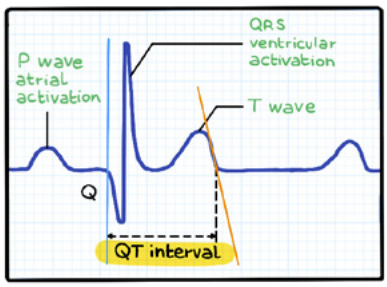
Hereditary thrombosis

6% of the population is at a 4 to 5- fold increased risk. Most people are typically unaware of this risk, until they develop either a deep vein thrombosis, a potentially fatal pulmonary embolism or in some circumstances, a stroke.

Cardiac Genetics

Inherited Arrhythmia Syndromes

These can cause sudden cardiac death through a dangerous rhythm called ventricular tachycardia. Long QT Syndrome occurs in 1 in 2,000 people, with a specific genetic subtype identified in 75% of cases. Genetically defined drug treatment and risk factor avoidance advice can then be instituted.



The electrocardiogram (ECG) shows the QT interval. Many patients respond to beta blockers. QT prolonging drugs include antidepressants, macrolide antibiotics, antihistamines, and should be avoided.

Dilated Cardiomyopathies

These are a common cause of heart failure and the need for transplantation. A gene change is identified in 15-25% of cases.

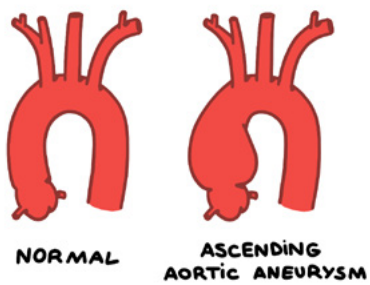
Hypertophic Cardiomyopathy

These occur in 1 in 500 people where the heart muscle is thickened. A gene change is identified in 30-60% of cases. We perform an echocardiogram in all Whole Genome Screening patients as this is an integral part of cardiac screening.

Aortic Aneurysms

The aorta enlarges with the potential to **rupture**, or the wall can **dissect** with a tear in the inner lining, causing blood to flow between the layers. Aneurysms occur in the abdomen and chest; their progression can be reduced, or they may be operable.

Ascending aortic aneurysms: 20-25% of cases have specific gene changes. Known associations include Ehlers-Danlos Syndrome, Marfan Syndrome, Loeys-Dietz Syndrome and Familial thoracic aortic aneurysms.



Aneurysm rupture is usually catastrophic. Preventive surgery is generally carried out on those with 5 and 5.5 cm aneurysm diameter. The specific gene type influences the timing, some aortas being more vulnerable.

Gene-Associated Risk Chart

TYPE OF CANCER:	BREAST (female)	OVARIAN	PROSTATE	PANCREAS	OTHER
UK Cancer deaths per year (2017-2019)	**11,400**	**4,142**	**12,039**	**9,558**	-
Lifetime risk of cancer in general population	12%	2%	18%	2%	-

Prevalence	MAJOR BREAST CANCER GENES:	BREAST	OVARIAN	PROSTATE	PANCREAS	OTHER
1/381 1/40 with Ashkenazi ancestry	***BRCA1***	85%	40-60%		1-3%	
1/277 1/40 with Ashkenazi ancestry	***BRCA2***	80%	17-25%	18-25%	higher risk with positive family history 2-7%	Melanoma 3-5%
1/770	***PALB2***	44%	5%		2-3%	
1.8/1000	***BARD1***	25%	7%			
1/3555 1/5476 90% chance of cancer by age 60	****TP53***	90%			10%	Soft tissue 20%; Brain 15%; Bone 10%
1/200,000	****PTEN***	67-85% av. age of diagnosis in the 40s'				other: Melanoma 5%; Kidney 30%; Thyroid 15-35%; Uterus 25%; Bowel 9%
1/25,000-1/280,000	****STK11***	32-54%	10-21%			
1/100	****ATM***	20-25%		40%	6%	
1/100-1/200	****CHEK2***	25%		25%		Bowel 12.5%
1/1,000,000	****CDH1***	39-52%				Stomach 45%

(XX%) Number in circle represents lifetime risk of developing respective cancer for each gene change

* Large variation in lifetime risk: dependent on type of gene change found and family history

** Encompasses kidney/ureter and bladder cancers. Risks vary between different organs

For a more etensive list of the genes included in our preventive Panel and their associated risks please see our website: https://thelondongeneticscentre.com/

There may be additional cancer risks associated with a given gene change where % lifetime risk is currently under discussion. Therefore, recommendations based on a test result may differ from what is suggested by the table.

TYPE OF CANCER:	BOWEL	UTERUS	OVARIAN	STOMACH	OTHER
UK Cancer deaths per year (2017-2019)	**16,808**	**2,453**	**4,142**	**4,216**	-
Lifetime risk of cancer in general population	6%	3%	2%	1%	-

*In addition to **ATM, CHEK2, STK11** and **PTEN** genes as above*

MAJOR BOWEL CANCER GENES:

Prevalence			Bowel	Uterus	Ovarian	Upper GI tract	+Urinary tract	Brain	Prostate
1/200,000	Lynch syndrome	***MLH1***	35-65%	37%	11%	18%	*5-7%	*2%	
1/3000	Lynch syndrome	***MSH2***	47-51%	50%	17%	17%	15%	*3-8%	24%
1/800	Lynch syndrome	***MSH6***	20%	41%	11%	11%	*1-8%	*1-2%	24%
1/714	Lynch syndrome	***PMS2***	13%	13%					
1/13,500		***APC***	70-95%						
1/40,000 (2 gene changes)		***MUTYH***	70%						
very rare		***BMPR1A***	30%			15-21%			
very rare		***SMAD4***	10-38%			15-21%			

*In addition to **BRCA1** and **BRCA2** gene changes – as above which are major contributors, lesser contributing gene changes include **PALB2, BARD 1, PSTK11** as well as some of the Lynch syndrome genes above.*

MAJOR OVARIAN CANCER GENES:

			Ovarian		Breast
1/416		***BRIP1***	9%		possible increase
very rare		***RAD51C***	10%	triple negative	20-40%
very rare		***RAD51D***	10-20%		20-40%

The aim of this chart has been to communicate gene risk association, but we should add there are many more genes and conditions which are not included for reasons of space. Please refer to our website https://thelondongeneticscentre.com/ for wider coverage.

Reproductive Options and Pharmacogenomics

Germline changes refer to the DNA you have in every cell of your body that you are born with, whereas **somatic changes** are seen in tumour cells and cannot be inherited unless they are directly related to your germline.

Genetic Screening could prevent couples who are planning a family from passing on genetic diseases. Couples who have a match for a harmful gene change may have the option to undergo assisted reproductive procedures such as pre-implantation genetic testing (PGT) through IVF. This involves testing embryos for harmful genetic changes shared by the couple, and only returning those embryos that do not carry the harmful gene change for a potential pregnancy.

Pharmacogenes are genes that control how we, as individuals, respond to different medicines. The metabolism of medicine varies greatly from person to person. An individual may have a particular pharmacogene change, causing their body to have adverse reactions to medicine in which they should use an alternative, or may require a larger dose if their body breaks it down quicker than average.

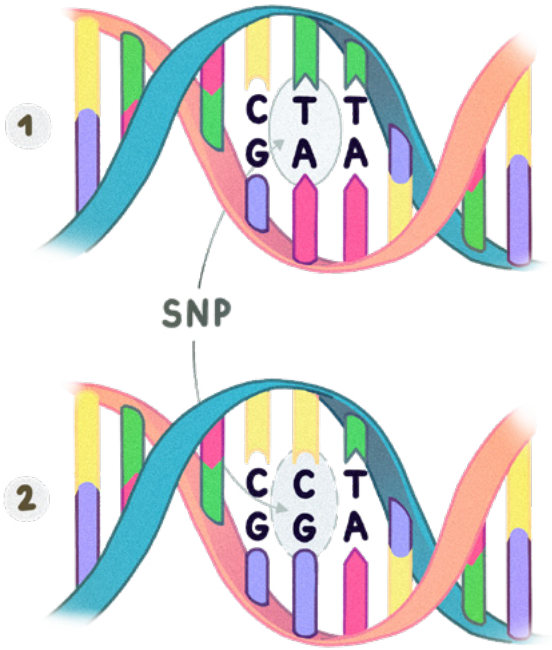
The following demonstrates an example:

Clopidogrel is a drug that stops platelets from sticking together and is used for heart attack and stroke prevention. It is a pro non-active drug that needs to be metabolised into its active component. In some patients, this enzymatic conversion does not occur due to having a pharmacogene change meaning the Clopidogrel is not effective for that patient. This problem affects 1 in 3 patients.

A patient having a coronary artery stent procedure is frequently prescribed clopidogrel and aspirin to reduce the risk of the new stent becoming blocked by a clot. If the patient and/or the doctor are unaware of one of these two preventative drugs being inactive, there is an increased risk of stent thrombosis/occlusion.

Single Nucleotide Polymorphisms (SNPs) and Polygenic Risk Scores (PRS) Explained

Single nucleotide polymorphisms (SNPs) are single base letter changes in the genome. As shown in the diagram, you can see that the T and A pair in the middle have become a C and G – this is a SNP.



SNPs can cause either a very small increased or decreased risk of developing certain diseases. However, when we combine and add up all the SNPs with their individual risk-weighting for a particular condition to form a Polygenic Risk Score (PRS), it can significantly increase the risk for developing a disease.

SNPs differ in the way they are passed down generations compared to single gene changes. SNPs are reshuffled like a pack of cards and randomly dealt out to each child whereas there is a 50:50 or 1 in 2 chance that a single gene change is passed down. This means that if an individual has a high PRS, it does not equate their child to having the same risk.

To identify individuals who have an increased risk of certain conditions, it is important that a genetic team can analyse both single risk genes and PRSs.

Early research indicates that for individuals who have a known single-risk gene change, (eg. *BRCA1/2*), their risk can be substantially modified by the addition of a PRS[1]. For example, a patient with a harmful *BRCA2* gene change has a 25% lifetime risk of prostate cancer when they have a PRS on the 5th centile; this increases to an 85% lifetime risk when on the 95th centile[2]. This exemplifies the contribution of PRSs in risk estimation.

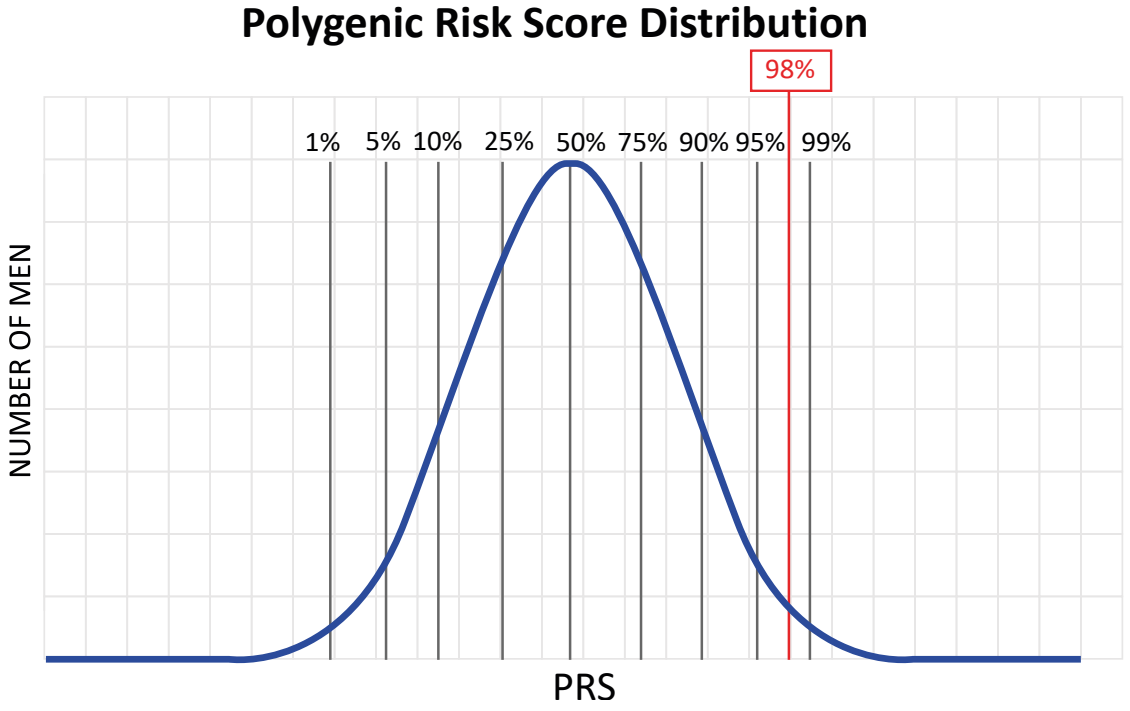
1. Fahed, A., et al. 2020. Polygenic background modifies penetrance of monogenic variants for tier 1 genomic conditions. Nat Commun 11, 3635.
2. Nyberg, T., et al. 2023. CanRisk-Prostate: A Comprehensive, Externally Validated Risk Model for the Prediction of Future Prostate Cancer. J Clin Oncol 41(5), pp. 1092-1104.

PRSs- Prostate & Colon Cancer

Two recent patient examples showing the role of polygenic risk profiles:

1) A 58 year old Scottish patient's * prostate SNP profile gave him a polygenic risk on the 98th centile.

The following chart illustrates the risk of prostate cancer for different PRSs. The final calculation demonstrated that he had **more than a five-fold relative risk of prostate cancer.** Taken together with the patient's other information, he resulted in having a prostate biopsy which showed significant cancer and subsequent prostate removal.



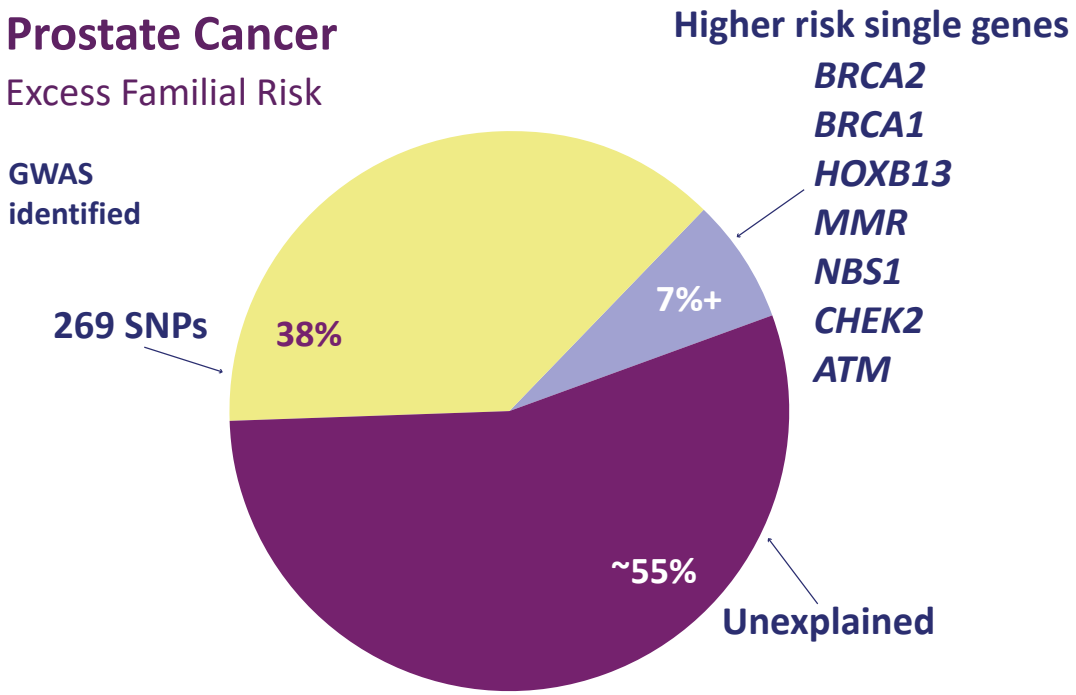
2) A French patient underwent surgery at age 30 for colon cancer (he is now 52). We had initially detected a particular ***APC* gene change** which inferred a two-fold increased risk of colon cancer in comparison to the general population. Given his young age, it was likely there was another contributing factor. His **colon cancer PRS** was then calculated, which conferred a **3.78-fold relative risk.**

*Identifiable characteristics in both profiles are changed for anonymity.

PRSs and Single Changes Combined

Prostate Cancer

The pie-chart below shows that the contribution of SNPs to familial risk is larger (38%) than the contribution of single-risk gene changes.



If we are to best identify people at highest cancer risk, we need to do both single gene analyses and SNPs.

Ovarian Cancer

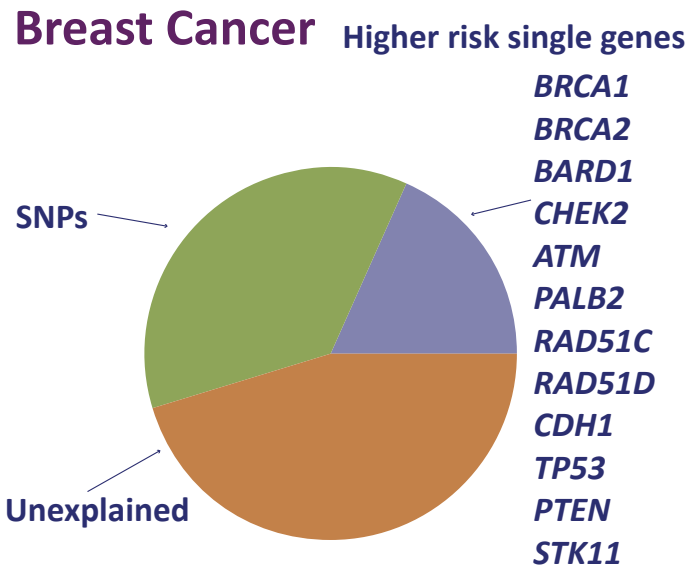
Current ovarian screening with ultrasound and CA125 (a tumour marker) is sadly not effective. Ovarian cancer usually presents when it is widespread and too late for a good chance of cure.

Genetic testing can find those at highest risk of ovarian cancer. If you are at a high genetic risk and have completed your family, you may choose to dramatically reduce your risk to nearly 0% by surgery to remove the ovaries and fallopian tubes.

We could likely save 2000 lives a year lost from ovarian cancer in the UK by using WGS to find those at risk, reducing ovarian cancer by a third.

The charts below elegantly show the concept of what the contribution of genes are compared with SNPs and how this varies for different cancers.

The SNP contribution in breast cancer is more than that of single gene changes, whereas in ovarian cancer SNPs play a small part.
As previously illustrated, SNPs play a dominant role in prostate cancer.



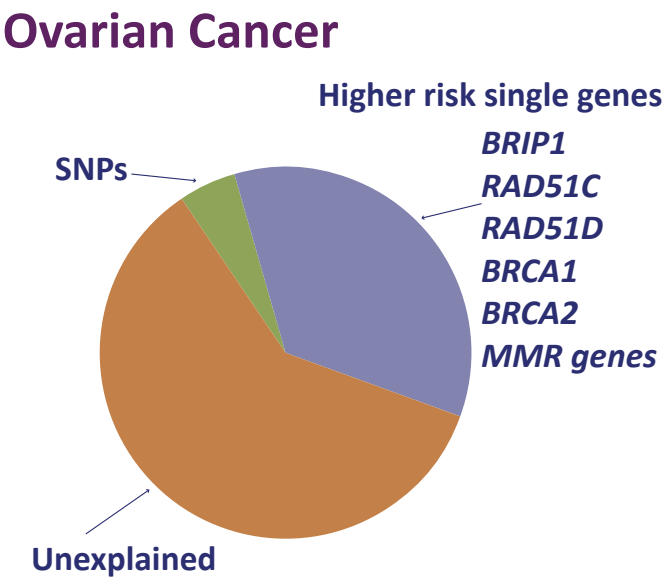


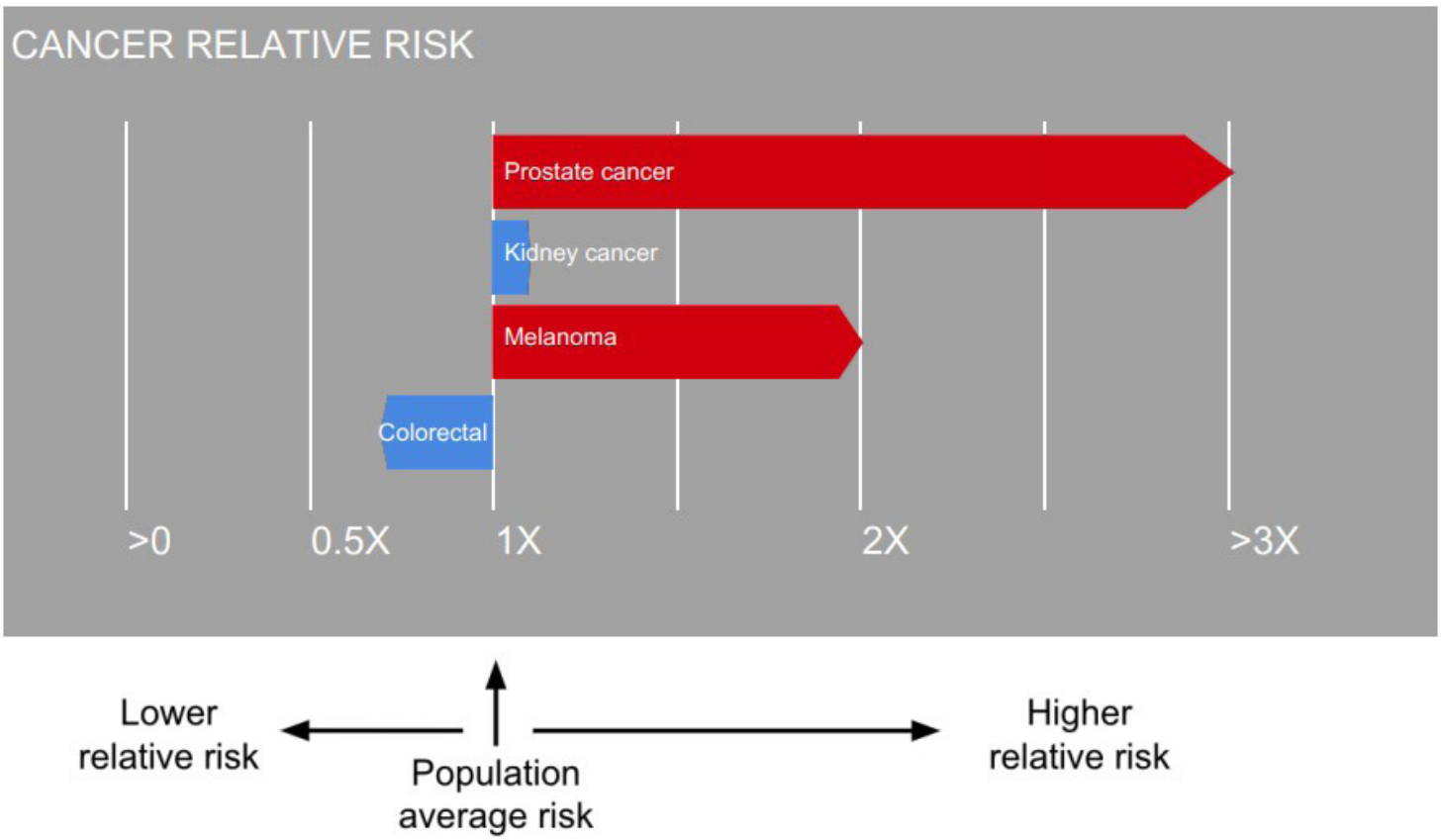
Figure created from amalgamated estimates from publically available data.

Polygenic Risk Score Report

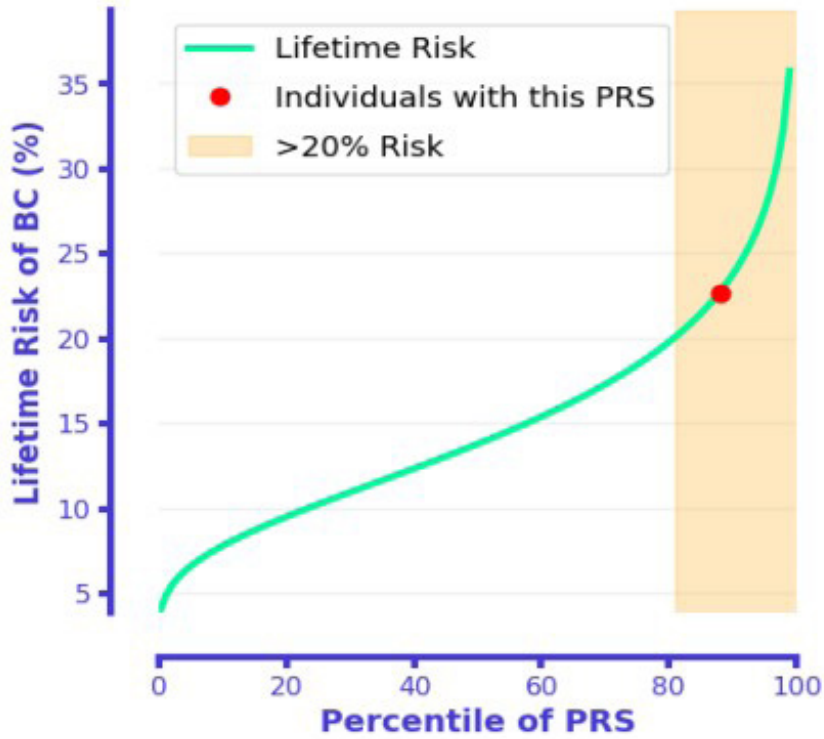
Below illustrates how the PRSs are displayed in the results report. The relative risk indicates your risk in comparison to the average person in the general population (i.e. <1 is lower risk, >1 is higher risk).

CANCER		
DISEASE / TRAIT	PAGE	RELATIVE RISK*
Prostate cancer	5	3.9
Kidney cancer	6	1.1
Melanoma	7	2
Colorectal cancer	8	0.7

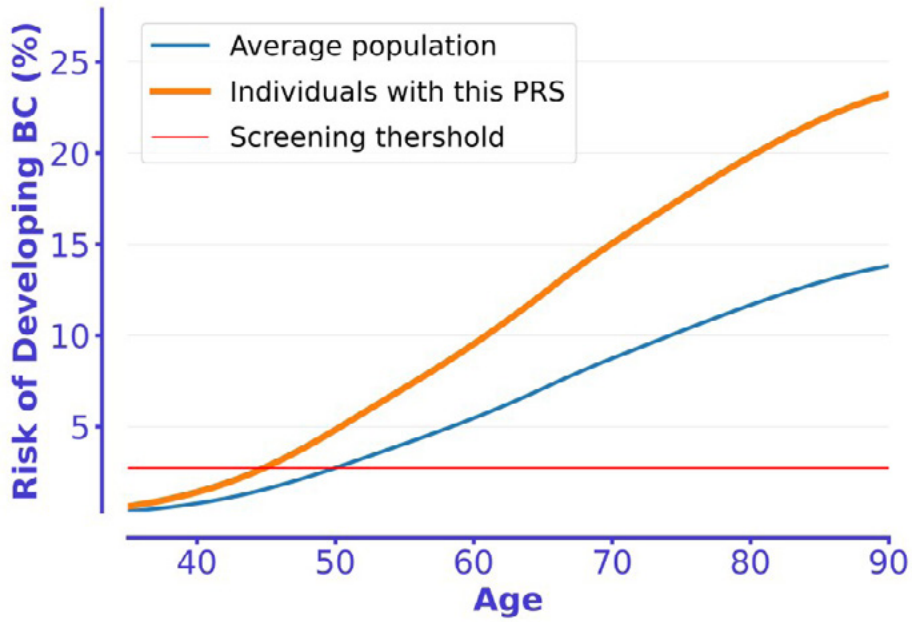
*Relative risk is X fold compared to the population average risk



Breast Cancer PRS on the 89th Centile: This patient's position on the risk curve gives her an 23% chance of breast cancer in her lifetime. If on the 5th centile, it would be 6%.

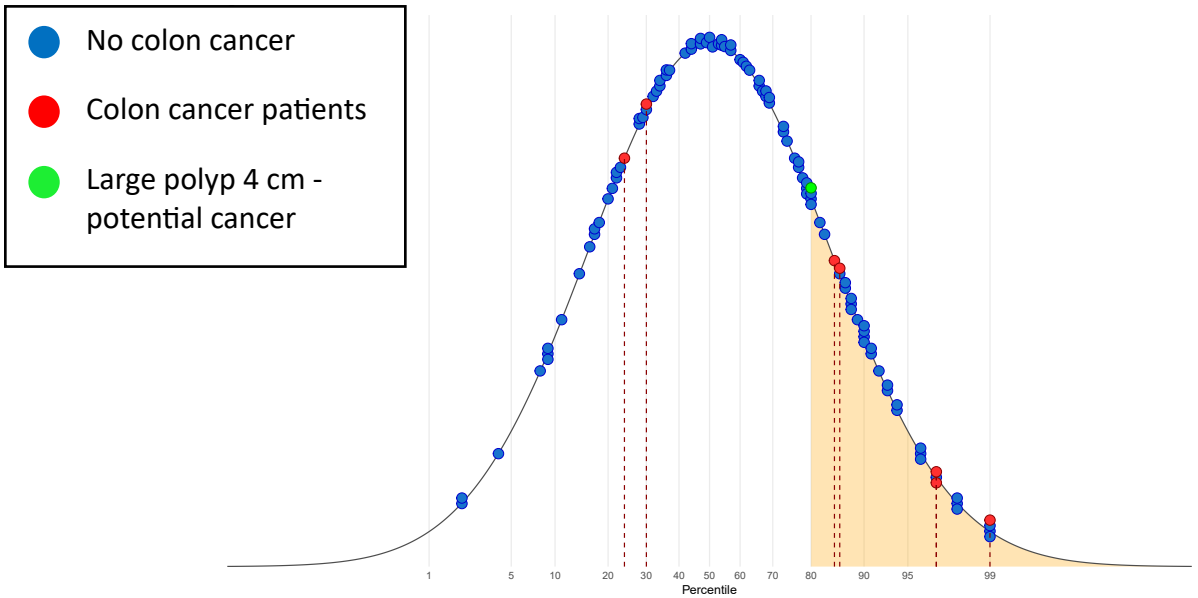


The general population breast cancer risk curve is shown in Blue, and the patient with a breast cancer PRS on the **89th centile is shown in Orange.**



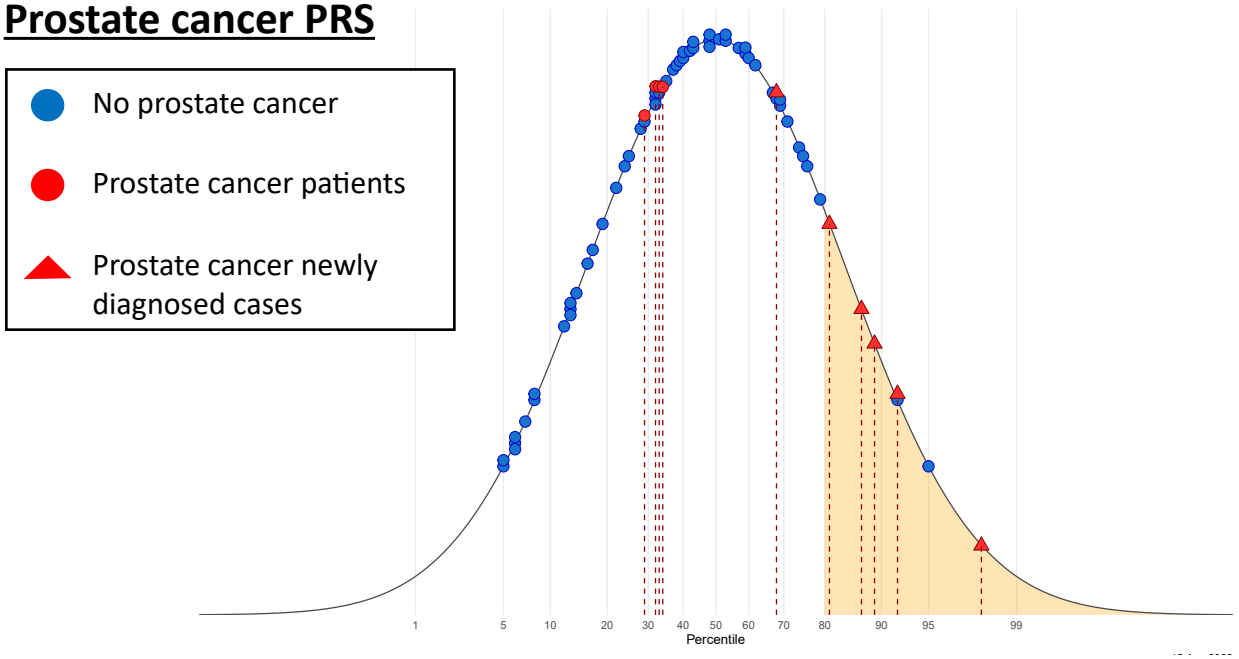
PRS Results – the First 104 Patients 90S Study

Colon Cancer PRS: Each dot represents an individual



This curve illustrates that 5 patients who have a previous diagnosis of colon cancer occur in the top 20th percentile of risk.

Prostate cancer PRS



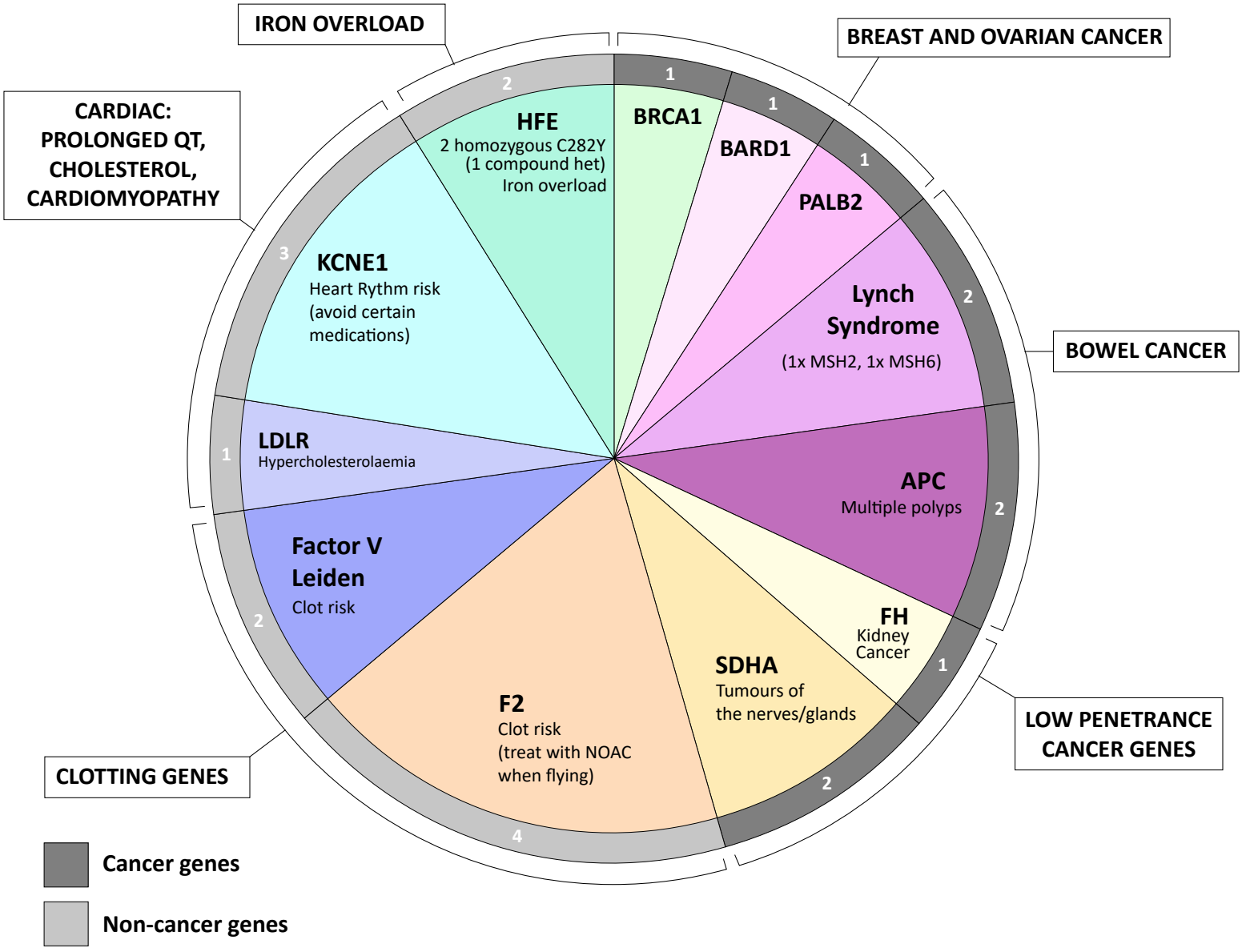
This curve illustrates that 5 patients who were newly diagnosed with prostate cancer occurred in the top 20th percentile of risk.

Of all cancers, colon cancer will take the most lives of individuals under 50 by 2030. These PRS curves show the potential for risk stratifying populations being offered screening. PRSs help identify those who are at the highest risk of specific diseases. The same paradigm can be applied to prostate cancer.

Gene Changes - the First 104 Patients 90S Study

This chart demonstrates the gene changes that we have found in the first 104 patients involved in the 90 Sloane Street Study.

First 104 WGS - Major Gene Changes Found

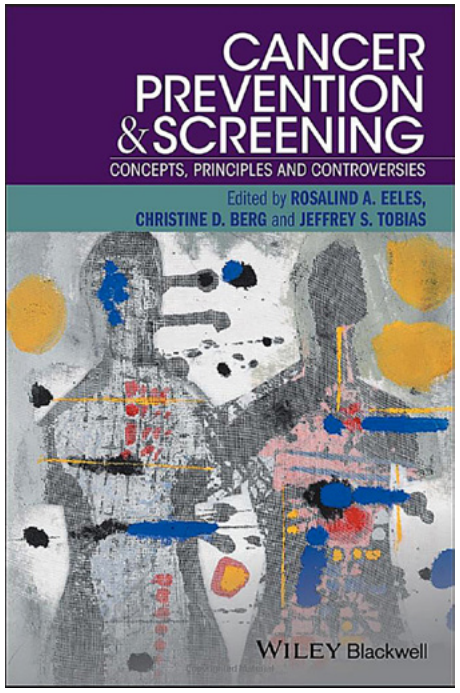


For risks associated with *BRCA1, BARD1, PALB2, MSH2, MSH6* and *APC* see Page 17-18 Gene-Associated Risk Chart. For a more comprehensive list of the genes in the Preventive Panel see our website: https://thelondongeneticscentre.com/.

Dr Michael Sandberg and Professor Ros Eeles.

Dr Michael Sandberg is the Medical Director of 90 Sloane Street, and he works closely with Professor Ros Eeles, who is a world-renowned Cancer geneticist, Oncologist and a radiotherapist. She has led an extensive number of worldwide genetic trial collaborations, repeatedly moving the boundaries in the world of prostate cancer and genetics. Professor Eeles' work has changed the management of prostate cancer internationally.

Her book, "Cancer prevention & Screening", is a global multi-authored definitive document in this area. The book is aimed at both medical professionals and the lay public and is the winner of the BMA Chairman's Prize for the 2019 book of the year.



The Times, June 2022

British scientists have developed a way to screen patients for "actionable"mutations - faulty genes that increase the risk of disease but can be mitigated through lifestyle or treatment

A study has revealed that one in four people carry potentially harmful genetic mutations that can be picked up through a simple blood sample.

Dr Michael Sandberg, a GP at 90 Sloane Street in London, which co-ordinated the research, said: “This study is pushing the boundaries of genomic screening by showing that it is feasible as part of GP care.

The Telegraph, June 2022

DNA testing revolution' at the GP could detect patients at risk of cancer

Using genomic sequencing in primary care means patients can get early treatment for health conditions before they become life-threatening, the first UK study of its kind has found.

Early modelling based on the study findings suggests thousands of lives a year could be saved by the tests, the researchers said, by reducing mortality by at least a fifth for breast and ovarian cancer.

The Health Secretary said on Thursday that the technology was “changing the future of healthcare”, and opened up the possibility for patients with life-changing illnesses to be diagnosed early via their GP.

Meet The London Genetics Centre Team

Professor Ros Eeles

Dr Gabriella Pichert

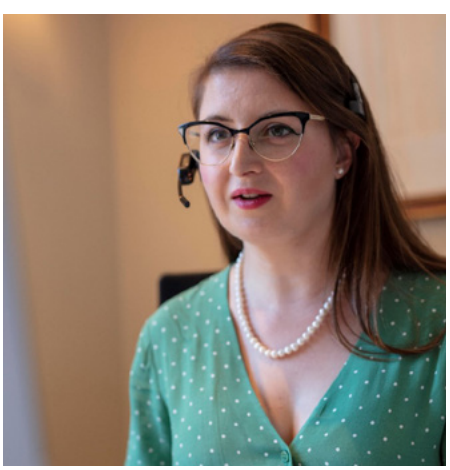
Dr Lucy Side

Dr Tessa Homfray

Dr Michael Sandberg

Lexi Noden

Sophie Hicks



Lucinda Seymour

In the UK there are only around 250 consultant geneticists. The geneticists on our team have over 185 years of experience between them. The team are also joined by Genomic Counsellor, Lexi Noden, Genomics Associate, Sophie Hicks and genetics secretary, Lucinda Seymour.

Prof Ros Eeles is a Professor of Oncogenetics. She is also a radiotherapist treating prostate and bladder cancer and leads many worldwide trials in prostate cancer genetics. She works at The London Genetics Centre in a private capacity and her work on the Whole Genome is not remunerated by the London Genetics Centre.

Dr Gabriela Pichert has over 20 years of experience in cancer genetics and was a consultant geneticist at Guy‘s and St Thomas’ Hospital for 8 years, some of them as joint lead in cancer genetics. She currently works at the London Genetics Centre, and several private hospitals in Switzerland. Dr Pichert is an author on over 75 papers and has edited a textbook on the diagnosis and management of rare hereditary cancers.

Dr Lucy Side is a Consultant Geneticist in Wessex and Honorary Associate Professor at UCL Institute for Women’s Health, having held Consultant posts in Oxford, where she trained in Clinical Genetics, and at Great Ormond Street. Her MD thesis was on Neurofibromatosis type 1. She chaired the UK Cancer Genetics Group from 2015-18 and is Clinical Genetics adviser for the NICE guideline on familial ovarian cancer.

Dr Tessa Homfray is a very experienced Consultant in Medical Genetics and has worked for many years in multiple centres of excellence. She has special interests in fetal and prenatal genetics and cardiac genetics, has multiple publications on both subjects and has written several book chapters. She works on the Harris Birthright Centre, Kings College Hospital, St George’s University Hospitals and The Royal Brompton Hospital and lectures both Nationally and Internationally.

Dr Michael Sandberg is a GP and Medical Director of 90 Sloane Street practice. He has trained in genetics, cardiology, and echocardiography. He has pioneered the use of ultrasound and echocardiography General Practice bringing in a full-time cardiac technician. He is a member of the American Society of Clinical Oncology (ASCO) and the European Society of Cardiology.

Alexandria Noden completed a Masters in Genetic and Genomic Counselling at the University of Glasgow during which she worked at the Western General Hospital in Edinburgh and the Queen Elizabeth University Hospital in Glasgow. Her current interests reside in Cancer Genetics; her dissertation investigated the psychosocial needs of individuals diagnosed with or at an increased genetic risk of ovarian cancer.

Dr Luis Izquierdo is the Medical Director at Veritas Intercontinental. He is a Doctor of Medicine and Surgery and has a Master of Science in Medical Genetics from the University of Glasgow. He has an extraordinary doctorate award from the UCM.

Dr Vincenzo Cirigliano is the Chief Technical Officer at Veritas Intercontinental. He has an extraordinary doctorate award from the Autonomous University of Barcelona and was previously head of Molecular Genetic in General Lab, Labco and SYNLAB. He is internationally recognized for being a pioneer in the development and introduction in clinical routine of innovative molecular tests in prenatal diagnosis.

Bibiana Palao is the Chief Product Officer at Veritas Intercontinental. She is a professional in Health Sciences and an expert in Medical genetics with more than 12 years of experience in the Diagnostic sector, having previous held the roles of Director of the Scientific Department of Innovation at SYNLAB International and Technical Director at Labec.

Matthew Dixon is the Chief Business Officer at Veritas Intercontinental. He plays an integral part in coordinating both teams and is the inspirational facilitator among us.

We are very grateful to the Consultant Cardiologists working at 90 Sloane Street:

Dr Vias Markides is the head of cardiology at The Royal Brompton Hospital. His subspeciality is arrythmias, ablations and pacemakers.

Prof Diana Gorog is a consultant cardiologist at The Royal Brompton and the Lister Hospital in Stevenage. As well as being an interventional cardiologist undertaking angioplasty, Diana also heads a research team in thrombosis and prevention of heart attacks.

Dr Denis Pellerin is a consultant cardiologist at St Bart's Hospital. He is a world authority on echocardiography having written many European guidelines. He established the largest echo department in the UK. He specialises in stress echo and transoesophageal echo.

The London Genetics Centre

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