*Article*

# Precision Medicine and Ancestry

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- Precision medicine is a valuable new tool to aid diagnosis and prescription
- A new generation of DNA sequencing technology makes it possible to obtain complete genomes for individual patients.
- Large-scale genome-wide surveys of associations between DNA markers and disease conditions are a key resource in this process.
- Individual patients will be able to get health advice based on their personal Polygenic Risk Score (PRS).
- All PRS statistics depend on knowledge about ancestry.
- In Aotearoa/New Zealand engagement with, or work led by, Māori and Pasifika voices is required.
- There are already existing examples to show how widely and successfully this programme has been adopted overseas.

## Introduction

New molecular technologies are making increasingly significant contributions to medicine. In particular, second and third generation DNA sequencing technologies are bringing genomic analyses within the reach of individual patients. Large scale population surveys reveal many previously unknown genetic variants which genome-wide association studies (GWAS) show to be significantly associated with genetic disorders. This information can be captured via a class of statistic known as the Polygenic Risk Score (PRS) which can identify those individuals at greatest health risk, even for those otherwise cryptic conditions, sometimes known as '*silent killer*'. If these new advances are to be distributed in a socially equitable fashion, then it is first necessary to recognise that characteristic genetic differences exist between ethnic groups and to adjust PRSs to allow for this. In practice, this turns out to be quite difficult to achieve for many reasons discussed here.

This account begins by tracing the developmental history of precision medicine starting from publication of the first human genome. It goes on to explain how this technology currently operates looking at several successful examples involving well-known conditions. This leads on to discussion of the difficulties created by differing patterns of genetic variation between ethnic groups and approaches to resolving them. It concludes with a prospective consideration of how all of the preceding information and considerations might apply to contemporary health issues across Island Southeast Asia and Oceania. A valuable comparative account of how this all works in a China context can be found in Wang et al. (2025).

## The origin of precision medicine

> *Maybe we are all born with the seeds of our destruction fully formed inside us*

If the above premise is true, then what should we best do about it? This review will suggest that good news is already to hand in the form of precision medicine. To evaluate this proposition, it is first necessary to ask just what is 'precision medicine' and how did it come into being? In short, the objectives of precision medicine are to deliver reliable, personalised diagnoses and prescriptions in a socially equitable and ethnically unbiased manner, based on large scale genetic information. It is made possible by advances in DNA technology, improved knowledge about biological networks and cell biology, including the effects of aging and the environment. It is delivered by efficient handling of 'big data' and sophisticated bioinformatics routines. It works. For example, a recent group of volunteers were given a saliva test and screened for prostate cancer markers. Several of them were identified as having a high risk of prostate cancer. Many were negative by the conventional prostate serum antigen (PSA) test. Some of those found to have the disease went on to be cured (Box A).

The potential of molecular mapping to identify diagnostic markers for inherited conditions was recognised long ago. Researchers looked for DNA markers in close linkage to a particular disease. Such markers were often single nucleotide polymorphisms (SNPs). The diseases themselves were generally well-known, single-locus, conditions like cystic fibrosis, Huntington's disease or Duchenne muscular dystrophy. The record of this heroic episode can easily be found in any standard human genetics textbook. Even though these SNPs etc. often gave valuable predictive information, e.g., for prenatal screening, they did not always identify the defective gene or shed light on the biochemical nature of the disorder. Discovering exactly where and what these were required many years of painstaking genetic

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**Box A:**

*UK BARCODE1 Prostate cancer Study (2025)*

This is a leading example of the successful application of contemporary precision medicine by McHugh et al. (2025). It gained considerable exposure in the scientific press and on TV (Devlin, 2025; Gallagher, 2025). The investigators used a Polygenic Risk Score approach (Hunter, 2025) to screen 6393 males 55-69 (i.e. members of the high-risk age group) for 130 known variants associated with prostate cancer. A select group (468) of those in the 90th percentile risk category underwent MRI scans followed by biopsy. Among these 187 had cancer and 103 at high-risk required treatment. Further examination showed that 74 of them would not have been detected by the usual diagnostic PSA test.


Deeresh Turnbull (right) with his brother.

Deeresh Turnbull participated in the study and discovered he was in the high-risk category and had cancer. His brother joined later and discovered that he had an aggressive tumour.
From Gallagher (2025) with permission.

mapping work to edge ever closer to the defective locus.

The human genome project (1990 - 2003) was seen as a solution to the mapping dilemma. When the near full sequence was announced, it was anticipated that once a marker linked to a genetic disease was found, then all one had to do was search along the local chromosome DNA sequence section and look for functional units which carried mutations in affected individuals. Simple enough maybe, but still a lot of work, and not guaranteed to come up with the correct answer. As new sequencing methods (Box B) were spun off from the human genome project (Tafazoli et al., 2025) it became possible to compare whole genome sequences between affected and control individuals.

This type of approach was called a genome-wide association study (GWAS) and could be performed in cases where a prospective marker had been identified or where the cause might be complex (i.e. one involving several loci) see Jones et al. (2024). At the outset these seemed to be highly successful yielding what become known as 'Manhattan Plots' due to their resemblance to the New York skyline – see Figure 1 below for an example.

Significant markers can be identified with very high levels of statistical significance. All well and good, except that in many cases GWAS identified large numbers of markers across the genome with very small p-values, indicating statistically significant associations with the trait . Worse still, most of them might individually only account for 2 or 3% of the effect. In total, the entire collection rarely came close to explaining all the variance in the data., e.g., see Zhu et al. (2021) in relation to osteoporosis.

In short, it all resembled an embarrassing abundance of riches. But, what looked like being an expensive disaster was partly addressed through the development of the polygenic risk score (PRS), which combines information from multiple genetic variants into a single measure of genetic susceptibility. The strategy proved to be very effective as the prostate cancer example shown in Box A demonstrates and see Shah et al. (2024); Spears et al. (2024); Zhang and Zhang (2024). It has also proved its worth with relatively complex or multicausal diseases and

including for those with marked environmental influences – see Box C.

## Further Considerations

There can be no doubt whatsoever that PRSs are valuable predictive tools, but they do not stand alone as a one-shot solution.

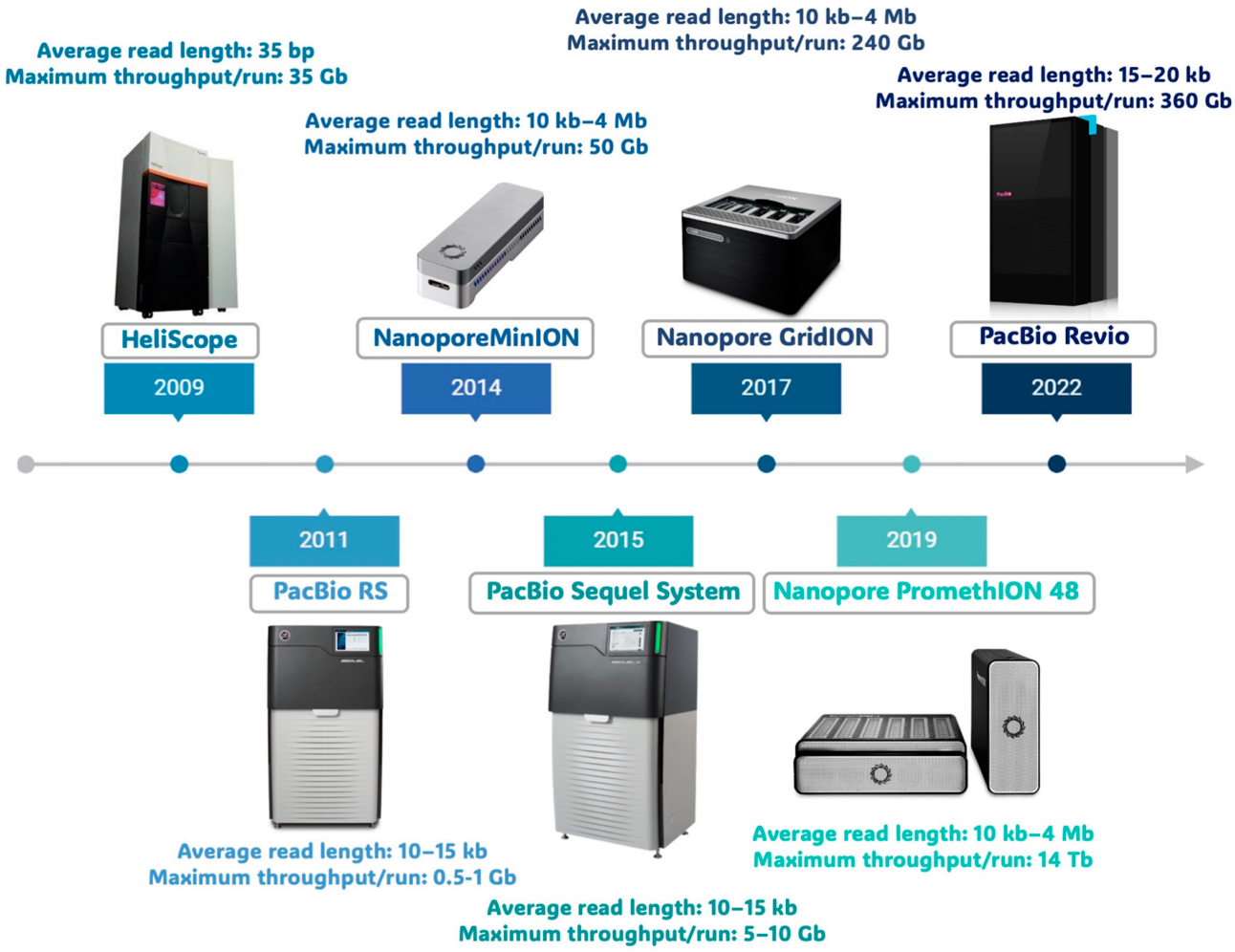
First, there is the eternally fraught issue of global genetic variation in human populations. As will be argued here and has been practised elsewhere (see later), these patterns are real and should be recognised as non-threatening facts.

**Box B:**

*The evolution of DNA sequencing technology*

The human genome project used methods developed by Fred Sanger and colleagues (1977). Their system created short (250 nucleotide) radioactive DNA sequences that could be read via a manual electrophoresis run. The original chemistry was later replaced by a semi-automated procedure using fluorescent dyes. Participating laboratories, like the Sanger Institute in Cambridge UK, resembled a giant warehouse filled with hundreds of Applied Biosystems DNA sequencers.

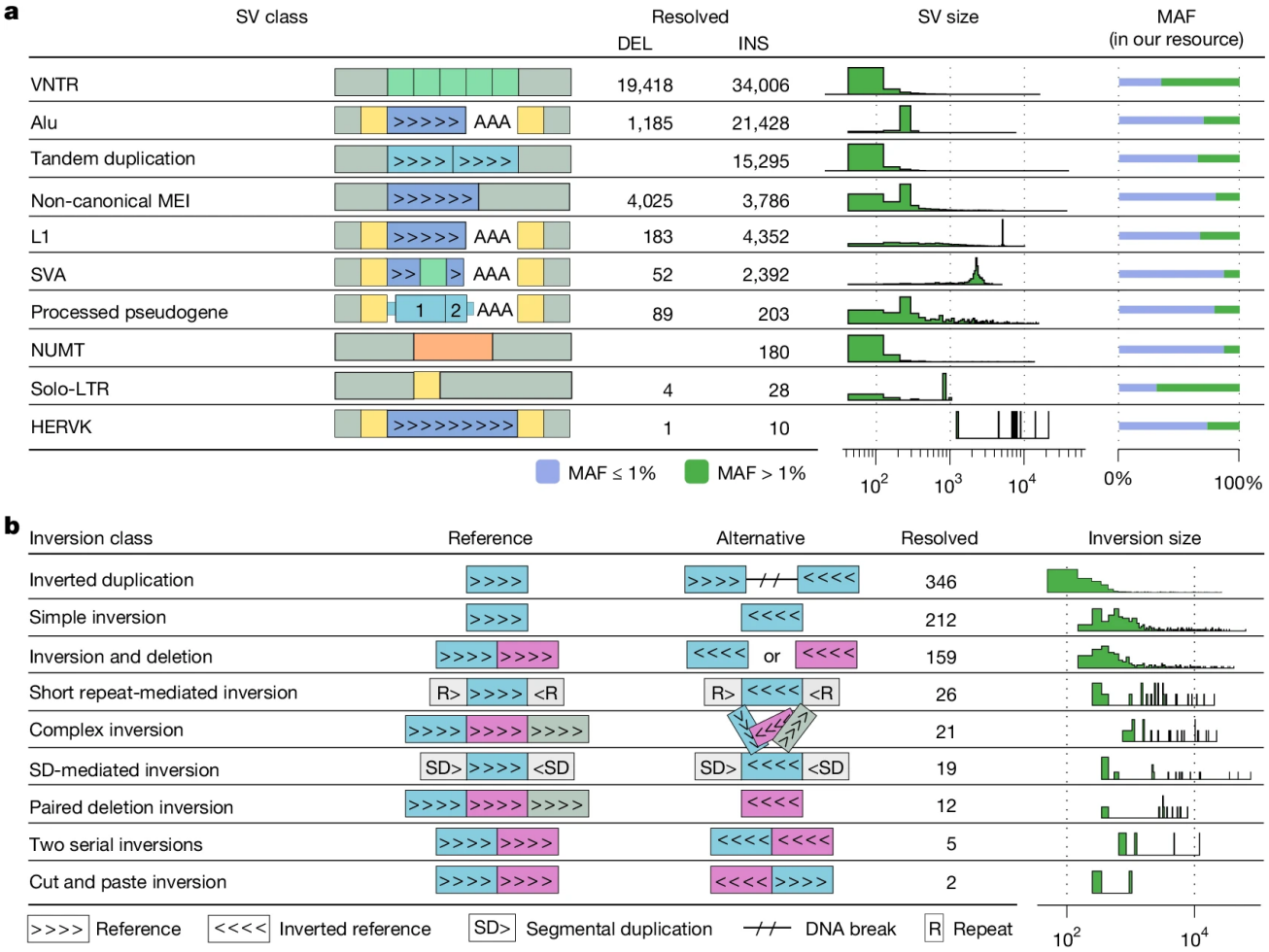
A step change came with the advent of next generation technology (see Wang et al. 2025 for a timeline of events). New machines provided very large numbers of short reads from DNA templates tethered in glass cells (Illumina) or fewer, but longer, reads from plates (Roche). Since then, a so-called Third Generation of technology has drastically increased outputs and reduced costs (see Scarano et al. 2024). The new PacBio machines still depend on Sanger chemistry but achieve single reads of 15 to 30 Kb in length with total outputs of up to 360 Gb per run. In contrast, the Oxford Nanopore system is based on physical chemistry. Here, a single DNA molecule is guided through a molecular pore. As each nucleotide passes it changes the electric potential across the pore and can be read directly as a digital signal. DNA sequence reads between 10 Kb to 4 Mb are obtained with up to a 14 Tb total output.




A brief pictorial history of next generation DNA sequencing technology
From Scarano et al. (2024) republished under CC BY 4.0.

*Types of genomic DNA sequence variation*

Comparative genomics consistently shows that population level DNA sequence variation is highest in African subjects. The full range of structural variation types (Schloissnig et al., 2025) is best revealed by long read methods (Carss et al., 2025). Polymorphisms range from SNPs in, and around genes, through simple tandem repeat (STR) expansion and contraction all the way to gene copy number variation or large insertions and deletions of varying complexity.




Types of DNA sequence variation from Schloissnig et al. (2025) republished under CC BY 4.0.

For some time, it has been a fashion among social science practitioners to deny the importance of such variation (see Chambers 2017 for one account and references therein). This view stems, in part, from Richard Lewontin's (1972) calculations showing that the majority of genetic variation in humans is distributed among individuals, rather than between ethnic groups. His classification of population groups is highly suspect but his basic conclusion has stood the test of time. Sadly, the core observation is beside the point. In what Anthony Edwards (2003) unkindly calls 'Lewontin's Fallacy' he points out that the smaller percentage of variants that do define populations still amount to thousands of diagnostic Ancestry Informative Markers (AIMs). Reluctance to recognise such differences may stem from concerns that they might be used negatively as descriptors of groups of people. Alternatively, that they may have some intrinsic value and become dissociated from their intended meaning and exploited . Hence, the drive for what has become known as 'data sovereignty' i.e., exclusive control of a group's own genetic information.

There is no doubt that in order to work as intended, PRS schedules must be refined or weighted to include information about patterns of genetic variation. Some steps have already been taken in this direction (Box D).

This does not represent what some have called 'race-based medicine', but rather ethnically/ancestrally informed precision medicine. However, adjusting PRSs in this way and using them in practice is far from straightforward. For example, GPs will need to develop a safe form of dialogue to determine ethnic groups of patients. Next, there is the question of the reliability of metadata records in DNA variant databases. Are they genuinely ancestral as should be the case for medical genetic information, or are they derived as 'self-declared ethnic affiliation'? These are quite different, and the latter definition is often used as a 'safe' gold standard in New Zealand. Finally, there is the political drive for social equity to consider. This requires that equality of medical treatment should be delivered based

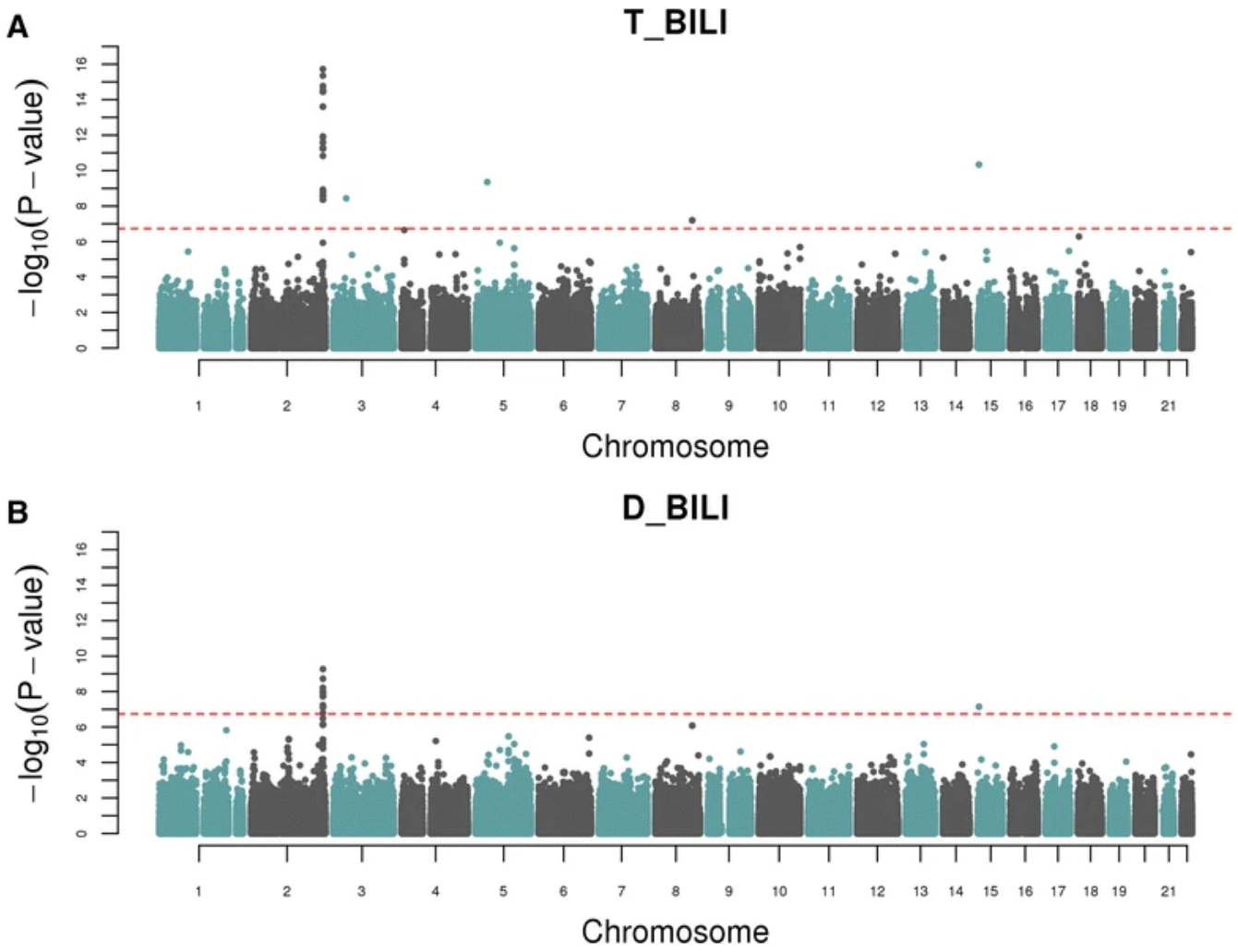



Figure 1: GWAS Manhattan plots for; A Total Serum Bilirubin, and B Direct Serum Bilirubin. The adjusted correction probability threshold of $1.84 \times 10^{-7}$ is indicated by the horizontal dashed line.
From Benton et al. (2015) republished under CC BY 4.0.

on the gold standard definition. Deciding the wisdom or otherwise of such measures would require comparative studies where PRS schedules weighted by ancestry alone versus weighted by declaration. If little, or no, difference in effectiveness is found, then using the existing system might solve the potential problem.

It would seem prudent to close this section with a prospective cautionary note about things to come. In other words, markers may have surprisingly different effects due to epigenetic modification and/or 'parent of origin' (POE). Evidence for such phenomena is already available. Hofmeister et al. (2025) examined 109,385 UK Biobank individuals in a GWAS search across 59 complex traits including 14,000 protein encoding loci. They discovered 30 POE contrasts including at least one third of them with opposing magnitude (aka 'bipolar'). For example, a Type 2 diabetes marker at chromosomal map position 11p15.5 showed an increased risk (1.14×) with a paternal allele, but a protective effect (0.91×) with a maternal allele. Many of these markers were related to growth and metabolism (see their Table 2) and located within heavily imprinted chromosomal regions. They validated 87% of these disease associations across 85,000 individuals from the Estonian Biobank and 42,346 offspring participants in the Norwegian Mother, Father, and Child Cohort Study.

Second, to be fully effective in delivering tailored medication PRSs must be informed by advances in cell biology (Pergola et al., 2023). Many genetic disorders can arise from multiple root or derivative biochemical causes. Knowing how these pathways work together can allow medical practitioners to direct a primary drug of choice at the most likely biological target system.

Third, there is the joint challenge of picking the best approach and medicine to effect a cure. The selection of pharmaceutical agents themselves is also a two-fold problem. Clinicians must use genetic evidence to pick the best prescription for the condition. They must also select one that the patient is likely to respond well to and without side-effects as far as possible.

Finally, there is the dilemma of the so called 'missing variance' in GWAS analyses: note comments in Zhu et al. (2021). To some extent this can be explained by the effect

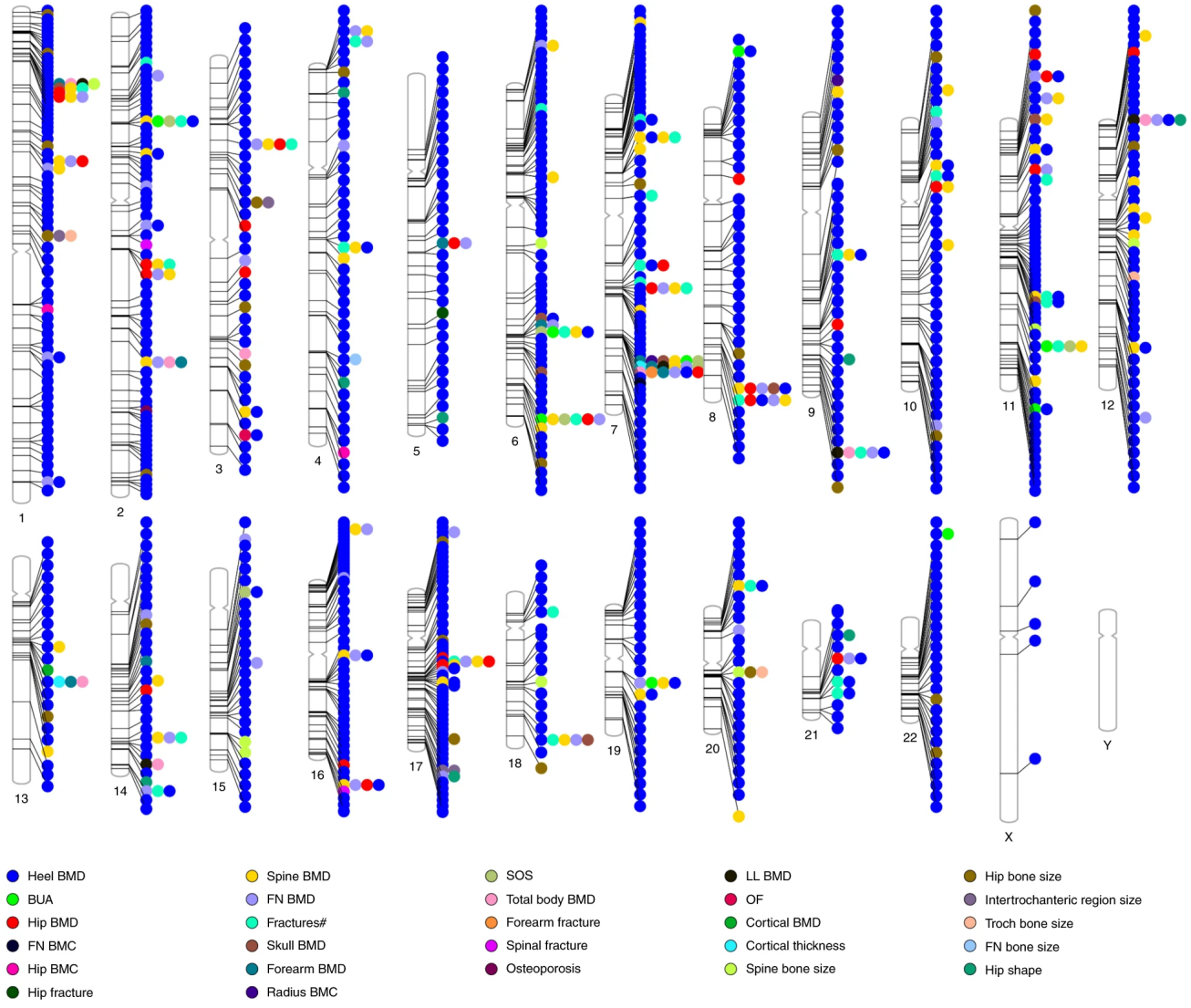
**Box C:**

*Case study examples of GWAS and PRS in action*

**Diabetes**: This disorder is primarily classified as Type 1 or Type 2 based on European phenotypes. It also shows more diverse expression in other places and is associated with numerous gene markers (Deutsch and Udler, 2025). Smith et al. (2024) list 650 Type 2 associated variants gleaned from 37 studies over four ethnic groups and one admixed population and with a broad array of biological profiles. The PRS method has been evaluated by Prasad et al. (2025) for clinical use.

**Breast Cancer**: Preliminary GWAS studies revealed more than 200 risk loci. Jia et al. (2025) compared 172,737 patients versus 242,009 controls across three ethnic groups increasing this number by 131 new markers. Among these 50 could be located to a single unique DNA sequence variation. Integrating these data with functional studies identified P13K/AKT, TNF/NF-$\kappa$B and Wnt/$\beta$catenin biochemical pathways.

**Osteoporosis**: This disease affects around 200 million people worldwide. Zhu et al. (2021) reviewed twelve years of GWAS work and the vast number of loci and biochemical systems implicated are shown in their Figure 2 below (republished under CC BY 4.0).




**Pharmacogenetics**: Kasela et al. (2025) examined choice and dose rate for antipsychotics, statins and several other classes of drugs using large patient cohorts compared with Estonian Biobank reference genomes. They recovered signals in relation to specific loci, such as CYP2D6 for the blood pressure medication metoprolol and CYP2C6 for the anticoagulant warfarin. The well-known case of resistance to clopidogrel found in some cardiovascular patients was investigated in the admixed population of Puerto Rico by Monero-Paredes et al. (2023). They found 50 SNPs related to poor response in Europeans manifested in Caribbean Hispanics due to ancestral gene fractions.

**Schizophrenia**: This is well known to be a complex non-unitary condition. It has very high heritability (60-80%) but its genetic basis remains unknown. All of these factors serve to confound both diagnosis and prescription.

Pergola et al. (2023) identified 287 risk loci and developed a 'parsing' routine to assign them to cell biological pathways. Bigdeli et al. (2024) tested 13,012 affected and 54,266 reference participants from the Million Veterans and the All of Us Programmes (Gouveia et al., 2025) and discovered new PLNXA4, PMA1P1 and TRPA1 markers in Africans. Overall, 86,981 patients and 303,771 controls were eventually tested finding 708 variants across 376 loci active in various cell types in brain. These figures support a metanalysis linking candidate markers to aspects of synaptic biology notably glutamate subunit receptor GRIN2A and TfSP4 (Trubetskoy et al., 2022). With so many widely dispersed loci implicated concerns were raised that other otherwise neutral or anonymous loci might be caught up implicating their causal involvement. Any such findings might raise serious social concerns about other screening technologies. Fortunately, this does not seem to be the case for conventional panel(s) of forensic casework systems (Yang et al., 2024).

of our environment via epigenetics. Lifestyle and aging alter patterns of genetic control mechanisms by inducing specific chemical modifications to genomic sequences giving rise to epigenetic modification of gene expression. Hence one's innate PRS may predispose one to a particular condition, but it is only realised via behavioural and/or environmental experience. It is probably impossible to try to get round this problem by running surveys and simultaneously controlling for all contributions due to sex, aging and lifestyle factors.

## The situation in New Zealand

At the outset it must be recognised that the New Zealand population contains many different ethnic groups. Principally, these consist of indigenous Polynesians (Māori) and settler colonists (European) and their descendants. But there are many others too, including Mainland Asians (e.g., Chinese), Austronesians, other Polynesians and Melanesians (aka Australopapuans) from Island Southeast Asia and Oceania (e.g., Filipino, Cook Islander/Tongan and indigenous Fijian), Continental Asians (e.g., Indian). As will be explained below, all these groups have (often unrecognised or unappreciated) deeptime ancestry and varying degrees of admixture with other (ancient) hominids (e.g., Neanderthals and Denisovans). Since these are facts of life and accidents of history, each group will likely require their own PRS schedule calculated from their own large and reliable genomic DNA sequence database. This is no simple matter. Indian citizens are often lumped along with Chinese into a compound group called simply 'Asians' e.g., as Lewontin (1972) did, and others after him have also done and continue to do. What is certain, is that a detailed knowledge of the historical relationships between all ancestral lineages will be important if delivery of precision medicine is to fully effective. Consider, for example, the significant residual effects of ancient admixture. Specifically, these are with Neanderthals in Europe and with Denisovans in Melanesia (as recently as 28,000 years ago) and later passed on to Polynesians. Neanderthal heritage is manifested in multiple immune system markers in part responsible for the burden of autoimmune diseases in Europeans. Whereas Denisovan ancestry is shown in metabolic markers leading to the well-known spectrum of gout, obesity, Type 2 diabetes and coronary heart disease common in those with any degree of Austronesian descent.

In a short article like this one, it is impossible to give a full account of the adventures and experiences of anatomically modern humans as they spread across the entire face of the globe (but see Nägele et al. (2022) for the full story). So, what follows is only a brief explanation of the historical relationships between the different groups of today's New Zealand inhabitants (more detailed accounts can be found in Chambers and Edinur 2020; Matisoo-Smith and Gosling 2025. All humans have a common origin in sub-Saharan Africa and migrated outwards by various routes. The first branch lead north into Europe and ends there for the purposes of this story. Two other lineages spread out across northern and southern Asia. The northern branch filled up mainland Asia and one subgroup made it to Taiwan by around 10,000 years ago. The southern branch reached island southeast Asia and split with one population becoming the ancestors of Australian Aborigines and New Guinea Melanesians. A second group moved northwards and still exists as relict Negrito and Orang Asli groups found throughout island southeast Asia. Later, Taiwanese developed reliable methods of oceanic voyaging and moved through ISEA, south along the northern coast of Papua New Guinea and its outlying islands and reaching to Vanuatu, Fiji and Samoa as the Lapita civilization. The residual Austronesian people in the Bismarck Archipelago became admixed with the local Melanesian population to become Polynesians. There are many accounts of their later voyages across the Pacific to a central homeland in the Marquesas spreading to fill the 'Polynesian Triangle' with corners marked by Hawaii in the north, Rapa Nui (Easter Island) in the east and Aotearoa in the south (see Figure 2)

Today these stories are reflected in the different appearances and cultural practices of their various groups of descendants. Their underlying genetic differences (Littlefield et al., 2024) are reflected in their contrasting health needs which our new precision medicine might hope to better serve (Chambers et al., 2026).

The reason why the peoples of West Eurasia may be a poor medical genetic model for those in the Asia-Pacific region (and the much of the rest of the world) has emerged from an extensive aDNA study by Akbari et al. (2026). They show that many common genomic changes now found in West Eurasians have been driven by natural selection after the adoption of and agricultural lifestyle. A large fraction of these new mutations has pharmacogenetic significance and/or are important in shaping their wider disease schedule.

Meeting all these new requirements in a culturally safe

**Box D:**

*Ancestry weighting for PRS statistics*

Finding the correct identity descriptor(s) for human groups is a difficult problem. Even seemingly simple cases can turn out to be complex. For example, Europeans are derived from ancient tripartite admixture: see Nägele et al. (2022) for a wider discussion. Sun et al. (2024) also show just how significant ancestry effects can be. Gouveia et al. (2025) used a similar approach to classify 230,016 volunteers from the All of Us Research Programme in the US., linking markers to body musculature and height. Further, Gold et al. (2025) showed that racial disparity can exist even in relation to access to genetic testing.

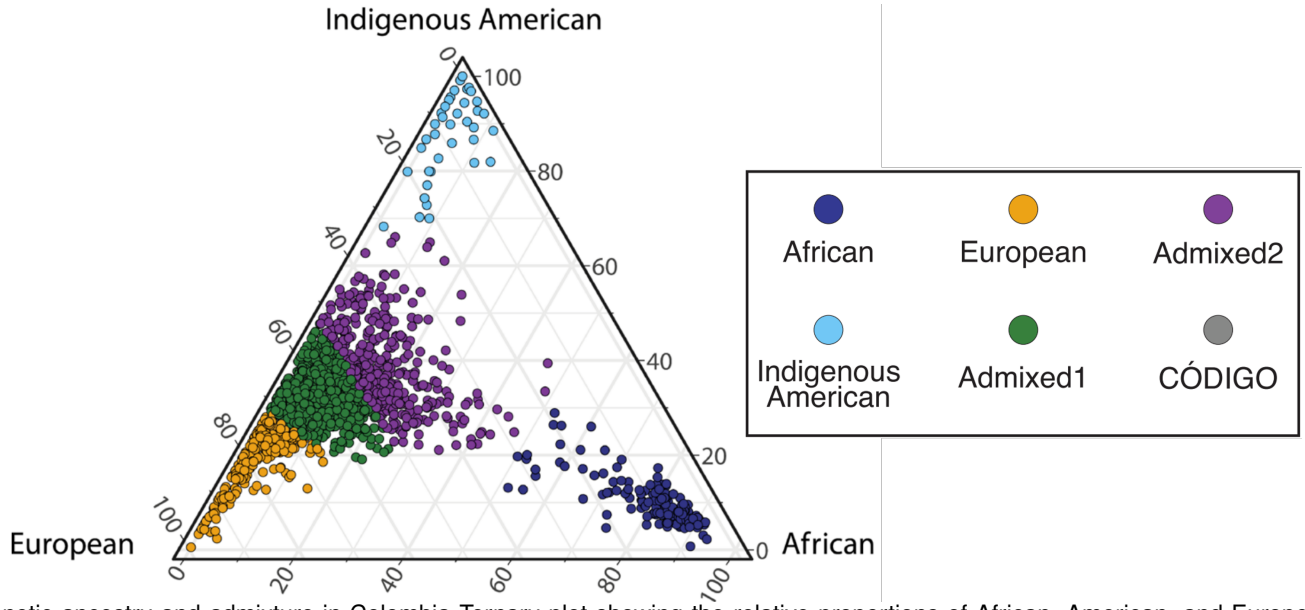
The wider issue of human diversity and health has been reviewed by Palma-Martínez et al. (2025) and echoes our own recent publications (Assyuhada et al., 2023; Edinur et al., 2022). Equally, Jones et al. (2024) note that many studies, including but not limited to those listed in the paragraph above, now aim to increase representation of diverse ancestry in large scale genetic surveys.

The US All of Us programmes recently reported whole-genome data on 199,000 volunteers (Rossen et al., 2026) representing three ethnic groups. They show how PRS values vary significantly between these groups reflecting similar experiences from the other national level studies listed in Box D2 below and see Pain (2025).

*Necessary considerations before weighting scores*

> *The question of how these data should be combined and the consequences that genetic variation across ancestry groups might have on GWAS results warrants further investigation.*
>
> Quotation from Jones et al. (2024).

This debate centres around group identity. There are fundamental questions about if this is best done based on ancestry alone and how that might be determined versus using the social science approach of self-declared ethnic identity? Some fear that using genetic data based on ancestry may lead to racism and a return to an era of eugenics (Wojcik, 2025). In this context Popejoy et al. (2020) have explained how poorly constructed name tags like Asian, Oceanic or Hispanic versus Latino really are, and how poorly ethnic identity defined in this way maps on to ancestry. The figure below shows how ancestry informative markers (AIMs) reveal genetic relationships between elements of the complex mixture of ancestral population groups in Colombia. Their groups Admixed 1 and 2 correspond approximately with Latino and Hispanic respectively.




Genetic ancestry and admixture in Colombia Ternary plot showing the relative proportions of African, American, and European ancestry for each CÓDIGO sample color-coded by their categorial ancestry group. From Mariño-Ramírez et al. (2025) republished under CC BY 4.0.

Readers are invited to consult the following references for further accounts of precision medicine programmes on a national scale; Australia (Lewis et al., 2024), Brazil (Araújo de França et al., 2025), Ecuador (González-Andrade, 2025), Estonia (Milani et al., 2025), India (Bhattacharyya et al., 2025), Mexico (Barberena-Jonas et al., 2026) and its general importance for the southern hemisphere in general (Jiwani et al., 2025).


Figure 2: Distribution of Austronesian people across the World
From Chambers et al. (2026)

and equitable manner will surely be challenging. However, Rye et al. (2025) show how the pathfinding Rakeiora Genomic Platform commissioned by MBIE in 2014 might contribute and see Hitchman et al. (2026) for recent results and insight into the important role that Genomics Aotearoa will likely play in the future of precision medicine in Aotearoa/New Zealand. They will build on existing strengths derived from the earlier New Zealand studies they describe e.g., in relation to drug metabolism and CYP2C19. One early report by Robertson et al. (2026) has already been published recommending dose modification for three drugs taken by approximately 50% of patients in their test panel. Equally, the draft Pacific Pangenome Reference of Littlefield et al. (2024) will surely help will aid this initiative and the Consortium for Genomic Diversity, Ancestry, and Health in Colombia (CODIGO) initiative shows just what can be achieved with some external support. For instance, this team reports illustrative markers for six conditions ranging from Tacrolimus metabolism to malaria resistance and their effects across the population subgroups described earlier (Box D). Aotearoa/New Zealand will have to face the same problems as shown by the results of our various immunological genetics surveys conducted with NZ Blood bank volunteers from the late 1980s. At that time self-declaration returned approximately equal numbers of Full Ancestry Māori and Māori of Admixed Heritage (as initially determined by interview and confirmed by experimental results). Among the latter group the genetic data revealed around 50:50 Polynesian:European admixture (Edinur et al., 2013). Matching data have also emerged from our many surveys across Malaysia (Chambers et al., 2026).

The Universiti Sains Malaysia genetics programme is run by direct descendants of the Austronesian Diaspora. Equally, it is important that future initiatives in Aotearoa/New Zealand and the Pacific will require engagement of Māori and Pasifika leaders and analysts to ensure that developments in precision medicine serve their particular needs. Any such initiative might well leverage partnerships with other research collaborators and networks formed across the huge populations (350 million +) of Austronesian and Australopapuan descent.

As a whole Aotearoa/New Zealand has a strong foundation in small scale medical genetic studies. However, an integrated and well-resourced effort will be required to take full benefit from new developments in precision medicine. The (CÓDIGO) study shows what is possible and the Welsh Government's 'Genomics for Precision Medicine Strategy (2017)' shows that other nations are already well ahead with implementation planning. This includes the Wales Gene park facility hosted by Cardiff University - https://walesgenepark.cardiff.ac.uk/

## Overview and Conclusion

Starting from a simple strategy combining GWAS data with PRS scores this review has traced the origin of precision medicine and examined its prospects in relation to the health of Pacific peoples. Even at this early stage, it is clear that the system can be highly effective but that a more sophisticated approach is even now emerging. GWAS will generate the raw PRS statistics, but these will need to be weighted for POE effects. These modified PRS values

must next be considered in light of ethnicity information, including knowledge about the nature of the metadata in patient and reference databases. Next, leading candidate markers must be parsed to target via some form of Biological Pathway Factor (BPF) leading to selection of an Optimum Drug Choice (ODC) conditioned by a Drug Sensitivity Measure (DSM) calculated from the patient's own genomic data. As time progresses, it might be hoped that some form of Medical Impact of Lifestyle (MIL) could be developed by patients and their GPs perhaps involving an epigenome scan and allowing for factors such as sex and age.

Finally, some thought must be given to the question of how best to ensure the digital security of volunteer's genomic records and further what steps might be taken if a breach were to occur. The recent Manage My Health Episode (Satherley, 2026) is an object lesson in this regard. Indeed, future investigators may even consider it prudent to include such information in their 'informed consent' documentation for the benefit of study participants. That such concerns reflect real threats is illustrated by the recent reports of that appearance of UK Biobank records for sale on the Alibaba online market site (see Kelly and McMahon (2026)). Even more alarming is their allegation that those responsible are scientists rather than professional criminal computer hackers.

So, the world seems to be at the dawn of a golden age of improved understanding about serious health problems and well-equipped with powerful tools to combat them. Even hitherto cryptic conditions, like chronic fatigue syndrome, are beginning to yield to the GWAS/PRS approach (Genetics Delivery Team et al., 2025). Newly empowered by GWAS analyses potentiated by PRS statistics, the new cell biology learnings and marvels of the digital age like high-speed computing, big data handling, cloud storage and AI have the capacity to provide great improvements. All will be well provided that our society is able to recognise and treat genetic variation between individuals and groups as non-threatening badges of ancestral origin. The ideal outcome will be a sort of next generation NZ National Science Challenge. This will aim to produce a fully inclusive, well-supported, integrated national initiative in precision medicine.

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