23andMe R&D Day

January 18, 2022

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Agenda

Introduction -- **Anne Wojcicki, CEO and Co-Founder**

23andMe Therapeutics

- Therapeutics program overview – **Kenneth Hillan, Head of Therapeutics**
- Target discovery vision – **Joe Arron, Chief Scientific Officer, Therapeutics**
- Genetics-based target discovery – **Adam Auton, VP of Human Genetics**
- CD200R1 immuno-oncology program – **Jennifer Low, Head of Therapeutics Development and Adrian Jubb, Sr. Clin. Dev. Fellow, Therapeutics**
- CD96 immuno-oncology program – **Jennifer Low, Head of Therapeutics Development**
- Using genetics to inform clinical development – **Jennifer Low, Head of Therapeutics Development**
- Therapeutics program concluding remarks – **Kenneth Hillan, Head of Therapeutics**

23andMe Consumer

- Genetics-based primary care – **Paul Johnson, VP, General Manager, Consumer**
- The power of polygenic risk scores (PRS) for personalized health – **Geoff Benton, Director, Product R&D**
- Delivering a genetic-based primary care service – **Davis Liu, Chief Clinical Officer**

Concluding Remarks – **Anne Wojcicki, CEO and Co-Founder**

Q&A

Introduction

Anne Wojcicki
CEO and Co-Founder

Today’s News on 23andMe and GSK Collaboration

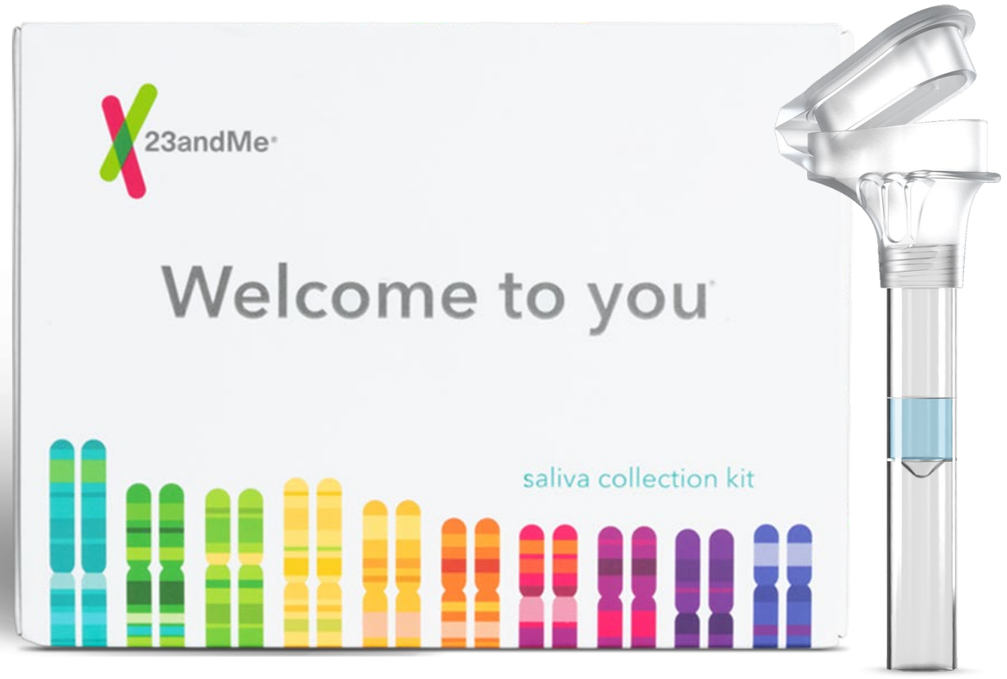
- **GSK has elected to extend the exclusive target discovery period of the collaboration for a fifth year**
  - We will continue to discover and validate novel drug targets using 23andMe’s proprietary genetic and health survey database
  - 23andMe will receive a one-time payment of $50 million

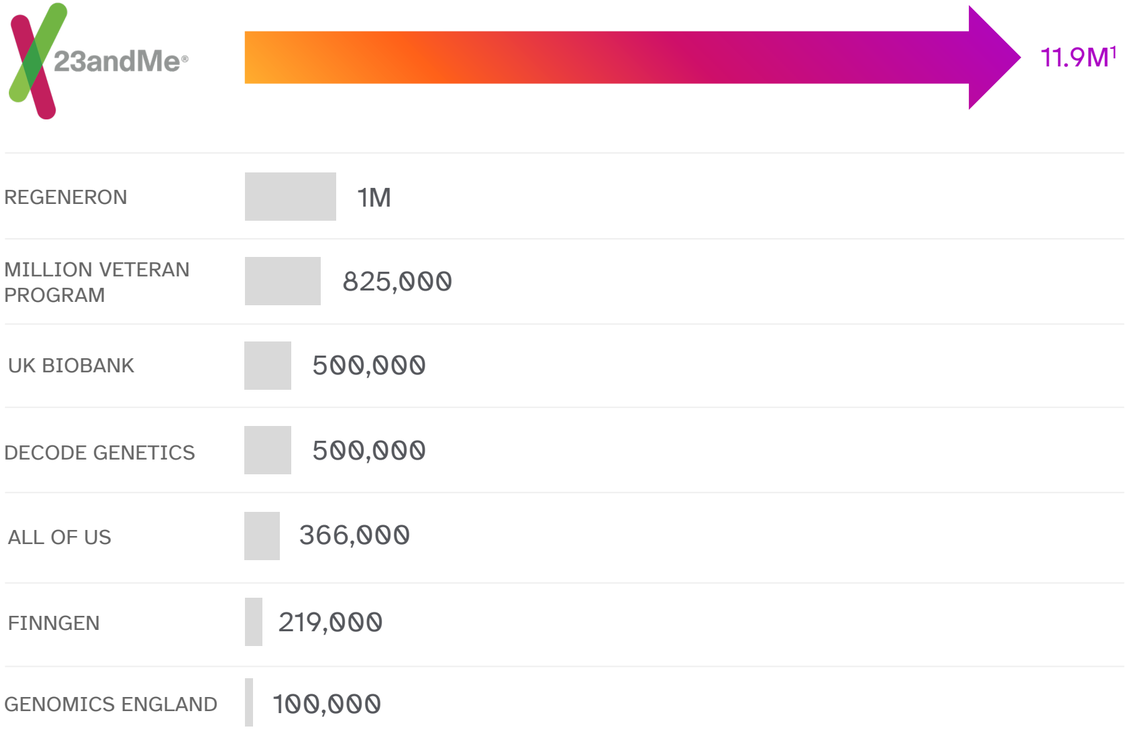
- **23andMe elects for royalty option on collaboration program targeting CD96**
  - 23andMe will be eligible to earn tiered worldwide royalties up to the low double digits
  - The worldwide royalty option curtails 23andMe’s future investment in this program and provides 23andMe with a potentially high value revenue stream if the program is successful

FY2022 Highlights

- Advanced a **wholly owned immuno-oncology program into Phase 1** study
- Acquired **Lemonaid Health** to expand into Primary Care and Pharmacy
- Received **FDA clearance** for HOXB13 **hereditary prostate cancer**
- Released **14** new genetic **health predisposition reports**
- Reported on **key genetic research findings** on COVID-19, reproductive lifespan in women, depression, Parkinson's disease, and more
- Added a new ancestry analysis, including additional insights into some customers' **indigenous genetic ancestry** from **North America** and **ancestral connections to 25 African ethnolinguistic groups**

Our Mission is to Help People **Access**, **Understand**, and **Benefit** from the **Human Genome**





Size and scale of 23andMe enables rapid, novel discoveries

1 As of September 30, 2021.

Consumer Powered Healthcare Flywheel

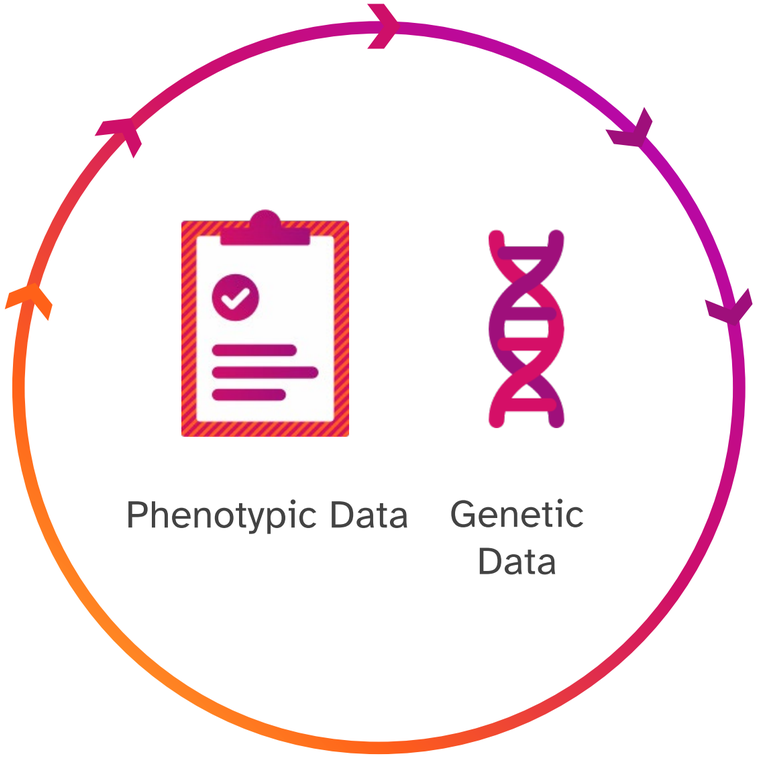
We run hundreds of billions of association tests per year
that further our unique understanding of human biology

Consumer | Research | Therapeutics / Product

11.9M
Genotyped Customers[1]

Data

80%
Opt-In
to Research



Insights

4B+
Phenotypic
Data Points[1]

Drug Discoveries
40+ Programs[2]

Novel Consumer Products

Innovative Results Return Value to the Customer

1. As of September 30, 2021. 2. As of March 31, 2021. Programs include collaborated, 100% owned and royalty interest targets.

What’s Coming:

- **Next Generation Reports:** Polygenic Risk Score reports that incorporate life-style factors to improve risk estimates

- **Genetics-based Primary Care:** Delivering personalized, prevention-oriented, genetics-based healthcare at scale by integrating Lemonaid Health’s digital health platform with 23andMe’s personal genetic services

- **Advancing Therapeutics Pipeline:** Advancing a pipeline of multiple clinical and research stage investigational programs addressing targets validated by human genetics

Therapeutics Program Overview

Kenneth Hillan, M.B., Ch.B.
Head of Therapeutics

Limited Use of Genetic Data and Lack of Patient Engagement Constrain Productivity

Drug Development is Inefficient

7
years average time-to-IND[1]

~90%
failure rate[2, 3]

$2.6B
average cost of drug development[3]

1. IND = Investigational New Drug Application. fdareview.org, “The Drug Development and Approval Process” (2020).
2. Probability of success for a drug to be approved is estimated to be <12%.
3. PhRMA, “Biopharmaceutical Research & Development: The Process Behind New Medicines” (2015).

Pharmaceutical Industry

7
years average time-to-IND[1]

~90%
failure rate[2]

23andMe

~4
years to IND with CD96 drug

Targets with genetic evidence have historically had a higher Success rate[3]

NATURE GENETICS PUBLICATION

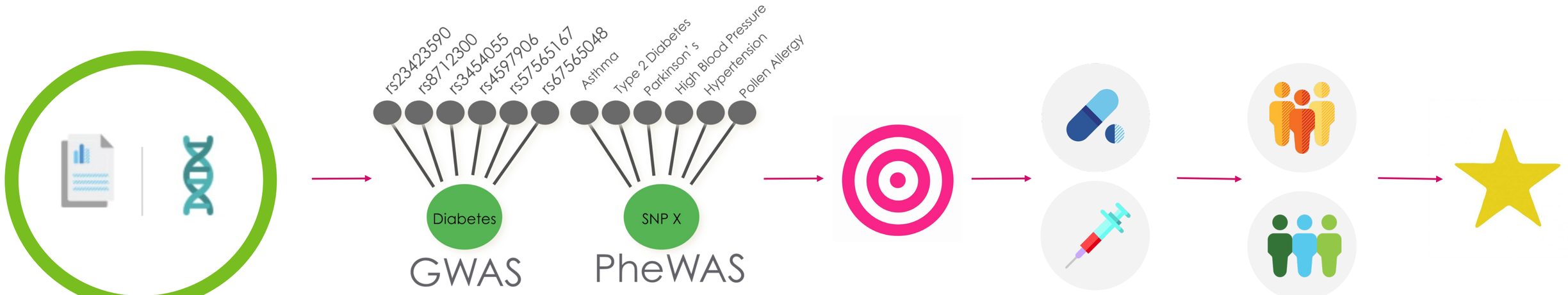
The support of human genetic evidence for approved drug indications

Nelson et. al 2015

Potential to More Efficiently Develop Novel Therapeutics by “**Power**, **Need**, and **Speed**”

[1] IND = Investigational New Drug Application. fdareview.org, “The Drug Development and Approval Process” (2020).
[2] Probability of success for a drug to be approved is estimated to be <12%. PhRMA, “Biopharmaceutical Research & Development: The Process Behind New Medicines” (2015).
[3] Nature Genetics Publication, “The support of human genetic evidence for approved drug indications” (2015).

DNA-based Target Discovery Playbook: How it works



Survey & Genetic Data | Target Identification by Phenotype | Drug Discovery | Clinical Trials | Product Approval

Our Scale Enables Real-Time Genetics Health Research[1]

1,876,573 High cholesterol	**358,275** Type 2 Diabetes	**37,853** Type 1 Diabetes
1,785,456 Depression	**2,355,068** APOE e4 carriers (Alzheimer's risk)	**85,604** Epilepsy
1,113,057 Asthma	**667,019** Eczema	**250,764** Psoriasis
634,734 Irritable Bowel	**107,126** UC / Crohn's	**64,800** Barrett's Esophagus
534,696 Arrhythmia	**159,135** Coronary Artery	**42,836** Pulmonary Embolism
9,047 Systemic Sclerosis	**7,334** Sarcoidosis	**4,528** Idiopathic Pulmonary Fibrosis

[1] As of August 2, 2021. [2] As of September 2021. [3] 23andMe COVID-19 manuscript live on MedRXiv September 7, 2020.

1,287,060[2]
COVID-19 study participants

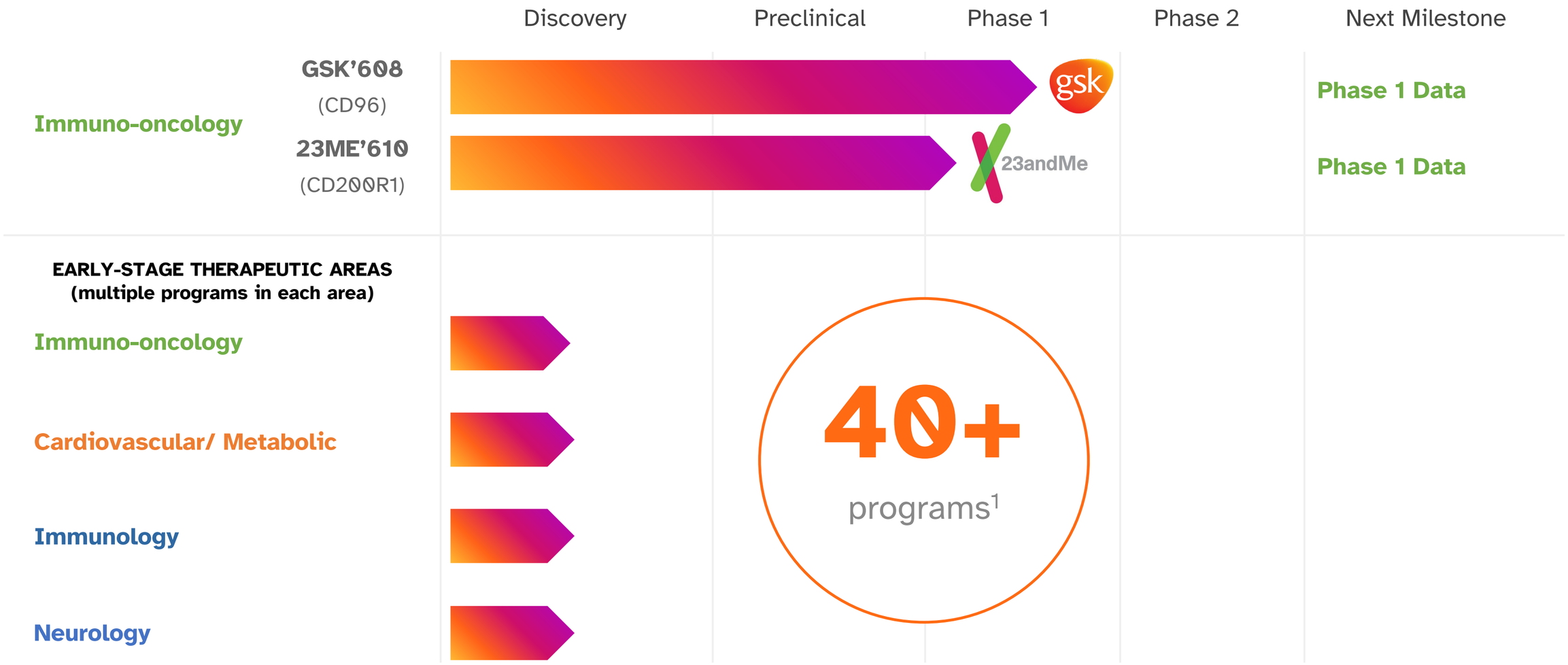
750K
Consumers participated in the COVID-19 study in the **first 90 days**

COVID-19 Research (2020)

- **March 16** Kicked Off Study
- **April 6** Launched Study
- **June 8** Preliminary Findings
- **Sept. 7** Posted Findings[3]

Re-contactable Customers Participate in Health Research

We Have Generated a Research and Development Pipeline Covering Multiple Therapeutic Areas



[1] 40+ programs in the combined therapeutic areas. Programs include collaborated, 100% owned and royalty interest targets. Note: As of March 31, 2021

Target Discovery Vision

Joe Arron, M.D., Ph.D.
Chief Scientific Officer, Therapeutics

My Background

- MD/PhD **Cornell** and **Rockefeller**
- Postdoc **Stanford**

- **Genentech** 2006-2021
  - VP and Senior Fellow, Immunology Research
    - Led target discovery for inflammatory, fibrotic, & ophthalmic diseases across >20 laboratories
  - Developed forward & reverse translational strategies across multiple disease areas
  - Contributed to >25 clinical programs from discovery through postmarketing
  - Authored >75 peer-reviewed publications

Human Genetics has the Potential to Double the Probability of Success in Drug Development

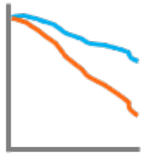
Reasons for failure

Wrong target

- Hypothesized target not a critical node in disease pathogenesis
- Safety issues associated with target

Wrong drug

- Insufficient affinity/avidity; off-target effects
- Poor PK/tissue penetration/inadequate dosing



Wrong outcomes

- Clinical outcome measure not related to biology of target
- Clinical outcome measure not relevant in trial population

Wrong patients

- Patients not properly stratified according to molecular, pathophysiological, or clinical heterogeneity
- Trials underpowered to detect an effect in the right subset

Our rich database and translational focus has the potential to mitigate these and increase probability of success

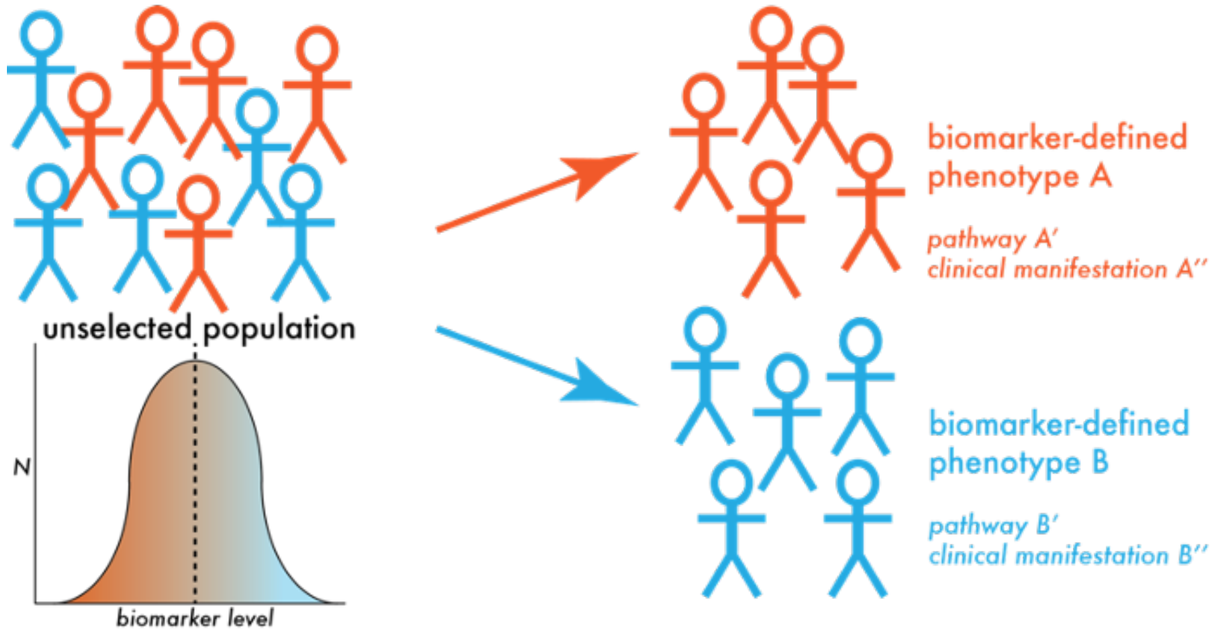
Focusing on Translational Research to Link Targets and Outcomes

TARGET/MOA ⟷ PATHOPHYSIOLOGY ⟷ CLINICAL ENDPOINTS ⟷ VALUE TO PATIENTS
meaningful benefit

identify indications and develop outcome measures most relevant to biologically compelling targets

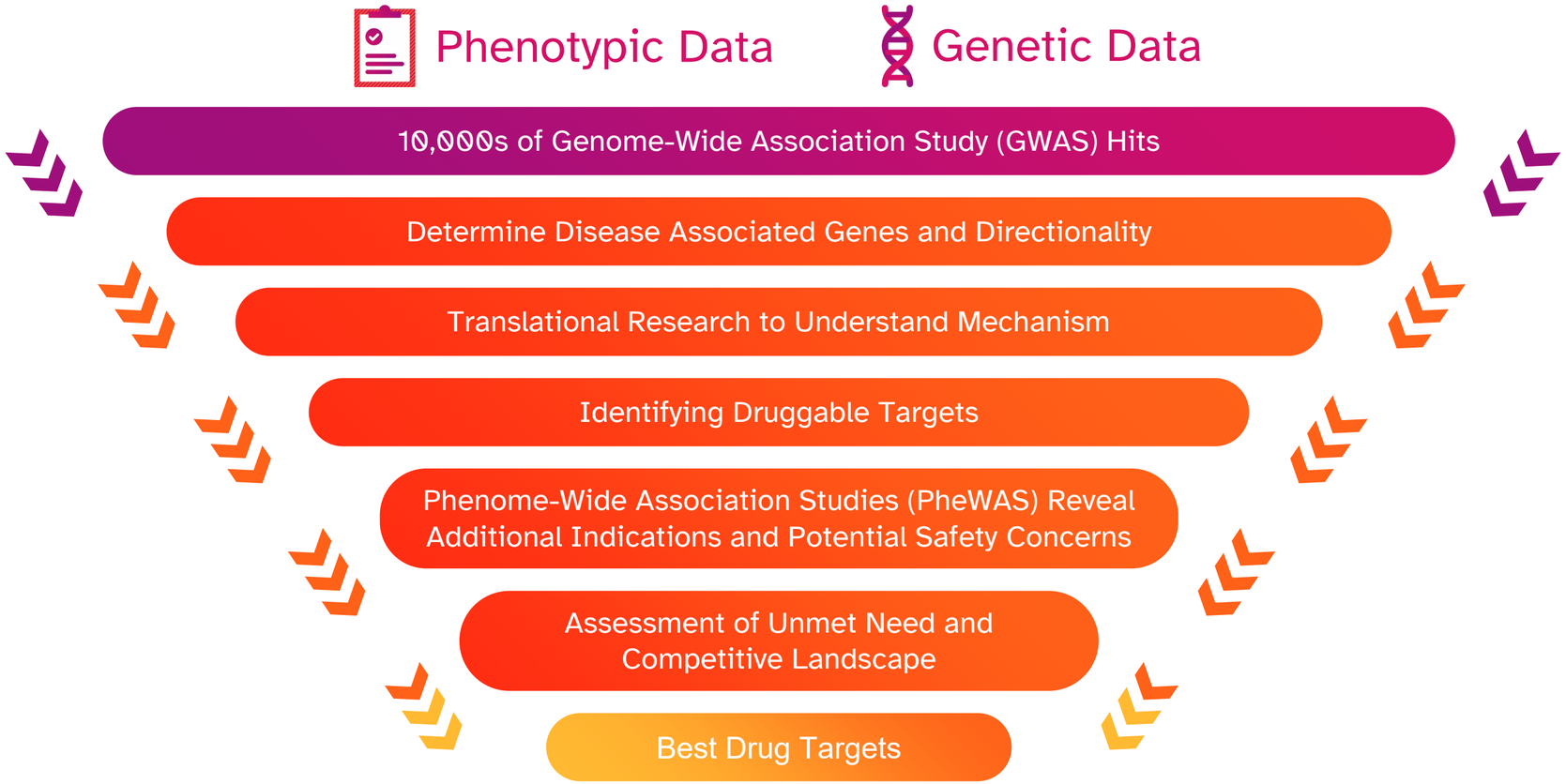
BENCH → BEDSIDE

develop targets that are most relevant to unmet needs in existing indications



Townsend & Arron, *Nat Rev Drug Discov* **15:**517 (2016)

Systematic, Scalable Research Platform Yields Novel Drug Targets



Wet lab validated targets progress through standard stages of research toward the selection of preclinical lead molecules and clinical development

23andMe's database yields thousands of GWAS hits

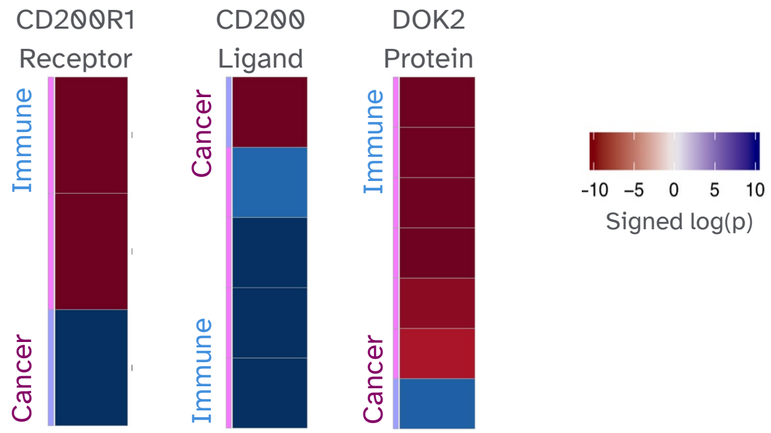
Advanced biology and medicinal chemistry guide design of optimal compounds from initial targets

Phenotypic breadth provides unique ability to uncover potential safety issues or possible indication expansions

Leveraging our database: I/O signature implicates CD200R1 pathway

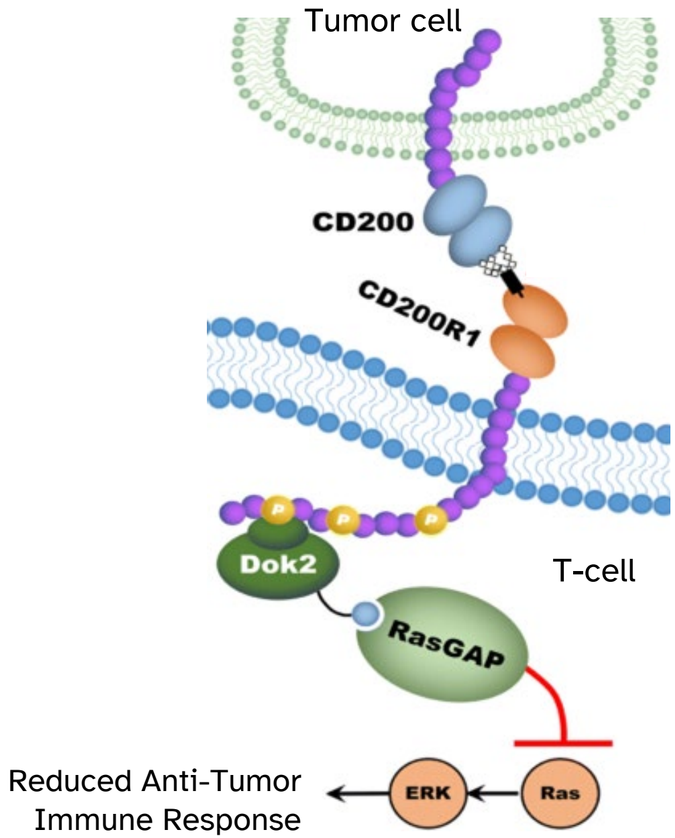
CD200R1 pathway identified as a critical immune checkpoint with our I/O genetic signature

I/O genetic signature shows opposing effects on autoimmune and cancer phenotypes



Implicates ***3 components*** of the CD200R1 signaling pathway

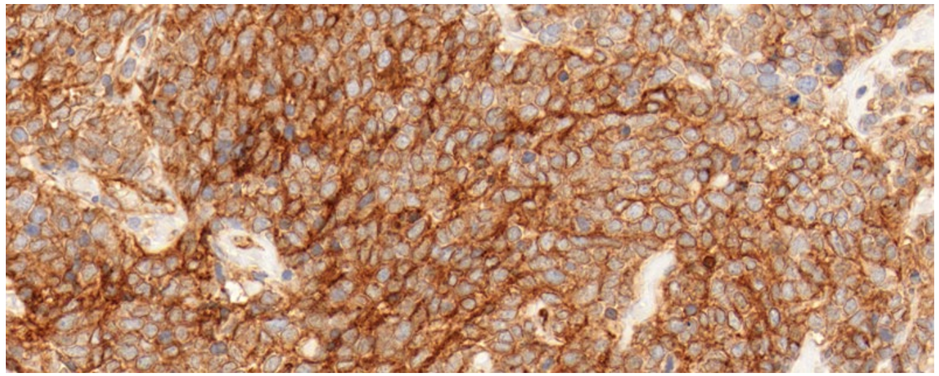
CD200:CD200R1 Signaling



We are applying similar approaches to many diseases that are well-represented in our database

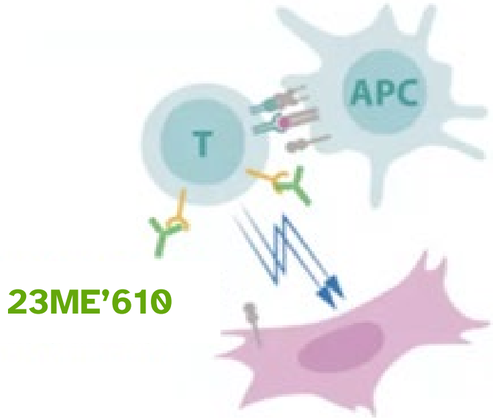
23ME’610 is an antibody against CD200R1 that can block CD200-mediated immune suppression

CD200 is strongly expressed in a subset of human tumors



CD200 immunohistochemistry (brown) shows expression on tumor cells

Inhibiting CD200R1 signaling enhances anti-tumor immune responses



Consumer Powered Healthcare Flywheel

We run hundreds of billions of association tests per year that further our unique understanding of human biology

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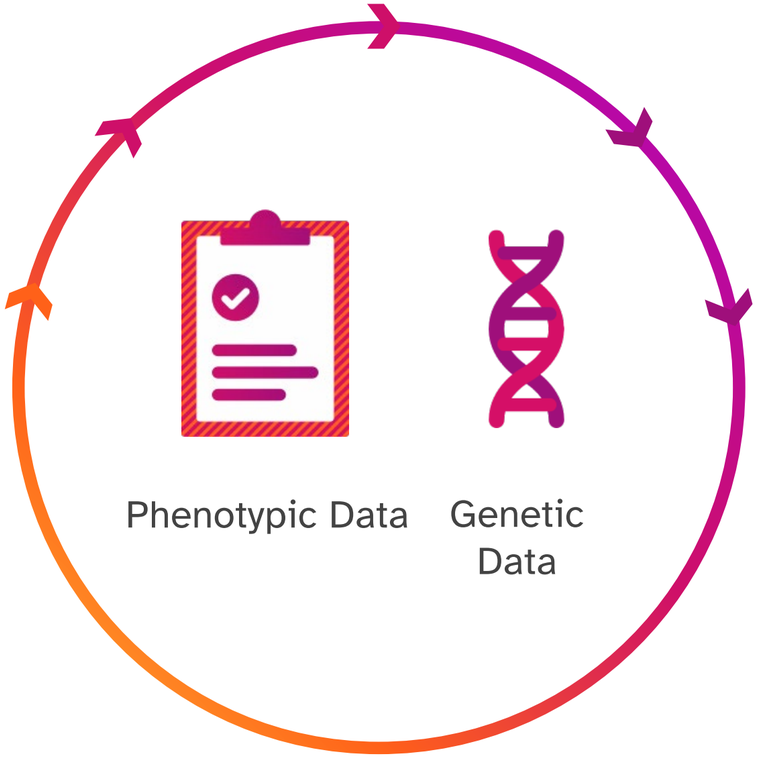
80%
Opt-In to Research



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Genetics-based Target Discovery

Adam Auton, Ph.D.
Vice President, Human Genetics

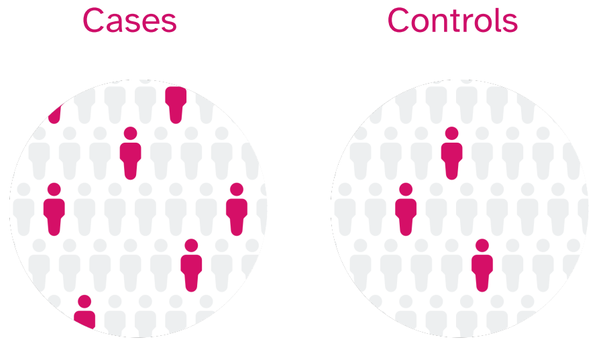
Genome-Wide Association Studies (GWAS)

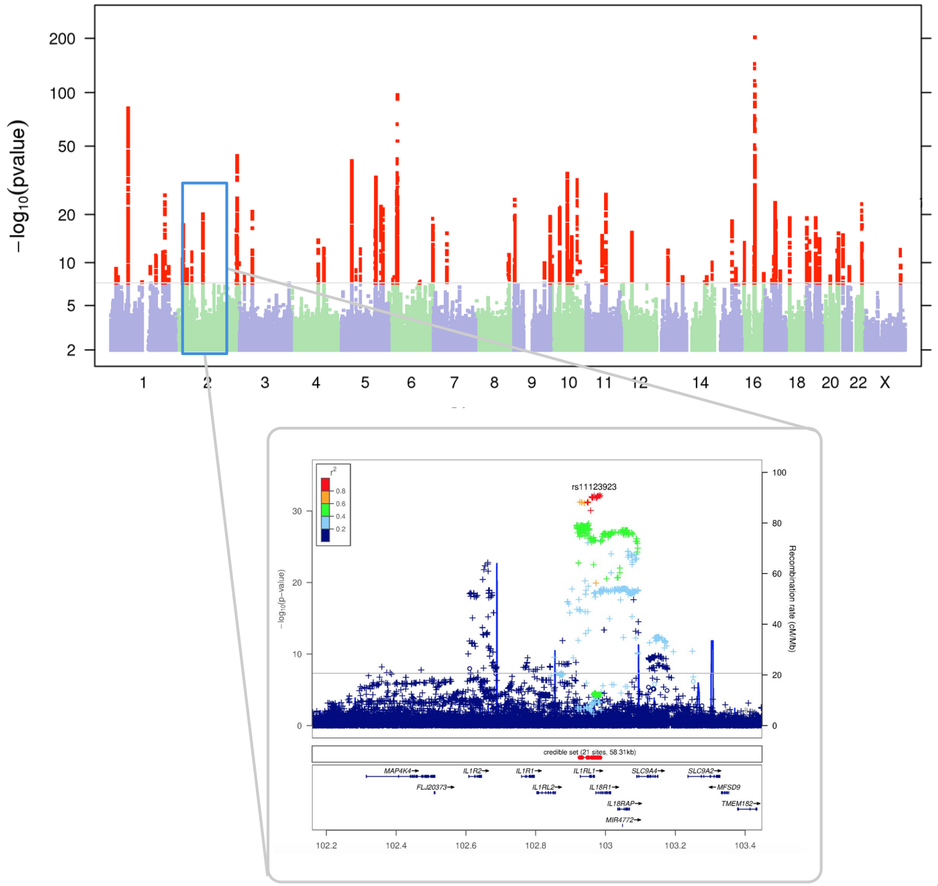
- GWAS is a statistical analysis of Single Nucleotide Polymorphisms (SNPs), looking To identify differences in frequency between disease cases and controls.
- SNPs linked with disease will be found at different frequencies in cases versus controls.
- Association is represented by the level of statistical significance (p-value) of the SNP frequency difference.
- SNPs can be tested across the genome and mapped to specific regions.

Single Nucleotide Polymorphism (SNP)

GGCCAGCTGGACGAGG

GGCCAGCTGGATGAGG

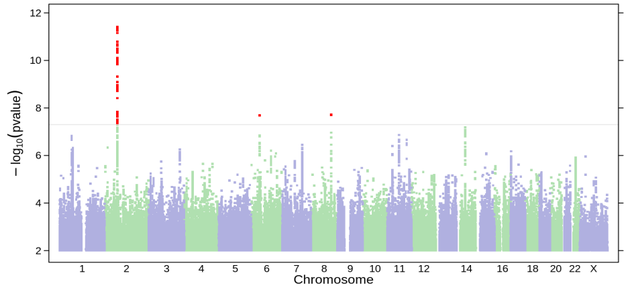




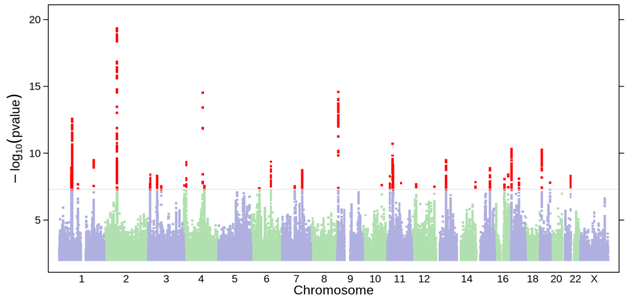
Size and Scale Accelerate Target Discovery

Example: Number of Osteoarthritis GWAS[1] hits dramatically increase as database grows

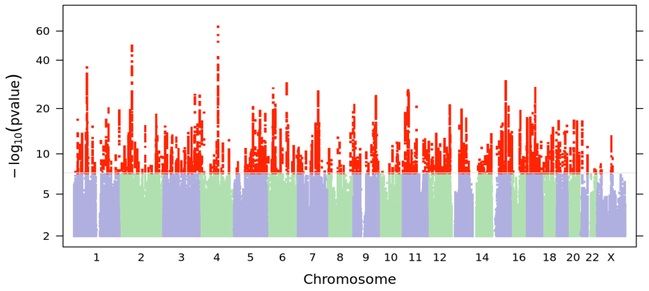
2016



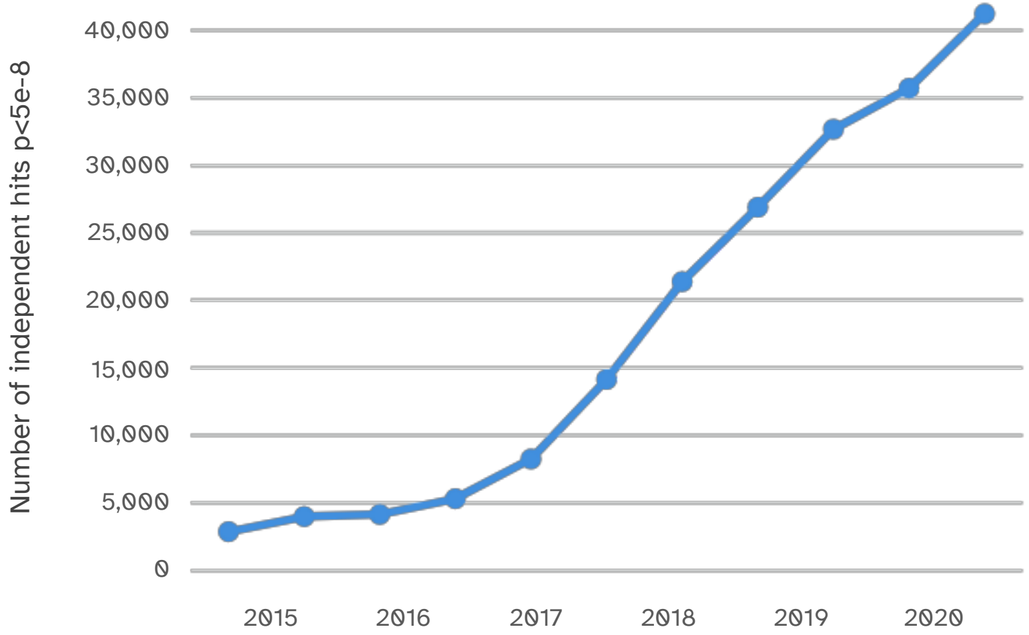
2017



2021

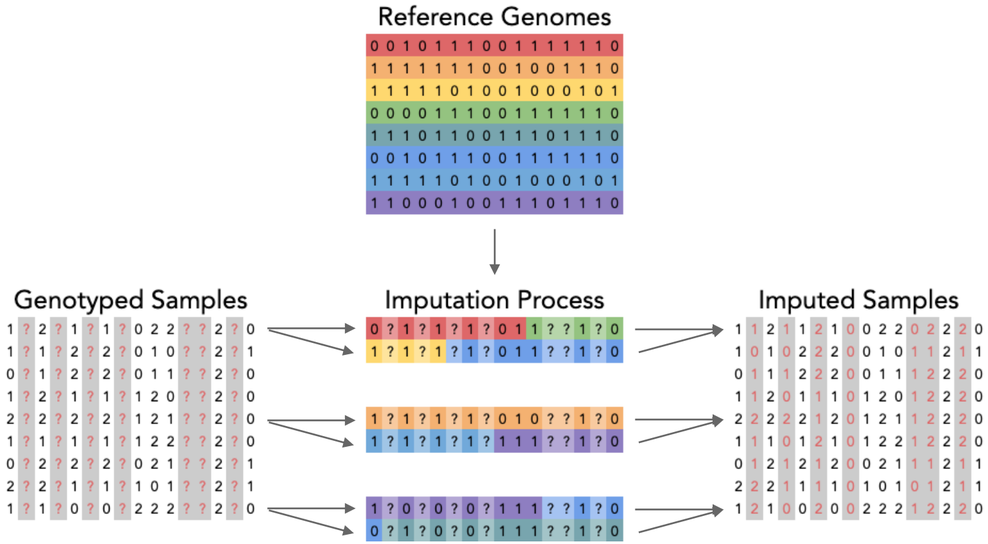


New programs are identified through GWAS[1] hits, which increase as size of database grows



1 GWAS: Genome-Wide Association Study.

The Vast Majority of GWAS Discoveries Can be Made Without Large-scale Sequencing



Nearby genetic variants are correlated with each other. Knowing the variants one position allows the nearby variants to be inferred.

- E.g. Fill in the blanks:

  The q*k brown f*x jumps ov*r the **zy dog.**

- The same principle applies in genetics. The process of filling in the gaps is known as ‘genotype imputation’.

The Vast Majority of GWAS Discoveries Can be Made Without Large-scale Sequencing

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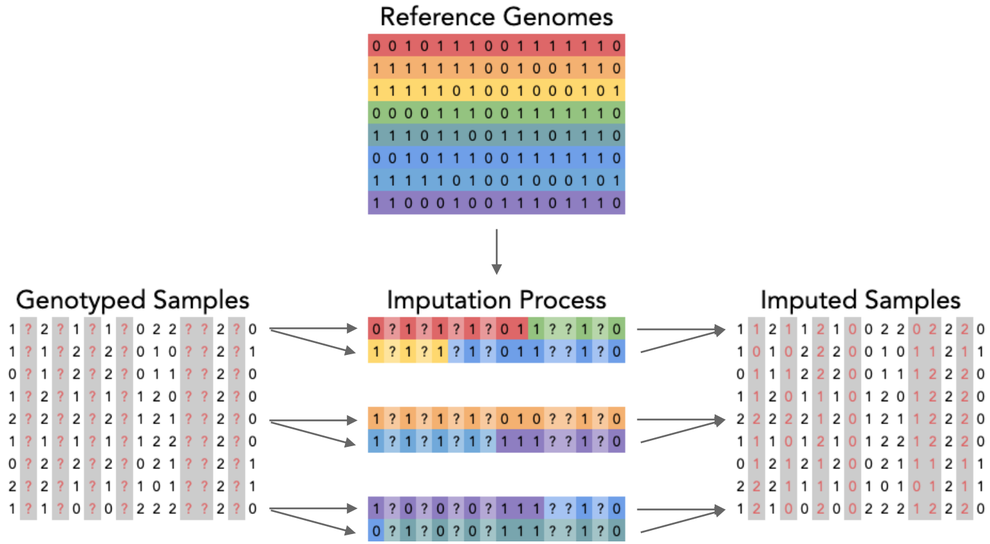
- The same principle applies in genetics. The process of filling in the gaps is known as ‘genotype imputation’.

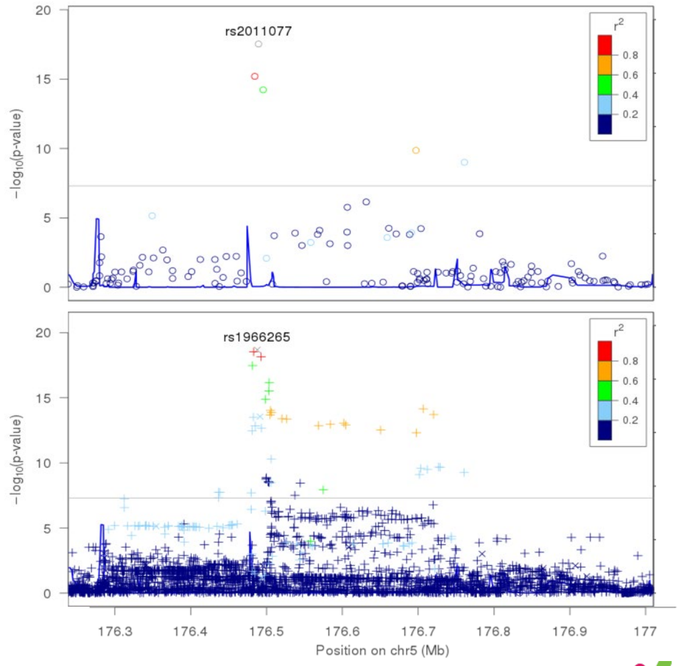
We type ~650,000 SNPs using our genotyping array, which allows accurate imputation for >35m SNPs in the genome.

Genotype imputation is much more cost effective than large-scale sequencing.

- Whole-genome sequencing ~$1000 / sample.
- Exome sequencing ~$400 / sample.
- Imputation < $0.01 / sample

We do deploy sequencing in situations where there is a clear benefit over and above imputation (e.g. rare disease).

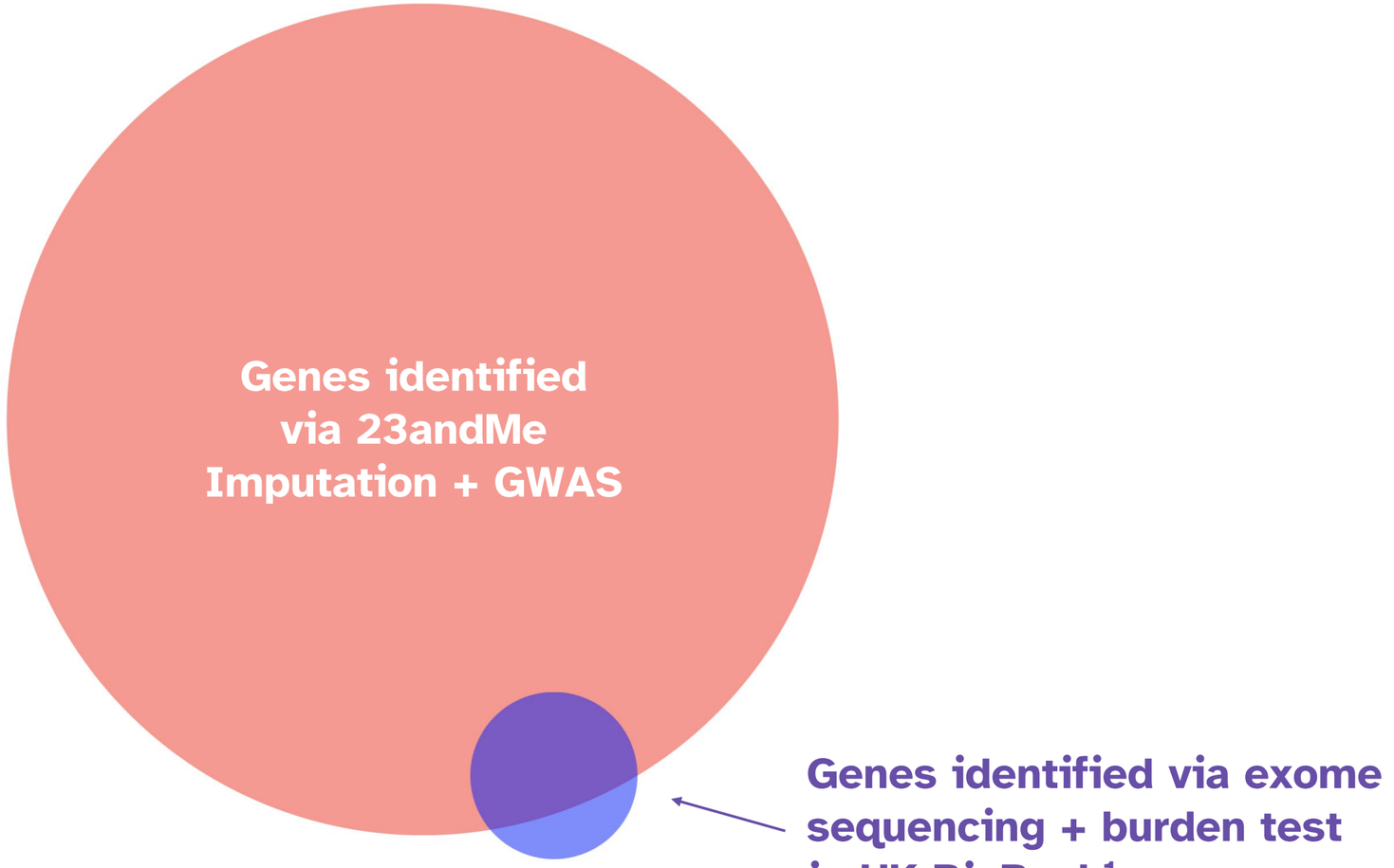




Before imputation

After imputation

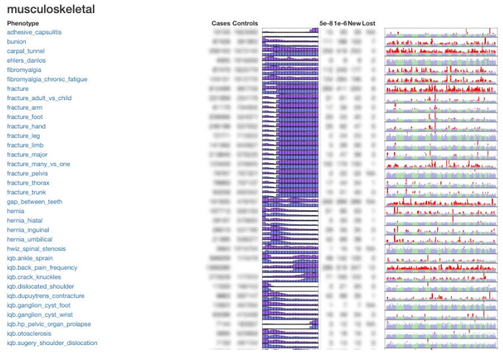
Imputation and GWAS Enables Discovery of Vast Majority of Genes Identified via Exome Sequencing



1. List of genes identified via UKBB exome sequencing: Backman *et al*., Nature 2021. Exome sequencing and analysis of 454,787 UK Biobank participants

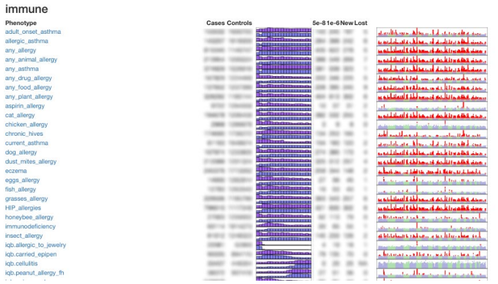
Hundreds of Distinct Clinical Phenotypes Across Major and Rare Diseases

Orthopedic

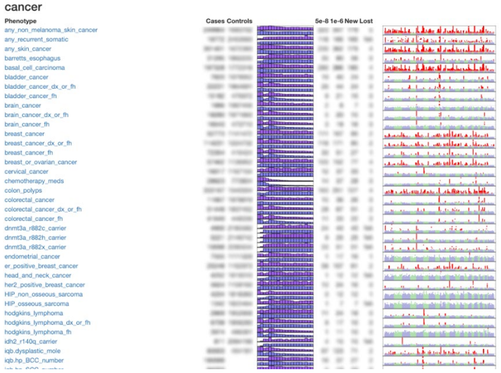


Neurology

Allergy

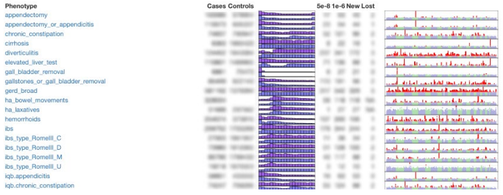


Cancer

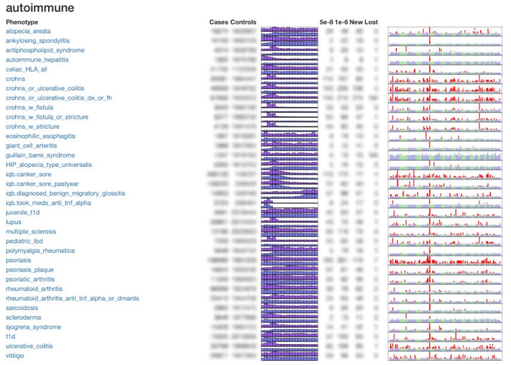


Cardiovascular

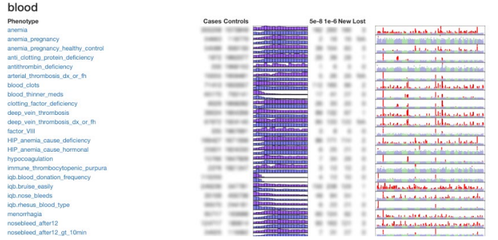
G.I.



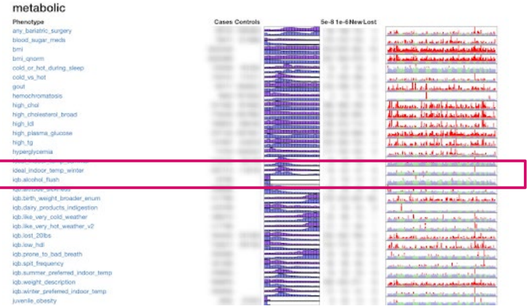
Autoimmunity



Hematology

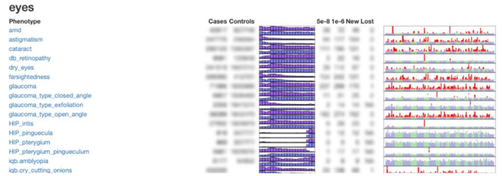


Metabolic Disease



Infectious Disease

Ophthalmology

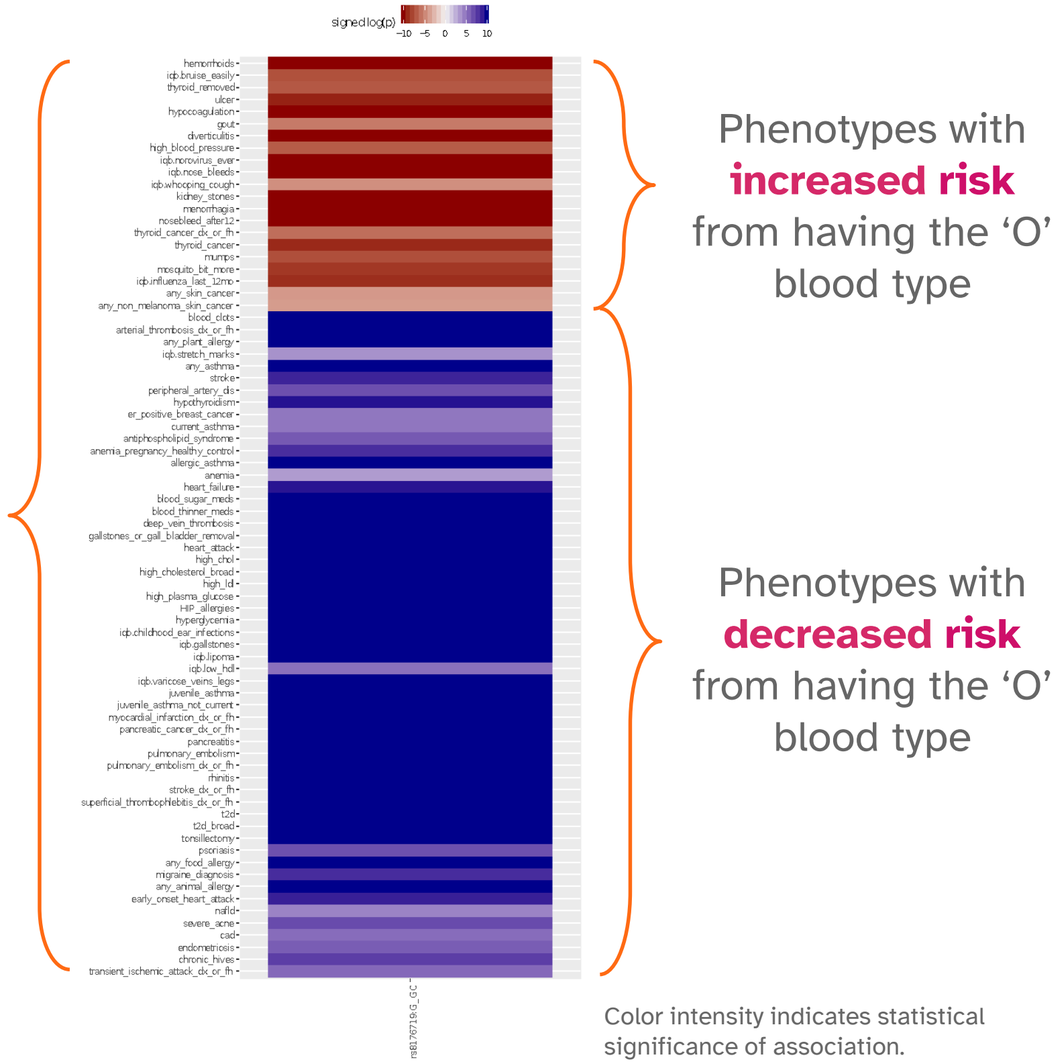


Endocrine

Phenotype	Cases	Controls	Hits	New	Lost
NAFLD (Non-Alcoholic Fatty Liver Disease)	48048	2517644	104	44	2

Breadth of Phenotyping Provides Deeper Genetic Understanding Across Multiple Diseases

- **PheWAS** = Phenotype Wide Association Study
- Every SNP in the genome can be interrogated at >1,000 medically related phenotypes
- Besides the role of a gene in a disease of interest, we can use genetics to learn **potential indication expansions or possible unwanted effects**

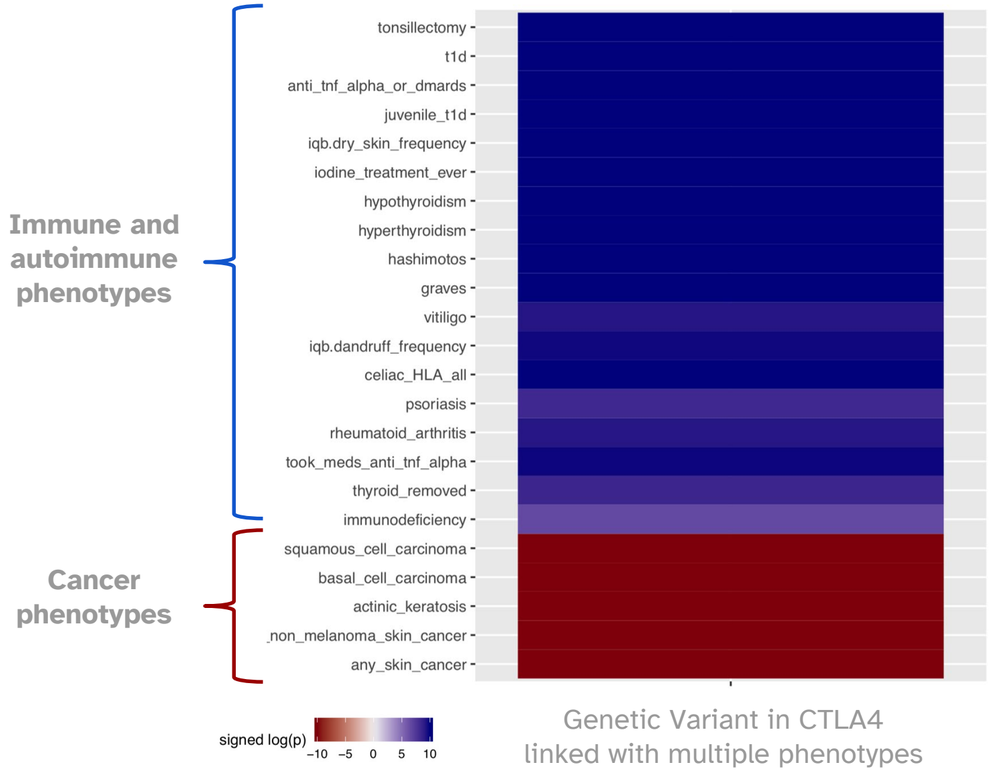


Color intensity indicates statistical significance of association.

Using PheWAS to Identify Immuno-oncology Targets

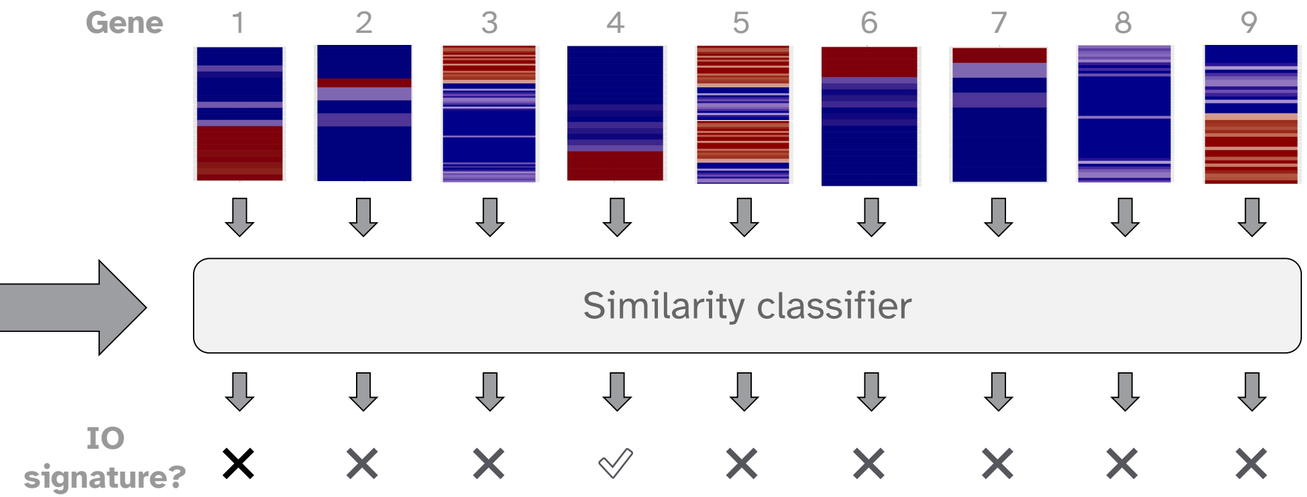
We have defined an '**immuno-oncology signature**'; genetic evidence that a particular gene both activates the immune system and simultaneously reduces cancer risk.

Identified novel immuno-oncology signature around CTLA4.



Genetic Variant in CTLA4 linked with multiple phenotypes in the 23andMe database

Use machine learning (ML) techniques to scan the genome and identify genes with similar signatures.



23ME-00610 (P006): A Novel Immuno-oncology Antibody Targeting CD200R1

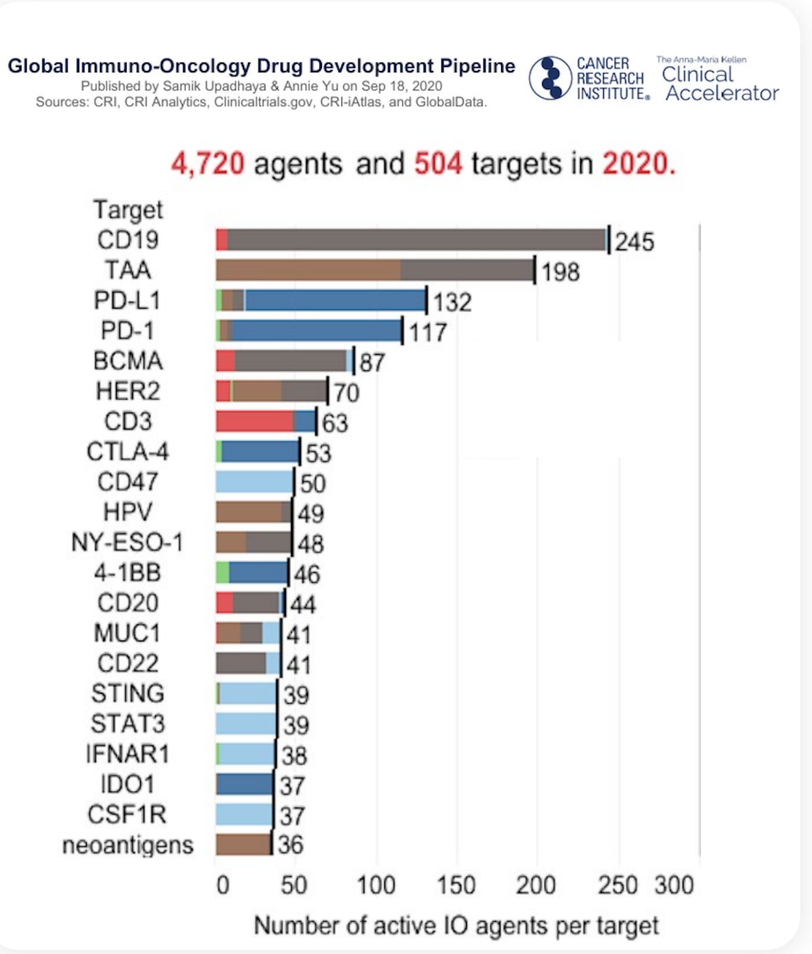
Jennifer Low, M.D., PhD
Head of Therapeutics Development, and
Adrian Jubb, M.B., Ch.B., Ph.D.
Senior Clinical Development Fellow

23andMe Immune-Oncology (I/O) Signature Highlights Genetically-Driven Targets

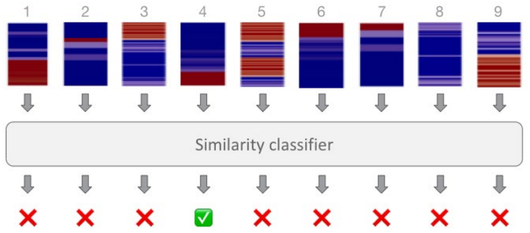
Large I/O market with over $41B expected in 2021 sales[1]

2021 projected sales of leading checkpoint inhibitors

KEYTRUDA	$17.0B
OPDIVO	$7.9B
YERVOY	$1.8B



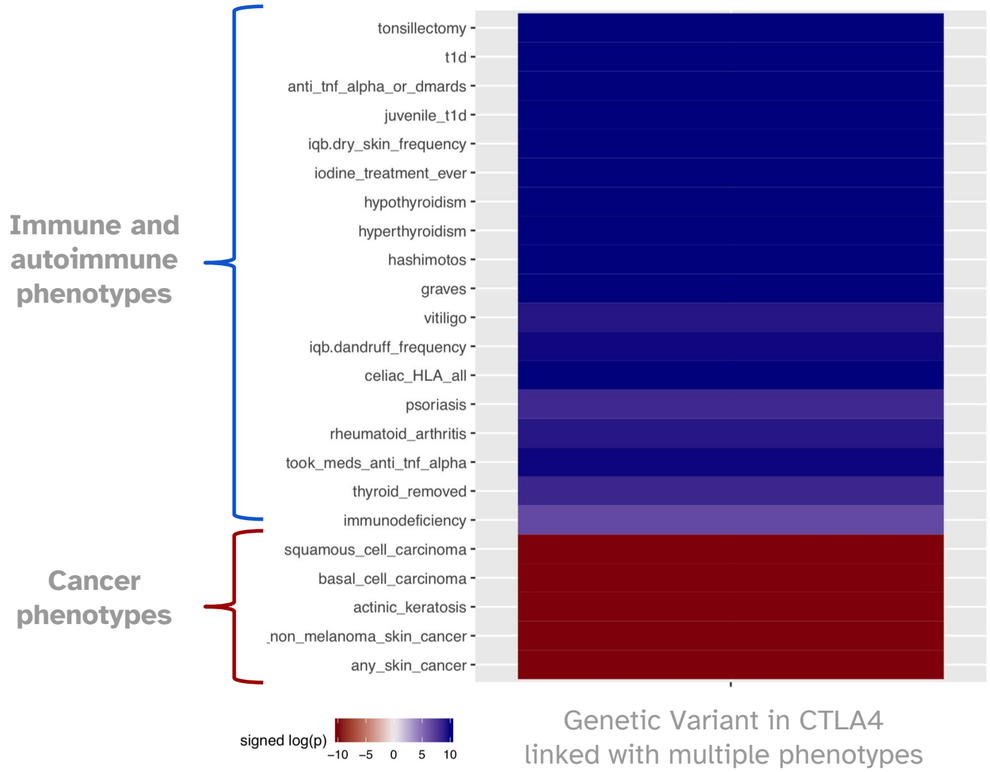
23andMe's I/O signature identifies targets that are genetically-driven



1 Source: Evaluate Pharma historical and forecast estimates.

CD200R1 was Identified as a Promising Anti-Cancer Drug Target with 23andMe's Proprietary Immuno-oncology (I/O) Genetic Signature

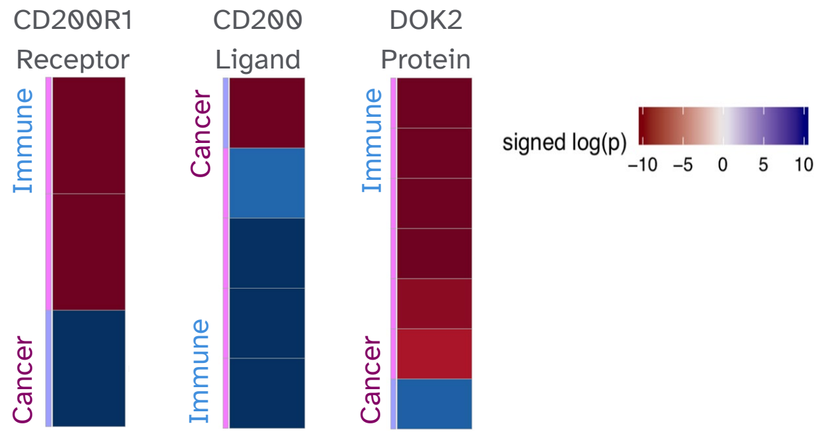
Identified novel immuno-oncology signature around CTLA4.



Genetic Variant in CTLA4 linked with multiple phenotypes in the 23andMe database

CD200R1 pathway identified as a critical immune checkpoint with our I/O genetic signature

I/O genetic signature shows opposing effects on autoimmune and cancer phenotypes

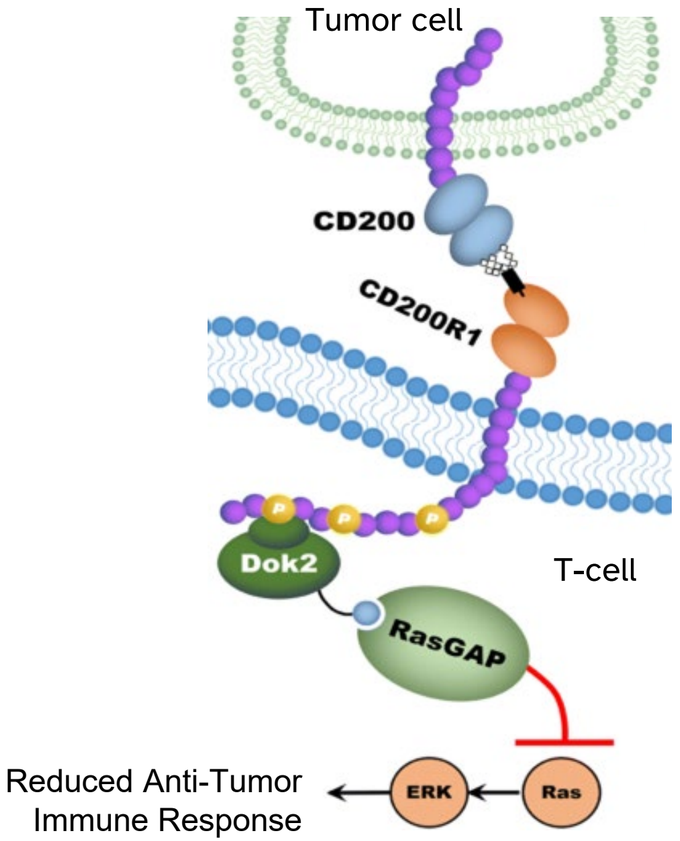


We discovered that *3 components* of the signaling pathway for CD200R1 have a similar genetic signature to other I/O drugs

CD200R1 is an Immune Checkpoint

- CD200R1 is an inhibitory receptor expressed on T-cells and myeloid cells
- CD200 is the only known ligand for CD200R1 in humans and is highly expressed in certain cancers
- Binding of CD200 to C200R1 decreases the ability of T-cells to recognize and kill cancer cells
- Several viruses, including HHV8 have co-opted CD200 analogues to suppress and evade the host immune response

CD200:CD200R1 Signaling

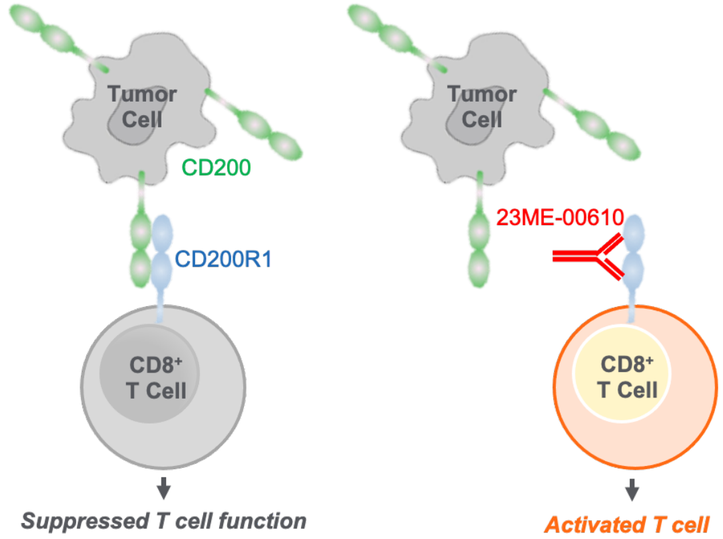


References: J Virol 2012;86:6246, J Virol 2004;78:7667, J Immunol 2005;175:4441, Structure 2013;21:820, JCI Insight 2018;3:e96836

23ME-00610 (23ME'610) Binds with High Affinity to CD200R1 and Inhibits Immunosuppressive Signaling

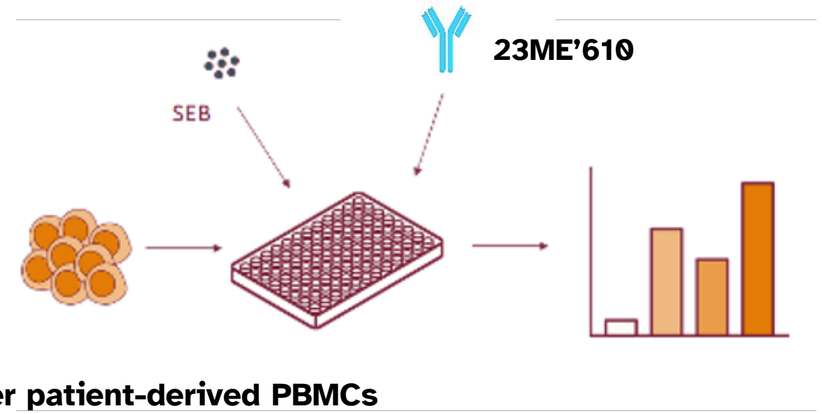
- 23ME '610 is a fully humanized, effectorless, IgG1 antibody against human CD200R1
- 23ME '610 binds CD200R1 with high affinity (K_D < 0.1 nM)
- 23ME '610 blocks CD200 ligand binding to CD200R1, resulting in inhibition of immunosuppressive signaling
- The restoration of T-cell activity by 23ME '610 was demonstrated using in vitro models of the tumor microenvironment
- No adverse effects of blocking CD200R1 have been observed in nonclinical toxicology studies

23ME'610 Activates T-cell Function by Blocking the CD200R1 Checkpoint



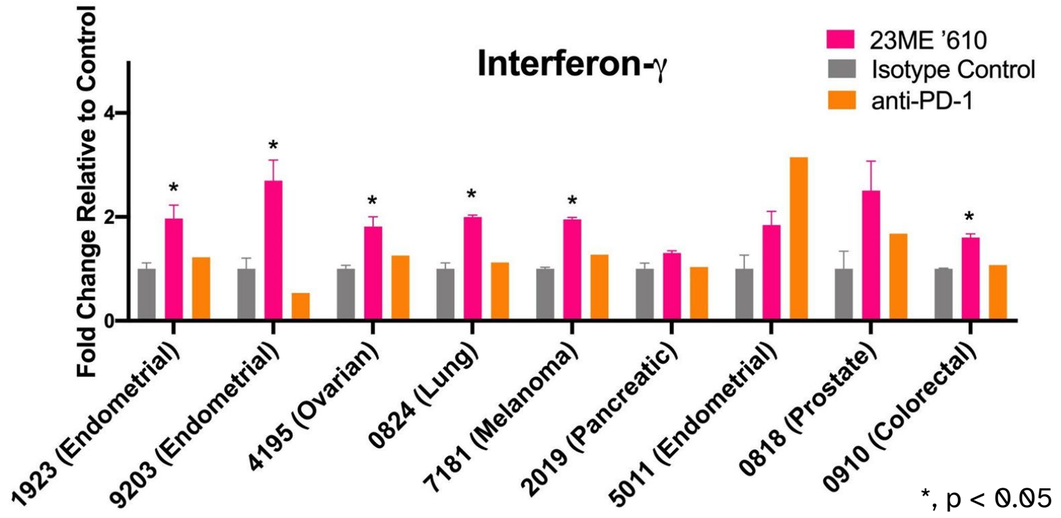
23ME’610 Shows Broader Enhancement of Proinflammatory Cytokine Secretion than Anti-PD1

Mixed immune cell assay that stimulates T cells with MHCII binding bacterial super antigen



PBMCs: Peripheral Blood Mononuclear Cell (T cells, B Cells, NK Cells, Monocytes)
SEB: Staphylococcal Enterotoxin B (bacterial super antigen)

23ME’610 Increases Interferon-gamma Secretion from SEB-Stimulated Cancer Patient PBMCs



- Interferon-gamma is a pro-inflammatory cytokine that is secreted by activated T cells
- 23ME ’610 increases interferon-gamma secretion from SEB-stimulated cancer patient PBMCs compared to the isotype control antibody and anti-PD-1 in the majority of tumor samples tested

Phase 1 Study of 23ME’610 in Patients with Locally Advanced or Metastatic Solid Malignancies

Study Design

1

Phase 1

Openlabel

Non-Randomized

Multi-center

Patients with locally advanced, unresectable or metastatic solid tumors that have progressed after or are inappropriate for standard therapy

Part A (n ≤ 26)

Monotherapy Dose Escalation (IV Infusion Q3W)

Accelerated Titration

3+3 Cohorts

RP2D / MTD

Part B (n = 75)

- **Expansion Cohort**
- **Expansion Cohort**
- **Expansion Cohort**
- **Expansion Cohort**
- **Expansion Cohort**

Objectives

Primary

- Part A: Safety (DLTs, AEs)
- Part B: Efficacy (ORR)

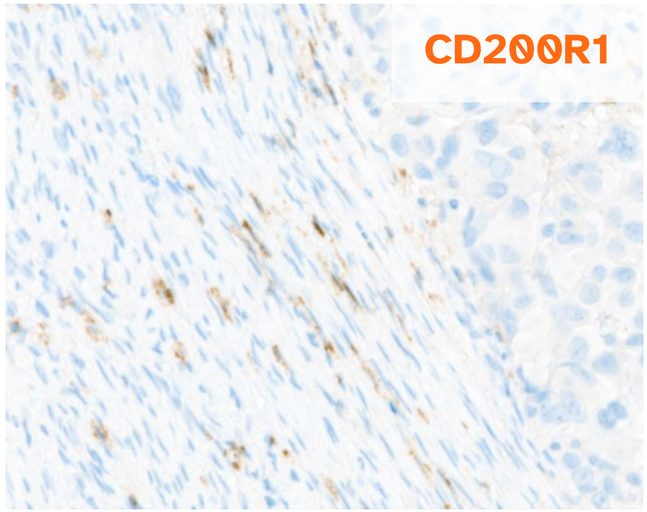
Secondary and Exploratory

- Efficacy (ORR [RECIST and iRECIST]), DoR, PFS, OS) and Safety
- Pharmacokinetics
- Pharmacodynamic biomarkers

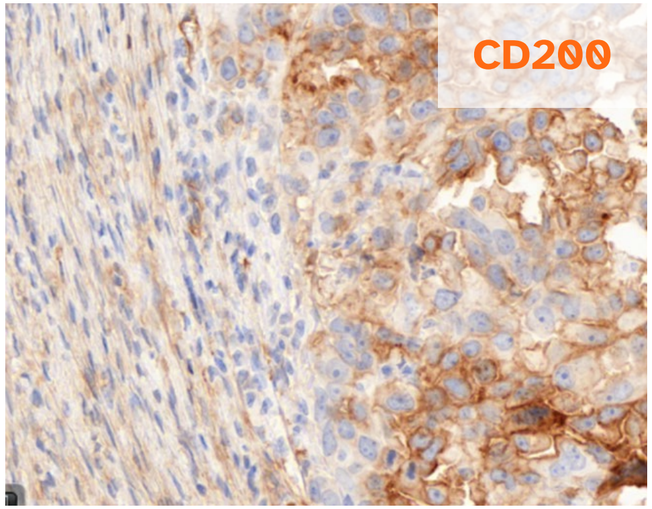
Abbreviations: AEs: Adverse Events; DLT: Dose limiting toxicity; DOR: duration of response; IV: intravenous; ORR: Objective Response Rate; OS: Overall Survival; PFS: Progression Free Survival; Q3W: every three weeks; RECIST: Response Evaluation Criteria in Solid Tumors; RP2D: Recommended Phase 2 Dose

CD200R1 Ligand (CD200) is Highly Expressed in a Subset of Human Tumors

CD200R1 and CD200 Protein are Co-expressed in Ovarian Cancer



CD200R1 immunohistochemistry (brown) shows expression on immune cells within and around the tumor



CD200 immunohistochemistry (brown) shows expression on tumor and stromal cells

Source: 23andMe Data

Why Target the Receptor (CD200R1) Instead of the Ligand?

- **CD200R1** expression is mainly expressed on immune cells
  - **CD200** (ligand) is broadly expressed on many cell types
- An anti-**CD200** monoclonal antibody, samalizumab (**ALXN 6000**) did not saturate cell surface **CD200**[1]
  - evaluated in patients with **CD200**-expressing B-cell malignancies in a Phase 1 trial[1]
- **23ME'610** is expected to **saturate CD200R1** and fully **block binding** to **CD200**

	CD200R1	CD200
Target distribution	Immune cells	Expressed on a wide range of cells (B cells, endothelial cells, neuronal cells, etc)
	23ME'610	samalizumab
Antibody affinity (K_D)	< 0.1 nM	~10 nM

1 Journal for ImmunoTherapy of Cancer 2019;7:227

23ME '610 Targeting CD200R1: A Genetically-Validated Approach to Anti-Cancer Therapy

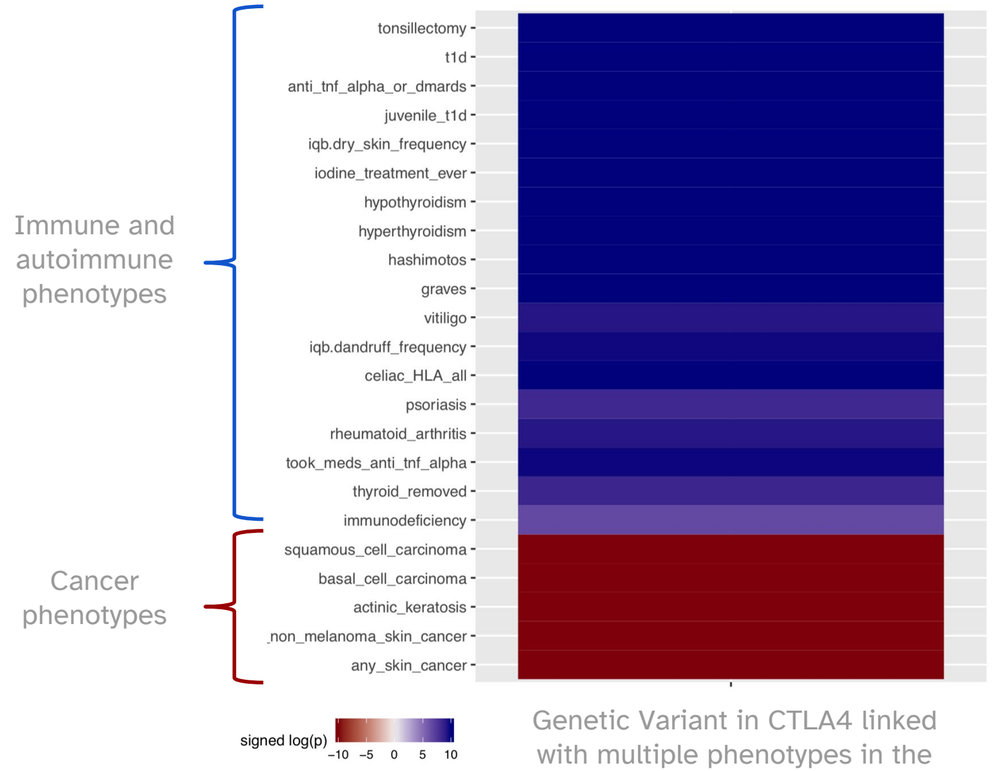
- 23andMe's I/O signature highlights potential targets with genetic evidence of importance
- CD200R1 is an immune checkpoint with a clearly defined I/O signature in three components of the pathway
- CD200R1 ligand is highly expressed in a subset of human cancers
- 23ME '610 is a potent monoclonal antibody against CD200R1 that has the potential to restore T-cell killing of cancer cells
- The Phase 1 study of 23ME '610 in patients with advanced solid malignancies has been initiated and the first patient was dosed in January 2022

CD96 Program: First Clinical-stage Immuno-oncology Antibody Targeting CD96

Jennifer Low, M.D., Ph.D.
Head of Therapeutics Development

CD96 was Identified as a Promising Anti-Cancer Drug Target with 23andMe's Proprietary Immune-Oncology (I/O) Genetic Signature

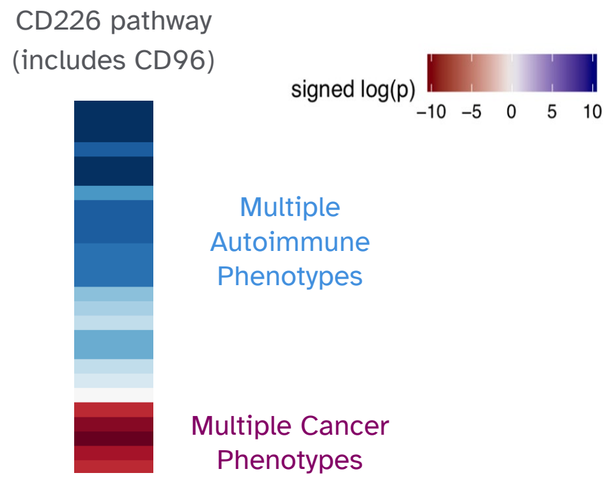
Identified novel immuno-oncology signature around CTLA4



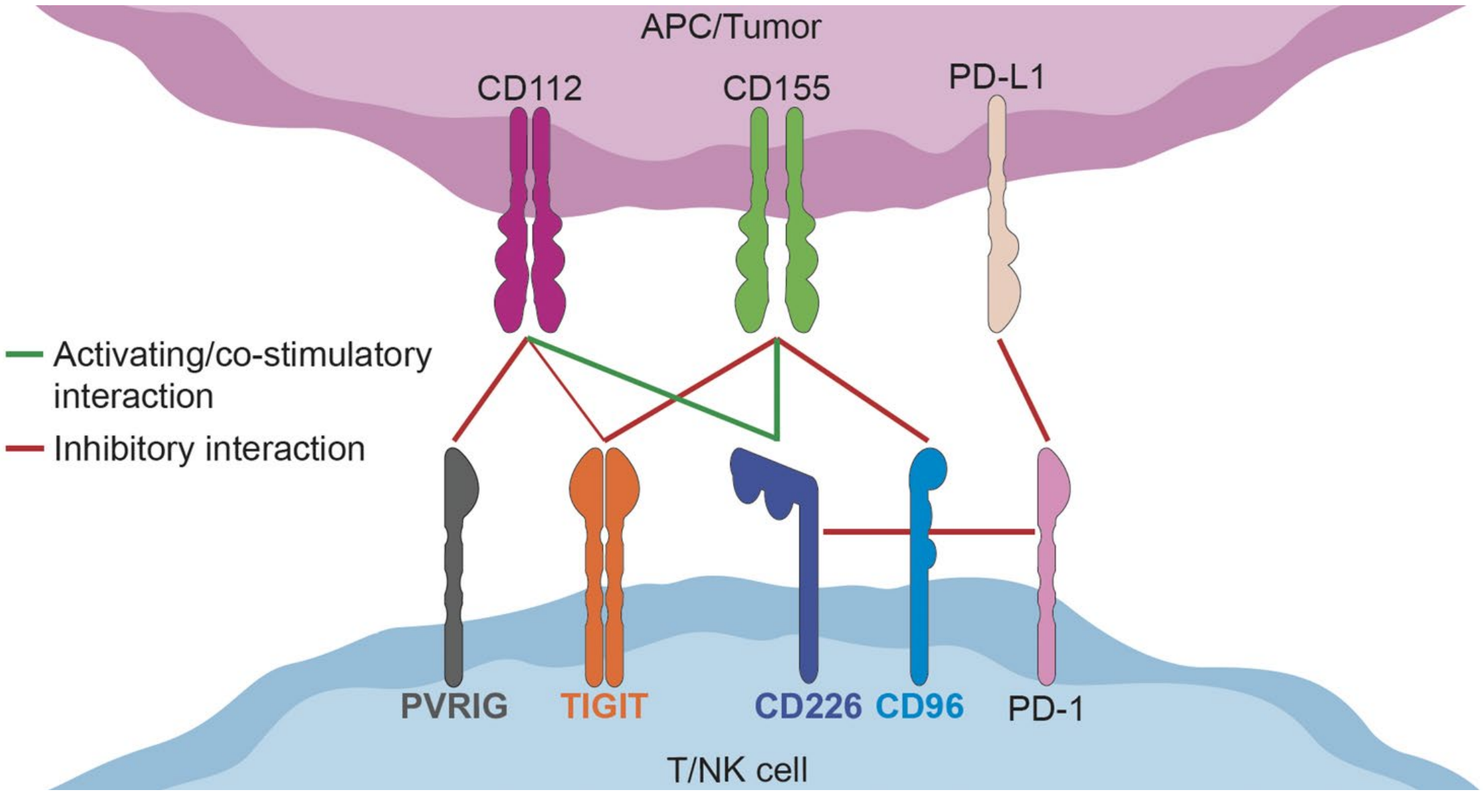
Genetic Variant in CTLA4 linked with multiple phenotypes in the 23andMe database

CD96/CD226 pathway identified as a checkpoint with our I/O genetic signature

I/O genetic signature shows opposing effects on autoimmune and cancer phenotypes



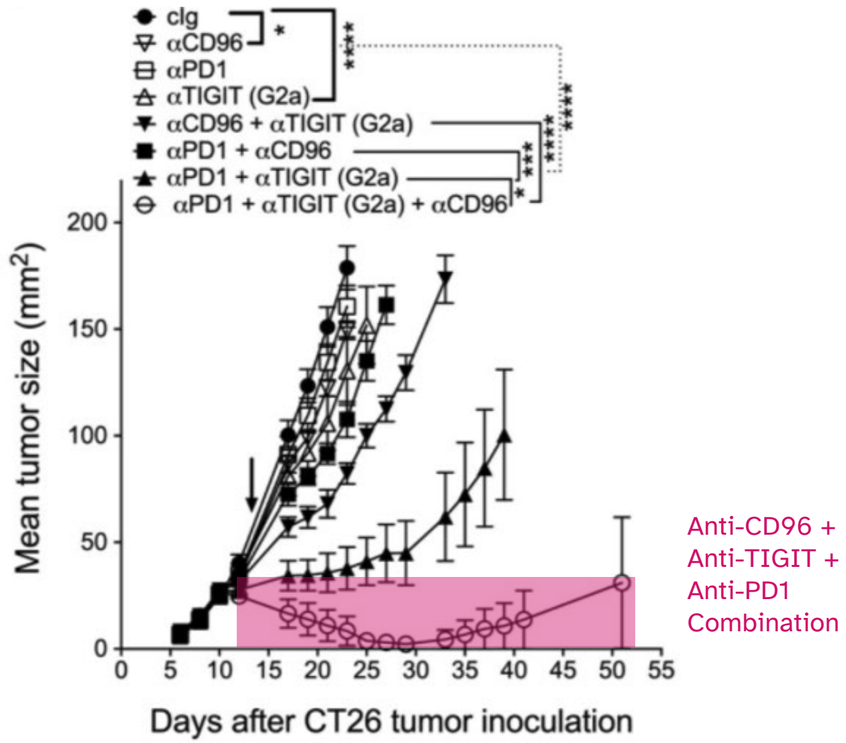
PD-1 is a Negative Regulator of the CD226 Axis
Inhibition of CD96 and TIGIT may enhance PD-1 activity



- CD226 activates NK/T-cells
- PD1 directly regulates CD226 activity
- TIGIT and CD96 indirectly suppress CD226
- Combining inhibitors (anti-PD-1, anti-CD96, anti-TIGIT) may have more activity that anti-PD-1 alone

Preclinical Data Supports Combining CD96 with PD-1 and TIGIT Inhibitors

CD96, TIGIT and PD-1 Combination Suggests Synergy

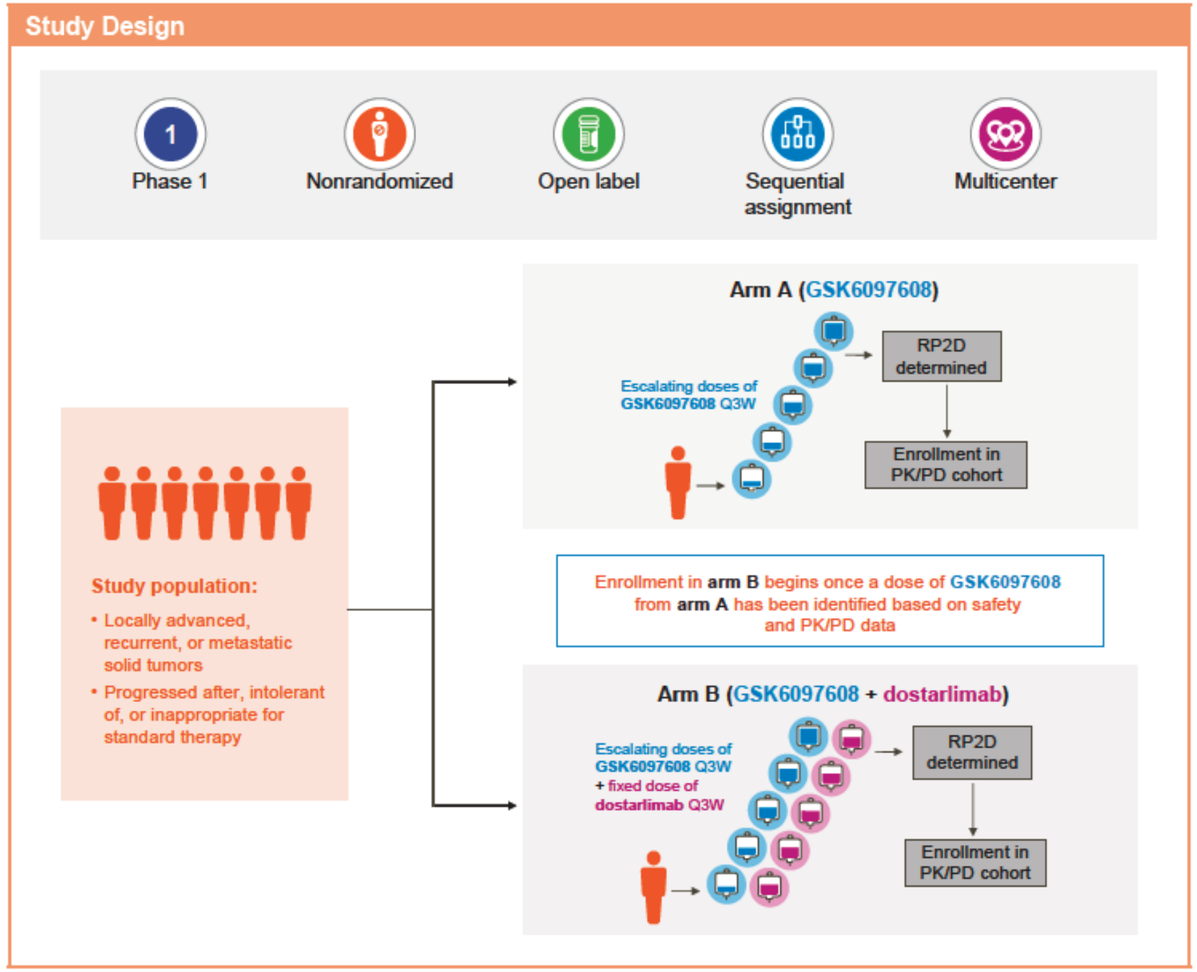


Cancer Immunol Res. 2019;7(4):559

CD226 pathway components owned by GSK		
Component	Molecule	Partner
PD-1	Dostarlimab	Acquired from Tesaro
CD96	GSK'608	23andMe
PVRIG	SRF813	In-license from Surface Oncology
TIGIT	GSK4428859 (EOS448)	iTeos

GSK6097608: Phase 1 Study Design

https://www.clinicaltrials.gov/ct2/show/NCT04446351



Study Endpoints

Primary	• Dose limiting toxicities • Adverse events	• Serious adverse events
Secondary	• ORR per RECIST 1.1 • ADAs against GSK6097608 and dostarlimab • PK parameters of GSK6097608 and dostarlimab	• Clinically important changes in laboratory parameters, electrocardiograms, and vital signs • Dose reductions or delay • Withdrawal due to AEs

Current Status

The study is currently open and recruiting.

OPEN

Commenced in 2020; data expected 2022

ADA, anti-drug antibodies; AEs, adverse events; ORR, objective response rate; PK, pharmacokinetics; PK/PD, pharmacokinetics/pharmacodynamics; Q3W, every 3 weeks; RP2D, recommended Phase 2 dose

CD96 is Part of the Genetically-validated CD226 Axis and is Progressing in Clinical Development

- The 23andMe immuno-oncology signature has highlighted the importance of the CD226 pathway which includes CD96 and TIGIT

- Combining components of the CD226 pathway may be more efficacious than inhibiting single components, but will require complex clinical trials
  - GSK has the relevant agents to target the CD226 axis

- The Phase 1 clinical trial with GSK’608 (anti-CD96) and dostarlimab is ongoing (conducted by GSK)
  - Data is expected in 2022

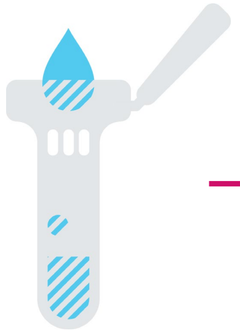
Using Genetics to Inform Clinical Development

Jennifer Low, M.D., Ph.D.
Head of Therapeutics Development

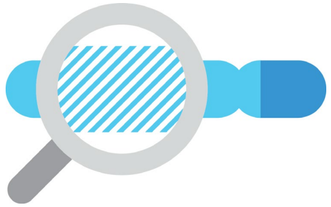
What if we could use **genetics** to predict **immune function** and **immune response** to I/O agents?

Genetics-Based Drug Development

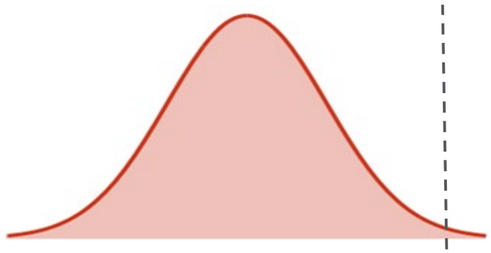
Taking a different approach across our development pipeline



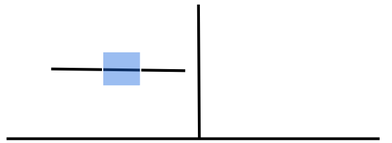
Spit



Genotyping

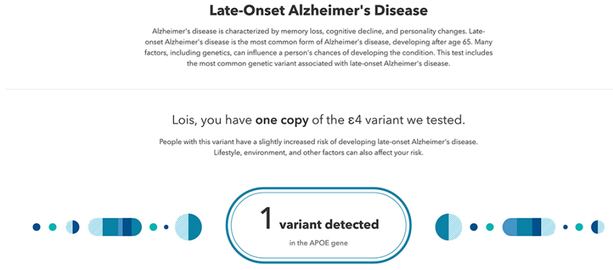


Genetic Classification



Exploratory Outcome Research

Clinically Informative Variants Detected Incidentally

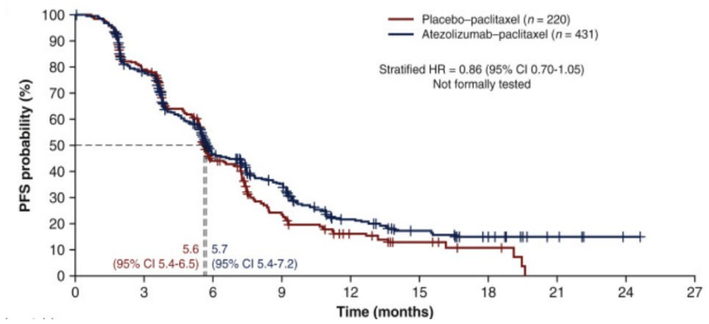


FDA-Authorized Report

Polygenic Scores for Hypothyroidism, Psoriasis Predicted Clinical Efficacy to Immune Checkpoint Blockade

Negative Phase 3 Studies

IMpassion131 Phase 3 (Breast Cancer)

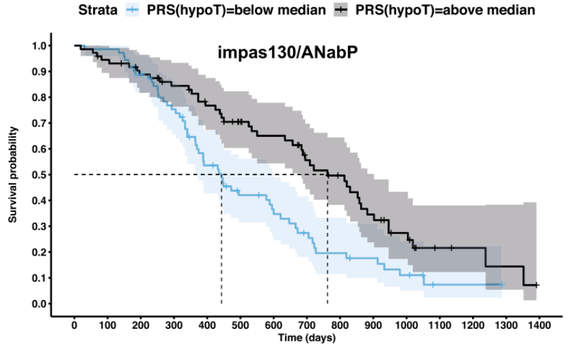


Miles, et al. Ann Oncol 32:994, 2021.

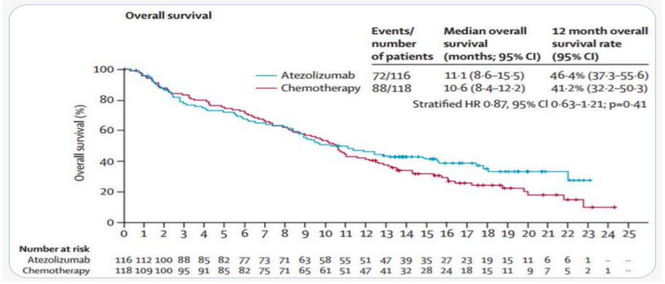
Genetic variation associated with thyroid autoimmunity shapes the systemic immune response to PD-1 checkpoint blockade

Zia Khan[1✉], Christian Hammer[1], Jonathan Carroll[1], Flavia Di Nucci[1], Sergio Ley Acosta[1], Vidya Maiya[1], Tushar Bhangale[1], Julie Hunkapiller[1], Ira Mellman[1], Matthew L. Albert[1,3], Mark I. McCarthy[1] & G. Scott Chandler[2✉]

NATURE COMMUNICATIONS | (2021) 12:3355 | https://doi.org/10.1038/s41467-021-23661-4 | www.nature.com/naturecommunications



IMvigor211 Phase 3 (Bladder Cancer)

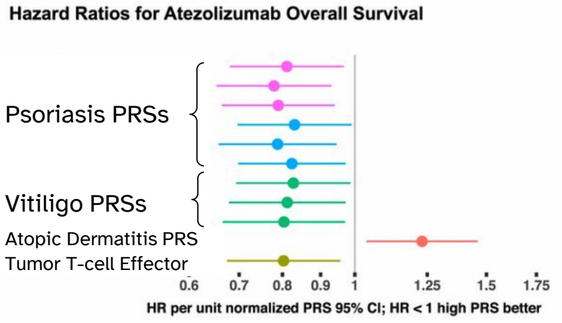


Powles, et al. Lancet 391:748, 2018.

Polygenic risk for skin autoimmunity impacts immune checkpoint blockade in bladder cancer

Zia Khan[a,1], Flavia Di Nucci[a], Antonia Kwan[a], Christian Hammer[a], Sanjeev Mariathasan[a], Vincent Rouilly[a], Jonathan Carroll[a], Magnus Fontes[a], Sergio Ley Acosta[a], Ellie Guardino[a], Haiyin Chen-Harris[a], Tushar Bhangale[a], Ira Mellman[a,1], Jonathan Rosenberg[b], Thomas Powles[c], Julie Hunkapiller[a], G. Scott Chandler[a], and Matthew L. Albert[a,1,2]

PNAS June 2, 2020 117 (22) 12288-12294; first published May 19, 2020; https://doi.org/10.1073/pnas.1922867117



Polygenic Scores May Predict Safety and Efficacy

- 23andMe is incorporating clinical genotyping into our clinical trials
- Use of polygenic scores could enable more efficient clinical development and improve the probability of success
- Developing drugs in genetically-defined patient populations may differentiate products based on better outcomes and improved benefit-risk profiles

Providing the right drugs to the right patients

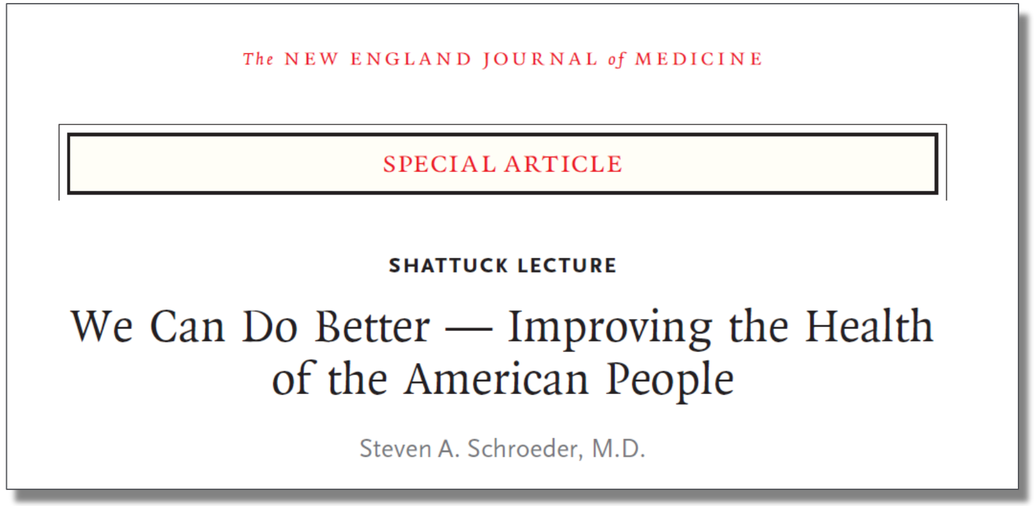
Executive Summary - Therapeutics

- 23andMe has generated a research and development pipeline covering multiple therapeutic areas in indications of high unmet medical need
- To date, more than 40 programs have been generated from the database as part of our collaboration with GSK
- GSK has extended their exclusive target discovery period of their collaboration with 23andMe for an additional fifth year
- 23andMe advanced a novel immuno-oncology antibody targeting CD200R1, 23ME-00610, into the clinic
- 23andMe has taken a royalty option on immuno-oncology antibody collaboration program targeting CD96 into later stages of development
- Managing our therapeutic portfolio investments based on scientific data to optimize investment, mitigate risk and maximize potential future returns

Genetics-Based Primary Care

Paul Johnson
Vice President, General Manager, Consumer

Impact of Different Factors on Risk of Premature Death



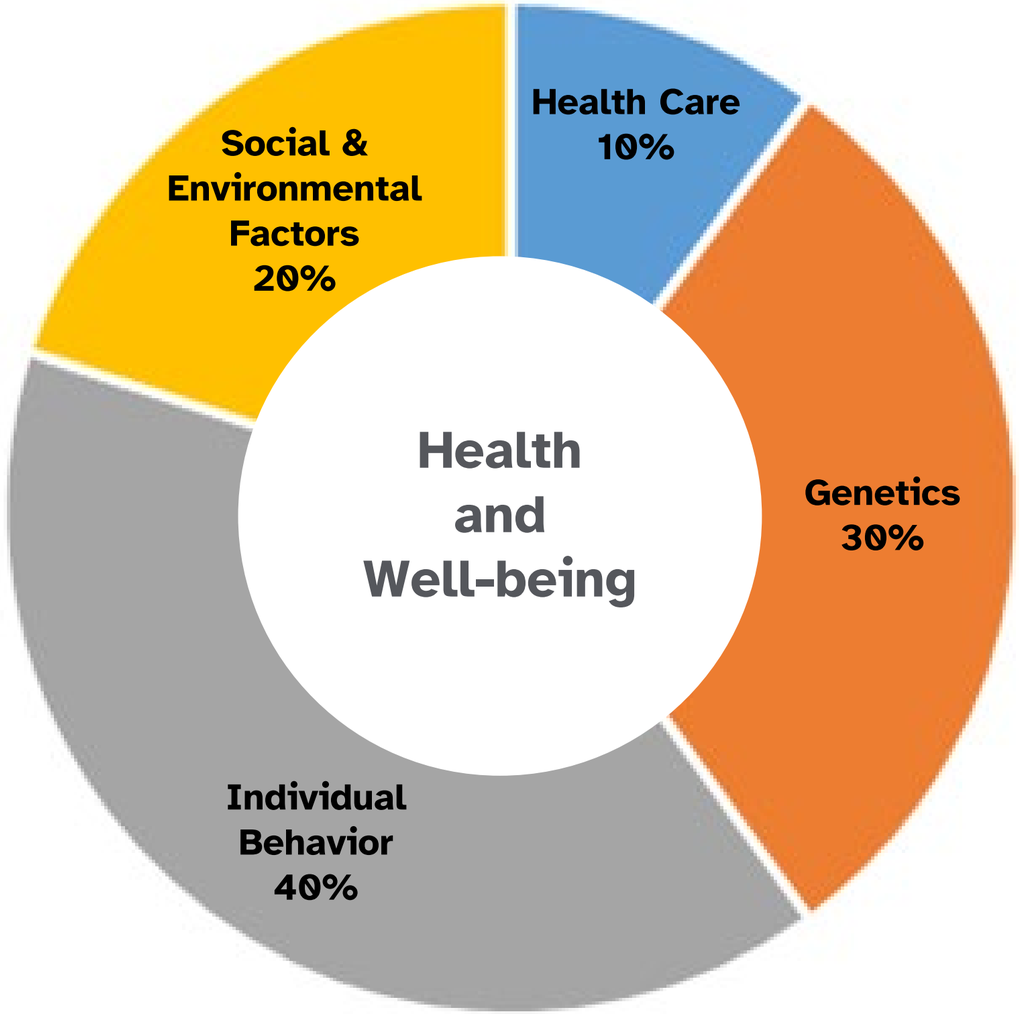
The NEW ENGLAND JOURNAL of MEDICINE

SPECIAL ARTICLE

SHATTUCK LECTURE

We Can Do Better — Improving the Health of the American People

Steven A. Schroeder, M.D.



Source: Schroeder, SA. (2007). We Can Do Better – Improving the Health of the American People. NEJM. 357:1221-8.

Opportunity to Deliver **Genetics-Based Primary Healthcare** at Scale

23andMe®

+

lemonaid

»»

- Genetics-Based Primary Care
- Telehealth
- Diagnostics Testing
- Wellness Reports
- Pharmacy / E-Prescribing
- Medical Records

1 Health Affairs, “Views Of Primary Care Providers On Testing Patients For Genetic Risks For Common Chronic Diseases.” (2018).

What is **Genetics-based Healthcare**?

Health Predispositions

Targeted prevention, monitoring, and management

Carrier Status

Understanding your potential risks

Wellness

Targeted to help you feel your best

Pharmacogenetics

Therapeutics that work for you

Personalized Healthcare at Scale

Healthcare based on a patient's wellness, choices, and genetics

Acquiring Lemonaid Health positions us to

- Provide personalized healthcare at scale
- Become a trusted holistic wellness and healthcare brand
- Create a fully integrated offering across 23andMe with accessible, affordable healthcare, driving strategic differentiation

The Power of Polygenic Risk Scores (PRS) for Personalized Healthcare

Geoff Benton, Ph.D.
Director, Product R&D

Consumer Powered Healthcare Flywheel

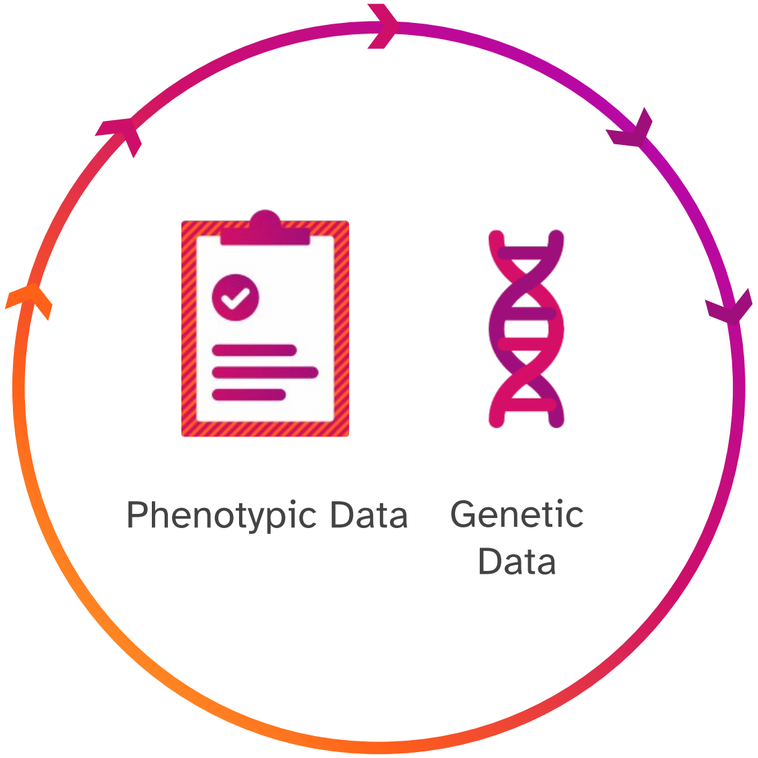
We run hundreds of billions of association tests per year that further our unique understanding of human biology

Consumer | Research | Therapeutics / Product

11.9M
Genotyped Customers[1]

Data

80%
Opt-In to Research



Insights

4B+
Phenotypic Data Points[1]

Drug Discoveries
40+ Programs[2]

Novel Consumer Products

Innovative Results Return Value to the Customer

1. As of September 30, 2021. 2. As of March 31, 2021. Programs include collaborated, 100% owned and royalty interest targets.

Our Health Service

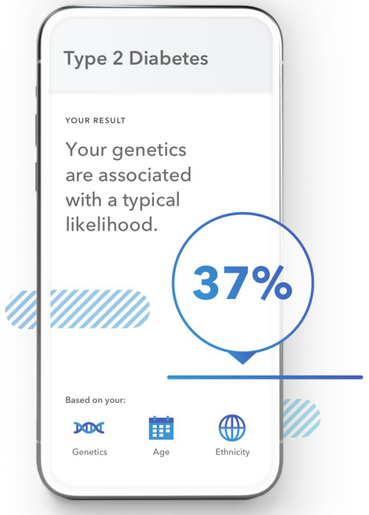
The First and Only Multi-Disease DTC Genetic Service That Includes FDA-Authorized Reports and Provides Personalized Genetic Insights and Tools

7 FDA Authorizations

Health Predispositions

30+

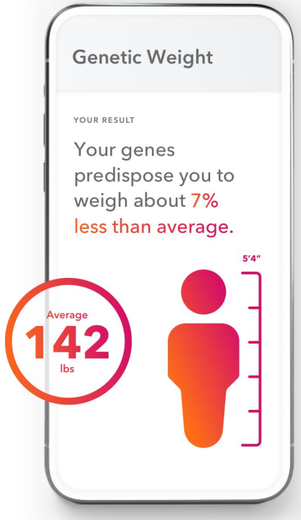
Including:
Type 2 Diabetes (Powered by 23andMe Research)
Coronary Artery Disease 23andMe+
Uterine Fibroids
Migraine 23andMe+
MUTYH-Associated Polyposis
BRCA1/BRCA2 (selected variants)
HOXB13 (prostate cancer)



Wellness [1]

10

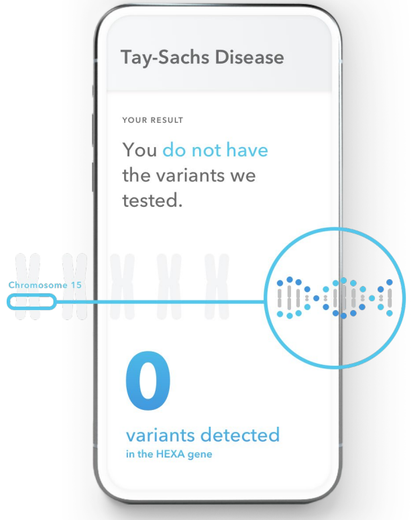
Including:
Muscle Composition
Genetic Weight
Alcohol Flush Reaction
Saturated Fat and Weight
Sleep Movement
Dog & Cat Allergies 23andMe+



Carrier Status

40+

Including:
Cystic Fibrosis
Sickle Cell Anemia
Familial Hyperinsulinism (ABCC8-Related)
Tay-Sachs Disease
Glycogen Storage Disease (Type 1a)



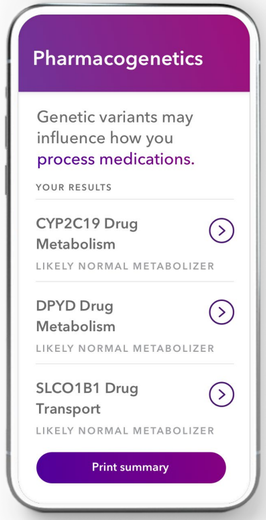
Pharmacogenetics

3

23andMe+

Including:
SLCO1B1 Drug Transport
CYP2C19 Drug Metabolism
- e.g., citalopram and clopidogrel

DPYD Drug Metabolism



1 Wellness information does not require FDA Authorization.

23andMe®

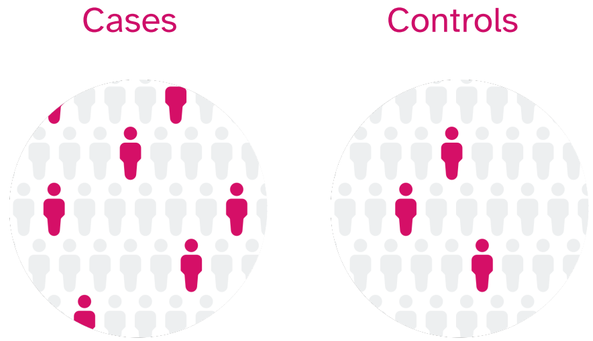
Genome-Wide Association Studies (GWAS)

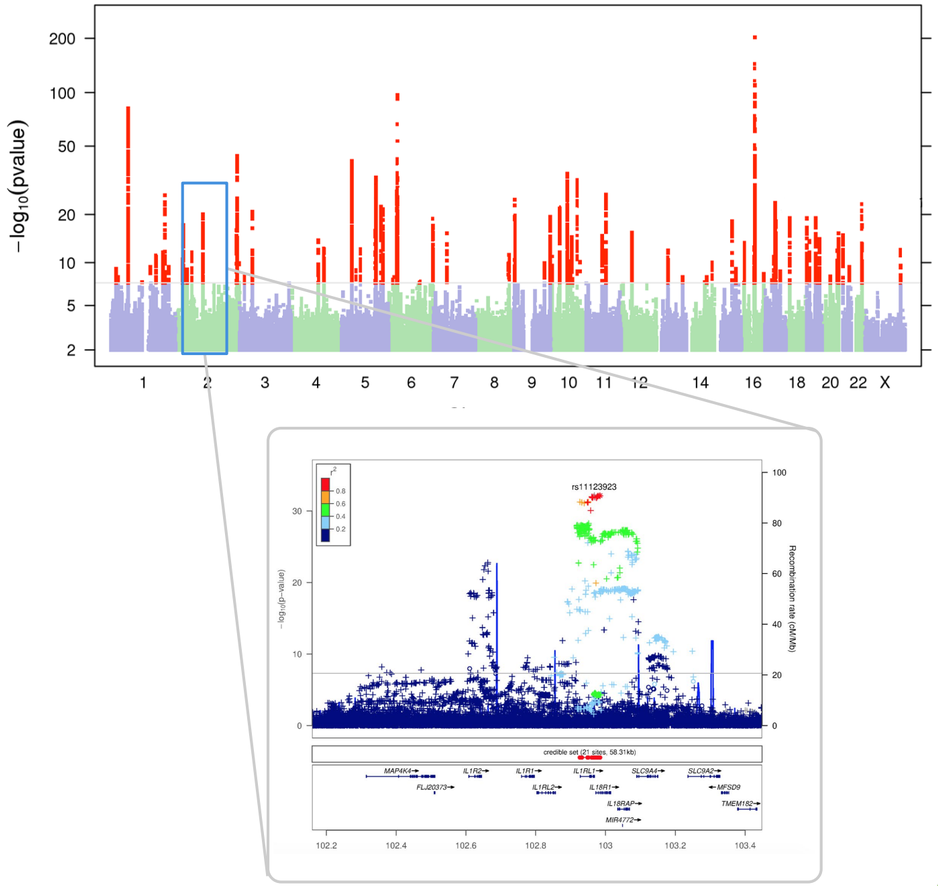
- GWAS is a statistical analysis of Single Nucleotide Polymorphisms (SNPs), looking To identify differences in frequency between disease cases and controls.
- SNPs linked with disease will be found at different frequencies in cases versus controls.
- Association is represented by the level of statistical significance (p-value) of the SNP frequency difference.
- SNPs can be tested across the genome and mapped to specific regions.

Single Nucleotide Polymorphism (SNP)

GGCCAGCTGGACGAGG

GGCCAGCTGGATGAGG





Prediction with Polygenic Scores (PGS)

GWAS

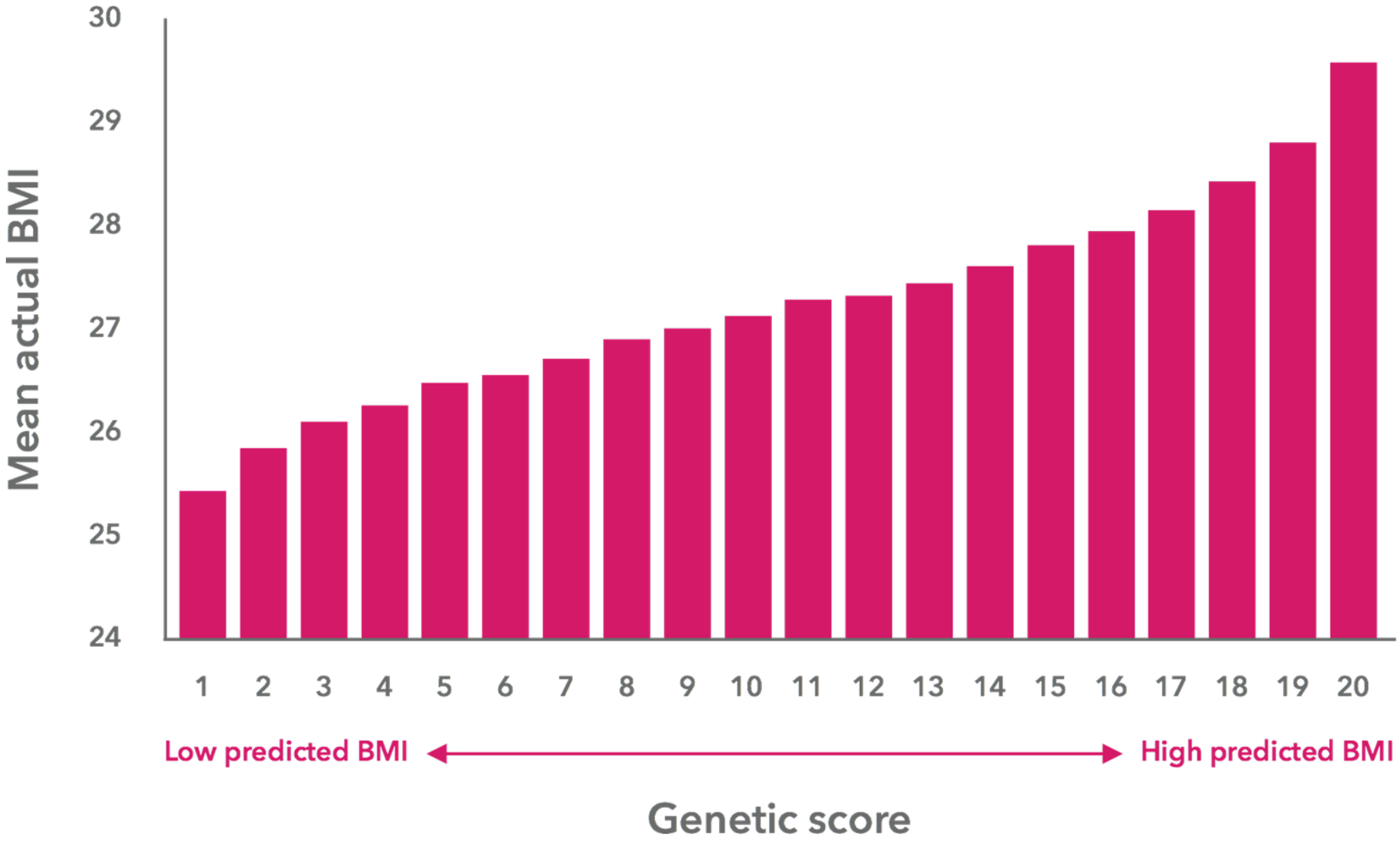
Feature selection

Modeling

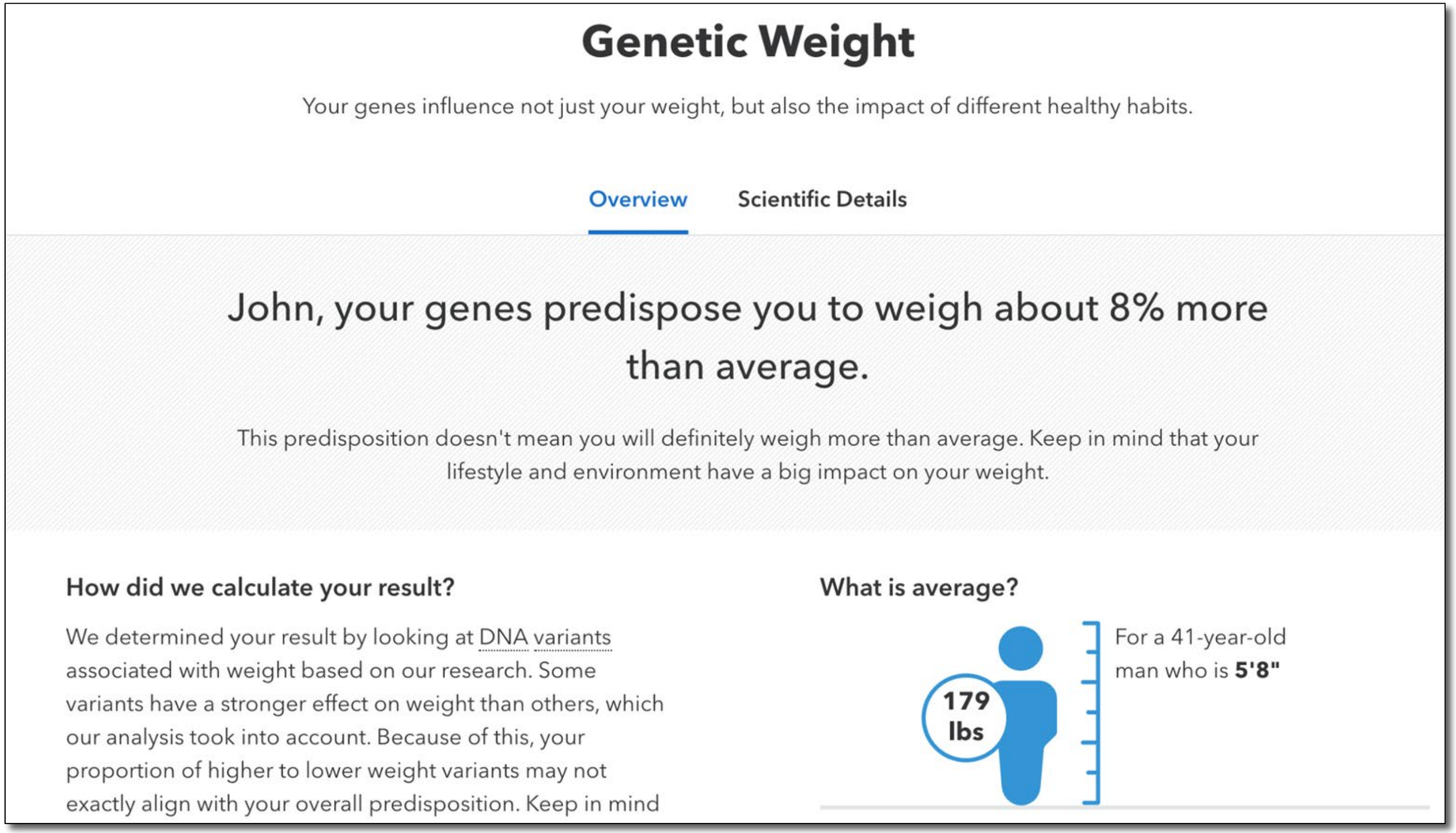
Personalized risk estimates

GWAS: Genome-Wide Association Study

Using Polygenic Risk Scores to Meaningfully Stratifying Actual Results



Personalized Risk Report



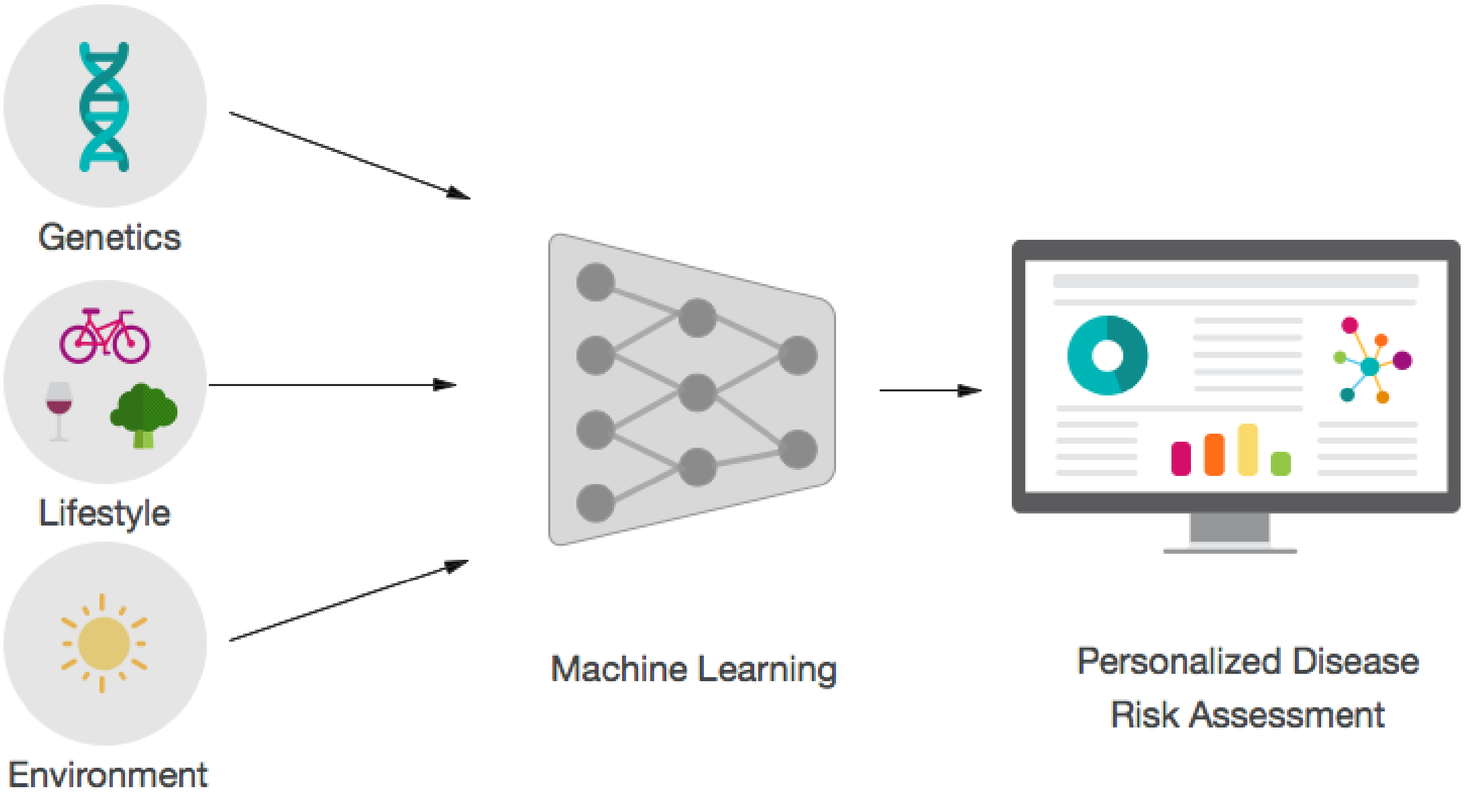
FY'21 Releases

- Coronary Artery Disease, Atrial Fibrillation, High Blood Pressure, LDL Cholesterol
- Migraine
- Uterine Fibroids
- Obstructive Sleep Apnea
- Restless Legs Syndrome
- Gout
- Non-Alcoholic Fatty Liver Disease
- Kidney Stones
- Polycystic Ovary Syndrome
- Triglycerides

FY'22 Releases

- Cat Allergy, Dog Allergy
- Eczema (Atopic Dermatitis)
- Low HDL Cholesterol
- Gallstones
- Gestational Diabetes
- Severe Acne
- Nearsightedness
- Coming soon
- Coming soon
- Coming soon

It's Not Just About Genetics, It's About **Prediction**



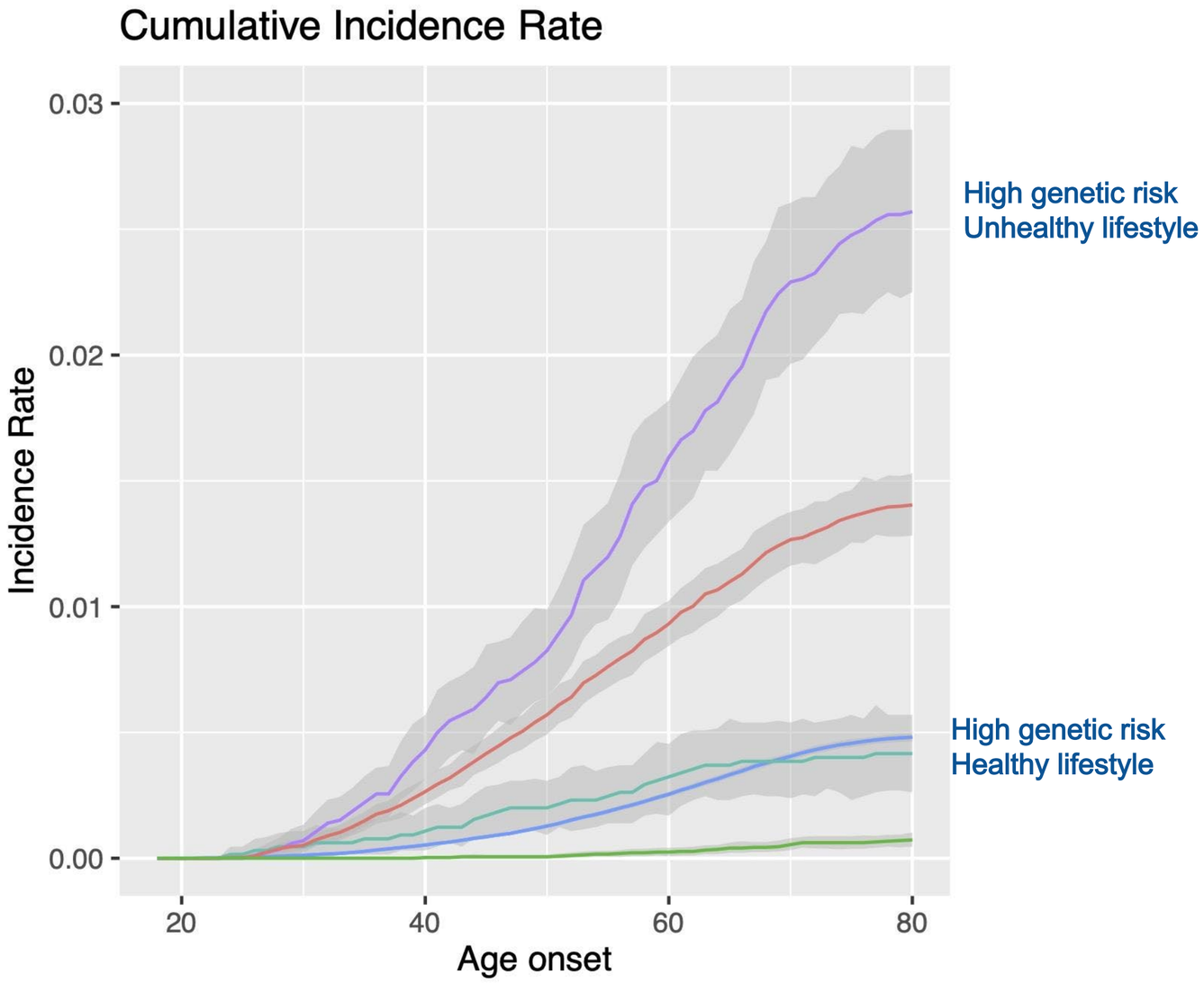
- Insight into potential **trajectories**
- Potential impact of different **interventions**
- **Risk over time** as behaviors, biometrics, and health change

Using PGS to Predict Real-world Outcomes

- Can use PGS to predict incident cases of Type 2 Diabetes (T2D)
- The 95th percentile of genetic risk has nearly 3x increase in risk for developing T2D

Bending the Curve with Lifestyle

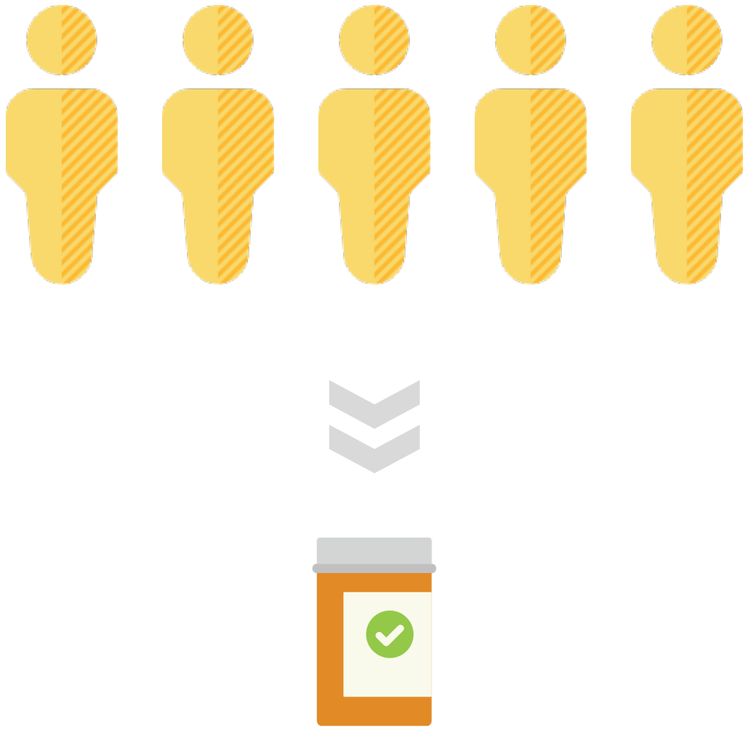
- Combine PGS with lifestyle factors to improve prediction of incident cases of Type 2 Diabetes (T2D)
- Lifestyle factors allow for greater precision of risk estimates and better personalization of results for customers



Opportunity for Personalized Healthcare at Scale

Practice of Medicine Today

Reactive – no customization until symptomatic



23andMe+®

Proactive – truly individualized from the very beginning

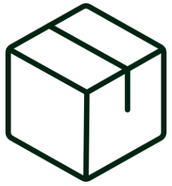
Delivering a Genetics-based Primary Care Service

Davis Liu, M.D.
Chief Clinical Officer

Lemonaid Health is Fully Integrated with a Broad Service Offering

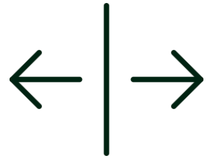
Online doctor visits

Cutting out the doctor waiting room – with fully integrated w-2 core clinical team



Mail order pharmacy

Cutting out the retail pharmacy – owned and controlled mail order pharmacy



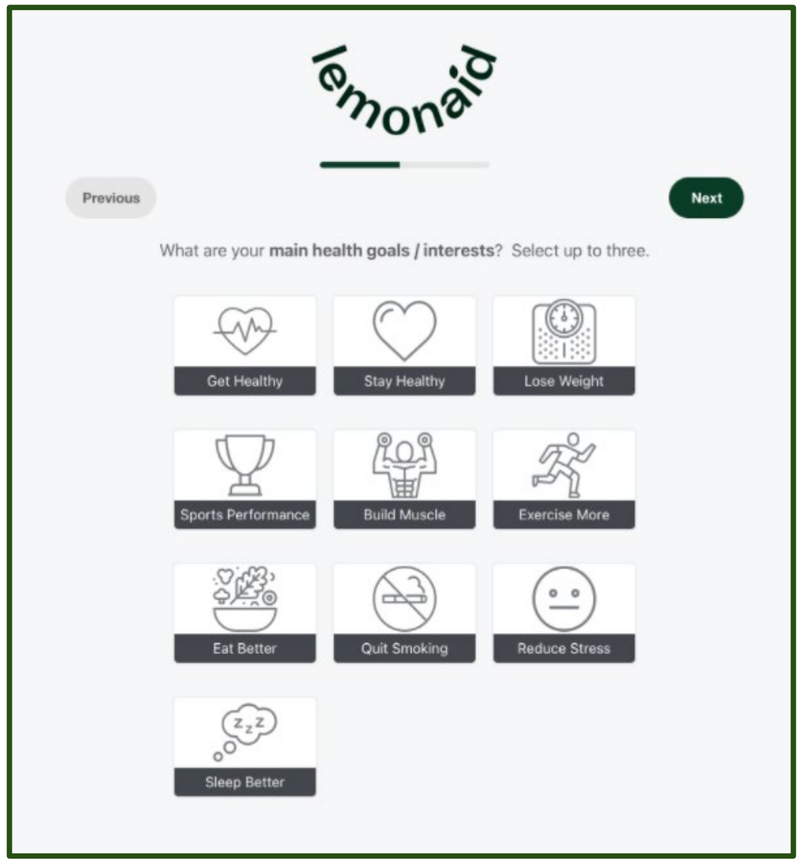
Broad range of services

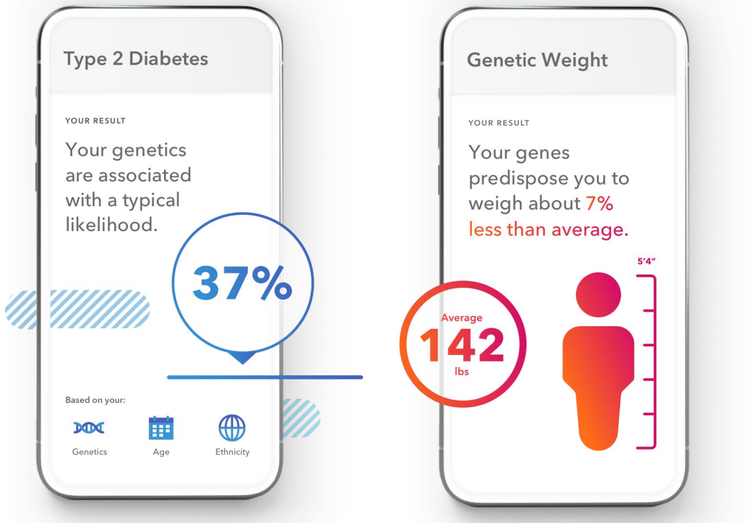
Building the online healthcare brand with the biggest impact

All connected using an algorithm-driven proprietary technology platform

The Future: Primary Care Complete

Will be matched with a doctor who is attuned to genetics, wellness goals, interests, and medical conditions.



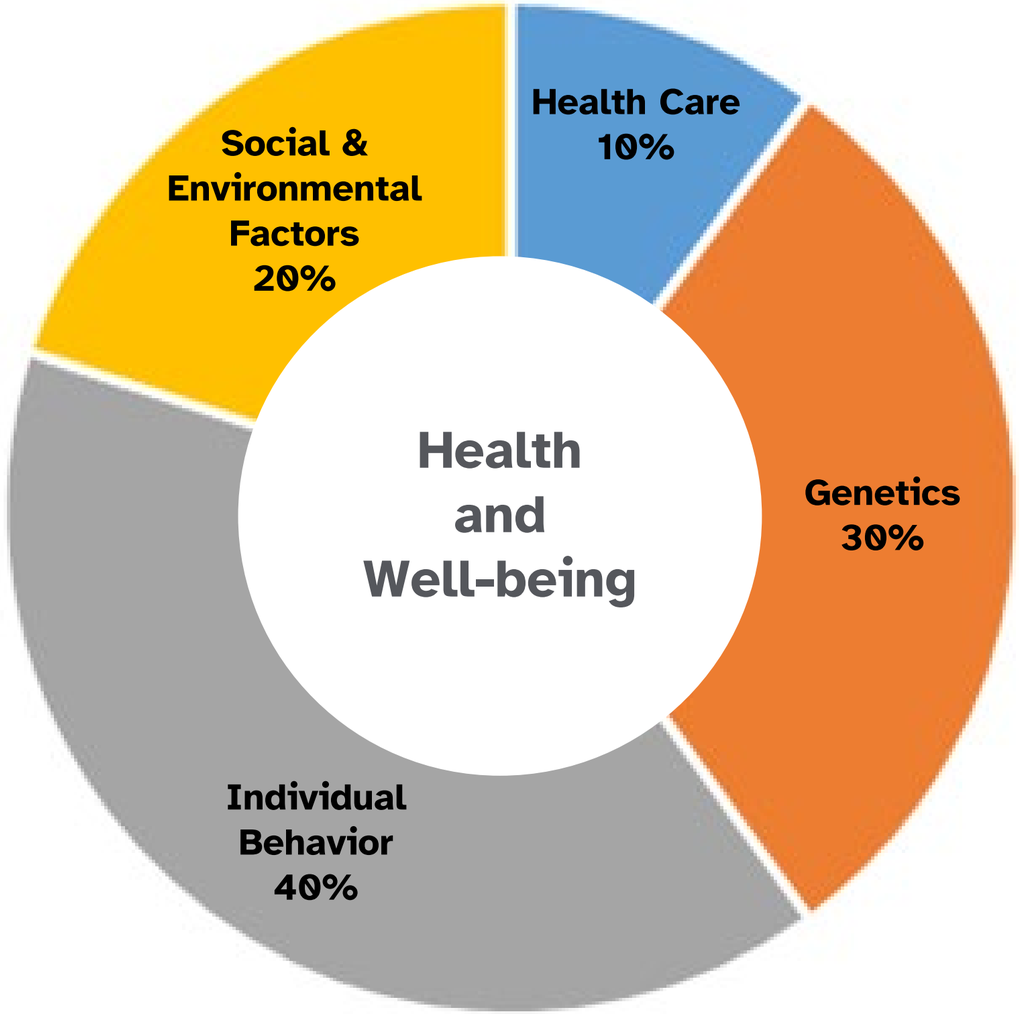


Lemonaid Health clinician

The Future: Primary Care Complete

- Initial video visit focused on overall health and well being - not just the 10%
  - Genetics
  - Individual Behavior
  - Wellness
  - Health Care
- Long-term relationship
- Leading to long, healthy, productive lives

Just the beginning!



Concluding Remarks

Anne Wojcicki
CEO and Co-Founder

Future of 23andMe

- Continuing to be **world leader** in direct-to-consumer **personal genetic health services**, growing annually

- Pioneering a **genetics-based primary care service** that empowers individuals to be proactive with their health

- Developing a pipeline of **over 40 clinical and research stage programs** addressing targets validated by human genetics

- **Leveraging strong balance sheet** to support investment in therapeutics portfolio and strategic initiatives in DTC personal health services

Q&A